Contents

Part I

General Introduction

Chapter 1

Introduction and Organization of the Thesis

1.1 Overview of Thesis

By nature, Ecology and Epidemiology are two distinct fields of research. A lot of works have already been done in these two subjects.

The main objective in the present investigation is to observe the dynamics of some ecological and epidemiological issues with some ecological and epidemiological factors. The present thesis is divided into two parts based on ecological and epidemiological issues. The first four chapters deal with the problem of ecological issues such as Phytoplankton-Zooplankton-Fish dynamics; prey-predator dynamics where prey population is subdivided into juvenile prey and adult prey; effect of harvesting and additional food in a food chain model (devoted two chapters). The last four chapters deal with the problems of some epidemiological issues such as Cholera (devoted two chapters), Malaria and Japanese Encephalitis.

The above issues are modelled with the help of ordinary differential equations. Basically all the mathematical models represent dynamical systems. In most of the models, the dynamics have been represented through autonomous dynamical system and some are represented through non-autonomous dynamical systems (Chapters 6, 8, 9). The dynamics of such system have been analyzed through local and global stability analysis. To study the global dynamics a suitable Liapunov functions and geometric approach are used. Moreover, the bifurcation analysis and exclusive numerical analysis have been performed to substantiate the analytical findings.

Before entering to describe the main motivation and objective of the thesis, I would like to take opportunity to present the brief historical literature reviews on ecological and epidemiological problems.

1.2 Historical Literature Reviews on Ecological and Epidemiological Problems

1.2.1 Historical Literature Review on Ecological Problems

An ecosystem model is the mathematical representation of an ecological problem, which is studied to gain understanding of the real system. First well-known ecological model on the predator-prey dynamics was developed by Lotka [138] in 1925 and then Vito Volterra [242] in 1926. This model consists of a pair of ordinary differential equations with a prey species and the other its predator. The dynamics of this particular type of model and its various modifications have received great attention since last 90 years or so. The book of Gause (1935), 'the struggle for existence' is a good representative of some mathematical models on ecology. In 1956, Lotka [139] published a book on mathematical modelling on ecological system. In this book, different predator-prey mathematical models had been discussed. Rosenzweig and MacArthur [191] studied the graphical representations and stability conditions of a predator-prey system in 1963. In 1967, MacArthur and Wilson [144] investigated the interaction between different species in an island. DeAngalis et al. [56] explored a modelling study on trophic interaction in 1975. Maynard-Smith [151] studied different types of mathematical models on ecological system in 1978. In 1980, Freedman [72] investigated some deterministic mathematical models on population ecology. It is noted that bifurcation is a common feature of a predator-prey dynamics. In 1981, Hassard et al. [91] studied the theory and application of Hopf bifurcation on a nonlinear system. Freedman and Waltman [73] investigated about the persistent of three species predator prey system. In 1985, Freedman and So [74] explored the global stability analysis of persistence of a simple food chain model. An important contribution in the field of ecological system has been made by J. D. Murray. We find an extensive literature on this subject in his book 'Mathematical Biology' (1989). Freedman and Mathsen [75] studied the effects of ratio-dependency on the persistence of predator-prey system in 1993. In 1996, Chattopadhyay [42] reported the effects of toxic substances on a two-species predator-prey system. Li and Muldowney [131] reported the geometric approach on the global stability of a nonlinear system in 1996. In the same year, Perko [181] investigated the general theory of dynamical system. Bazykin et al. [22] studied the dynamics of different types of interacting populations in 1998. In 1999, Stone and Hart [228] reported the effects of immigration on the dynamics of a simple population model.

It is noted that functional response has a great importance for the study of predator-prey dynamics. In ecology, it is the intake rate of a predator as a function of food (prey) density. At first, the concept of functional response has come from the work of Holling [98] in 1959. Then in 1977, Levin [129] investigated predator-prey system with different types of functional responses. After that, many research works have been done on predator-prey models with different types of functional responses. Gunog and DeAngelis [85] studied the effects of Holling type I functional response on predator-prey dynamics in 2011. In 2015, Madhusudanan and Vijaya [145] investigated the effects of Holling type II functional response function on predator-prey dynamics. Morozov [156] reported the role of Holling type III functional response on predator-prey interaction in 2010. Note that all these functional responses are prey dependent. In 1989, Arditi and Ginzburg [10] studied the impact of the ratio (prey and predator) dependent functional response on a predator-prey model. In 1992, Berryman [25] reported the necessity of ratio dependent functional response on predator-prey system. Kuang and Beretta [123] analyzed a ratio-dependent predator-prey model in 1998. Xiao and Jennings [252] investigated bifurcation analysis of a ratio-dependent predator-prey system in 2005. Li and Xiao [130] studied the bifurcation analysis of a predator-prey system with Holling and Leslie type functional responses in 2007. Yu [256] reported the effects of Bedington-DeAngalis type functional response on a predator-prey dynamics in 2014. It is better to mention that the use and types of functional responses are debatable issues whether a specific dynamics is to be prey dependent/predator dependent or both. Recently, scientists are thinking that it is better to represent the dynamics phenomenologically not mechanistically.

Plankton are multiple group of organisms that live in the water column of large bodies of water on sea, lake, river etc. and are the primary food source for the living animal in aquatic ecosystem. These are directly consumed by Zooplankton. So, the study about the dynamics of Phytoplankton and Zooplankton is very important in our society since these are the primary food source for all living organisms on the earth. In 1961, Hutchison [103] reported the dynamics of Plankton and its impact on the predator-prey system. Steel and Henderson [226] investigated the dynamics of the Plankton population through mathematical model in 1981. After that in 1992, Steel and Henderson [227] reported the impact of predation in Plankton system. Huxel and MaCann [104] investigated the food web stability and the influence of trophic flow on the predator-prey system in 1998. In 2002, Chattopadhyay and Pal [39] studied the impact of viral infection on the Zooplankton-Phytoplankton system through mathematical model. Again, in 2003, Chattopadhyay et al. [40] reported the effects of nutrient on the Phytoplankton dynamics in the presence of viral infection. In 2007, Pal et al. [169] investigated the impact of toxin for the occurrence and termination of planktonic bloom. In the presence of environmental fluctuation, Pal et al. [170] studied the effects of competition for the occurrence and control of planktonic bloom in 2009. The research is still going on. I like to devote a chapter on this issue.

It is well known that, refuge is the behavior of a prey which helps to escape itself from the predation by a predator. It has great effect to stabilize or destabilize of a predator-prey system. In 1987, Sih [215] studied the impact of prey refuge on the stability of a predator-prey system. Ruxton [195] reported the effects of using refuge in a short time duration of a predator-prey system in 1995. In 2006, Huang et al. [100] investigated the impact of prey refuge on a predator-prey system with Holling type III functional response. Liu and Han [135] studied the influence of prey refuge on a diffusive predator-prey system in 2011. In 2012, Jana et al. [107] investigated the global stability and bifurcation analysis of predator-prey system in the presence of prey refuge. In the same year, Sarwardi et al. [208] studied the effects of prey refuge on a competitive predator-prey system. In 2013, Devi [58] has reported the effects of prey refuge on a ratio-dependent predator-prey model.

Due to the prey refuge behavior, the predator population cannot consume always sufficient amount of foods for their growth. In such a case, the additional food supply may control this food crisis. Srinivasu et al. [223, 224] reported the impact of additional food on pest management and biological controls of predators. In 2012, Kar et al. [115] investigated the role of supplying additional food on an exploited predator-prey system. In the same year, Gakkhar and Singh [79] studied about the control of chaos in a predator-prey system by supplying additional food to the system. Prasad et al. [185] reported the effects of additional food to the dynamics of predator-prey system with mutually interfering predators in 2013. In 2014, Sahoo and Poria [198, 201] investigated the role of additional food to control the chaos and its effects on time delay in an eco-epidemiological model. Sen et al. [211] have studied the impact of supplying additional food in a predator-prey model with harvesting in 2015.

Now, harvesting is the collection of mature crops from the field. It is highly demandable area of research for the conservation biologist and ecologist who work on the biological conservations, because of continuous harvesting may cause the extinction of harvested population. So, the optimal use of harvesting is necessary to save the population and to maximize the harvesting. It has great effects on the predator-prey dynamics. In 1983, Hannesson [89] reported the effects of harvesting on a ecologically interdependent fish species. Ragozin and Brown [188] studied the harvesting policies and non-market valuation of a predator-prey system in 1985. Then, Clark [49, 50] investigated the bio-economic modelling, fishery management and also the optimal management of renewable resources. After that, Flaaten [69] reported the role of bio-economics of sustainable harvest between competing species in 1991. In 1998, Dai and Tang [54] investigated the effects of harvesting on the global dynamics of predator-prey system. Peng et al. [180] studied the impact of predator harvesting on predator-prey dynamics in 2009. In 2015, Pal et al. [172] reported the effects of predator harvesting in a fuzzy parameter based predator-prey model. Madhusudanan and Vijaya [145] studied the impact of quadratic harvesting on a predator-prey system with Holling type functional response in 2015. The above studies influenced me to study the dynamics of food chain models with additional

food and harvesting.

It is noted that in 1987, Ives and Dobson [106] studied the effects of anti-predator be-
havior in the dynamics of a predator-prey system. Abrams and Matsuda [2] investigated
the effects of adaptive predator and anti-predator behavior in a two prey one predator
system in 1993. In 1998, Krivan [121] studied the effects of optimal anti-predator behav-
ior of prey on predator-prey dynamics. In 2007, Ramao-Jiliberto et al. [189] reported the
effects of pre-encounter and post encounter inducible defence in a predator-prey system.
Choh [48] investigated the impact of predator-prey role reversals, juvenile experience and
adult anti-predator behavior on the predator-prey dynamics in 2012. In 2015, Tang and
Xiao [230] studied the impact of anti-predator behavior and bifurcation analysis of the
predator-prey dynamics. Gakkhar and Gupta [80] investigated the effects of prey pre-
dation, competition and commensalism in a predator-prey system in 2016. The above
studies motivated me to study the dynamics of prey-predator system by dividing the
prey population into two groups such as juvenile prey and adult prey.

1.2.2 Historical Literature Review on Epidemiological Prob-
lems

An epidemic model is a simplified means of describing the transmission of communicable
disease through individuals. The work of mathematical modeling of spread of infectious
disease was started in 1760 by Daniel Bernoulli [24]. In 1926, McKendric [152] inves-
tigated about the application of mathematics to medical problems. In the next year,
Kermack and McKendric [119] published the first authentic work on mathematical mod-
elling of infectious disease. They considered a simple deterministic model of an epidemic
problem and analyze the spread of the disease. After that many works of modelling of
infectious disease had been done by many researchers. In 1975, Bailey [18] studied the
applications of mathematical theory applied to the infectious disease modelling. May
and Anderson [150] investigated the regulation and stability of a host-parasite system in
1978. In 1979, Cooke [52] studied the stability of a vector disease model. Hethcote [93]
investigated the asymptotical behavior of a deterministic epidemic model in 1973. In the
same year, Hethcote [94] studied the effects of vaccination for controlling the epidemic.
In 1976, Hethcote [95] explored the dynamical analysis of various type of communicable
disease model. Hethcote and Van den Driessche [96] reported the impact of nonlinear
type incidence rate on the infectious disease models in 1991. In 1995, Smith and Walt-
man [222] studied the dynamics of microbial competition in a disease transmission model.
Zhao [262] published a book on "Dynamical System in Population Biology" in 2003. In
the same year, De Barros et al. [57] investigated the SI type epidemic model in a fuzzy
environment by taking transmission parameter as fuzzy. In 2006, Wang [243] studied the
backword bifurcation of an epidemic disease model. Zhang and Zhao [259] explored an
epidemic model in a periodic environment in 2007. Teng, Liou and Zhang [233] investi-

gated the persistence and extinction of a disease in a non-autonomous system in 2008. Again, in 2008, Wang and Zhao [244] studied the threshold dynamics of an epidemic model in a periodic environment. In 2010, Nakata and Kuniya [163] investigated the global dynamical analysis of a SEIRS type mathematical model on infectious disease in a periodic environment. The study of infectious disease is of great importance for human health and social environment. The present thesis aims to study the dynamics of such diseases e.g. Cholera, Malaria and Japanese Encephalitis by constructing some mathematical models.

Cholera is an infectious disease that causes severe watery diarrhea which can lead to dehydration. It is caused by eating contaminated food or drinking water with a bacterium called Vibrio Cholerae. Adams [3] studied the dynamics of bacteriophages to reduce the bacterium Vibrio Cholerae in 1959. Capasso and Paveri-Fontana [32] investigated the dynamics of a Cholera disease transmission in European Mediterranean region in 1979. Again, Chakraborti [33] reported the interaction between the organism Vibrio Cholerae with bacteriophage in 1996. In 2001, Codeco [51] investigated the role of the aquatic reservoir for the transmission of Cholera disease. Alam et al. [6] explored the effects of seasonality on the transmission of Cholera disease in 2006. In the same year, Jensen et al. [110] investigated the role of bacteriophage to control the Cholera disease outbreak. In 2008, Emch et al. [65] studied the effects of seasonality on the transmission of Cholera disease from 1974 to 2005. Nelson et al. [165] reported the dynamics of Cholera disease transmission as well as the interaction between host, pathogen and bacteriophage in 2009. In 2010, Gazi et al. [82] worked on the impacts of environmental fluctuation on the transmission dynamics of Cholera disease. Liao and Wang [132] studied about the applications of mathematical model on Cholera disease and its stability in 2011. Zhao et al. [263] explored the impact of imperfect vaccination on the transmission of Cholera disease in 2012. In 2013, Zhou and Cui [264] investigated the impact of time dependent transmission rate on the dynamics of Cholera. After that Wang et al. [248] studied the effects of human behavior on the transmission of Cholera in 2015. In the same year, Wang and Wang [247] have also reported the impact of bacterial growth and spatial movement on the transmission of Cholera. I gain some motivation on the above works and would like to present two chapters on this issue by constructing a suitable mathematical models.

In our social life Malaria is an infectious disease of humans transmitted by mosquito and other animals caused by parasitic protozoans. Symptoms of Malaria are fever, fatigue, vomiting and headaches. Therefore, research on Malaria transmission is very important to us. In 1988, Aron [12] investigated the modelling study on the dynamics of a Malaria disease. Bouma [28] reported that the climate change has great impact on the transmission dynamics of Malaria disease in 1994. In 1999, Craig et al. [53] investigated the effect of climate change on the transmission dynamics of Malaria in sub-saharan Africa. Singh et al. [219] studied the effects of environmental and ecological fluctuation on the transmission of Malaria in 2005. Wyse Ana et al. [251] investigated the effects

of treatment intensities to control the Malaria disease in 2007. In 2008, Wei et al. [249] studied the impact of time delay on the dynamics of Malaria disease. Cai and Li [29] reported the effects of direct transmission on the transmission dynamics of Malaria in 2010. Chitnis et al. [46] studied different types of intervention strategies to control the Malaria disease transmission through mathematical model in 2010. In the same year, Saker [202] investigated the effects of delay on the transmission of Malaria disease. Again, Govella Nicodem [83] studied the impact of using insecticide treated bed-nets to reduce Malaria disease in 2010. In 2011, Lou and Zhao [141] investigated about the biological control of Malaria disease by introducing larvivorous fish. There are lot of works on this issue. I would also like to contribute a chapter on this burning problem.

Japanese Encephalitis (JE) is a vector borne viral disease occurred in South Asia, Southeast Asia, East Asia and Pacific. It is transmitted to humans through bites from infected mosquitoes of the Culex species. Japanese Encephalitis Virus (JEV) is transmitted from pigs or birds to mosquitoes through the bite of mosquitoes. It causes several thousand of deaths in the whole world in every year. Very few mathematical models have been developed to study the dynamics of Japanese Encephalitis disease and its control strategies. In 1993, Mukhopadhyay et al. [158] formulated a mathematical model to study the dynamics of Japanese Encephalitis disease. Again, in 1994, Mukhopadhyay and Tapaswi [159] investigated a SIRS type mathematical model on Japanese Encephalitis disease. Tapaswi et al. [232] studied the complex dynamics of Japanese Encephalitis among mosquito, reservoir and human in 1995. In 2005, Keiser [118] investigated the impact of irrigated rice agriculture on the dynamics of Japanese Encephalitis disease. E Erlanger et al. [64] studied the different dynamics of Japanese Encephalitis in present, past and future in 2009. In 2012, Singh et al. [221] reported a persistent threat to the human population due to Japanese Encephalitis. In 2013, Bandyopadhyay et al. [20] studied the incidence of Japanese Encephalitis in west bengal. Flohicle [70] investigated the role of climate change, landscape and viral genetics on the spread of Japanese Encephalitis disease in 2013. In the same year, Lindahl [133] reported the life cycle of Japanese Encephalitis Virus (JEV) among pigs, mosquito vectors and human. Then, Sharma et al. [213] studied the spread of Japanese Encephalitis disease in Asam in 2014. Still now, there are many cases of JE in India. With this view, I like to devote a chapter on this issue.

1.3   Motivation and Objective of the Thesis

Ecological system is concerned about the interaction among same or different organisms with abiotic components of their living environments. Practically, it is very necessary to study such systems for keeping the existence of human being. In this regard, different works are going on this field mathematically. But till now, there exists also some unexplored directions in this field. So, in this research work, one of the objectives is to integrate mathematically different factors influencing the dynamics of predator-prey ecological system.

With the development of human life, different infectious diseases are attacking the human population around the globe. Epidemiology is the study on analysis of different patterns, causes and effects of such diseases identifying the various risk factors and suggest some measures for control. These diseases make several thousands of death due to their unknown behavior and due to lack of appropriate control strategies. For this reason, the study is necessary to explore the actual dynamics of these diseases. Since 1760, many research works have been performed mathematically. But, still there exists some directions which are yet to be unveiled in epidemiology. Therefore, our other objective is to investigate mathematically different epidemiological problems considering the influence of different factors on the system.

Many researchers [39, 40, 77, 170] have worked on the dynamics of Phytoplankton and Zooplankton interaction model. Recently, Yunfei et al. [257] have discussed two species food chain model taking Phytoplankton as prey and Zooplankton as a predator with harvesting of both. It is known that Phytoplankton is the primary producer in a food chain. These are consumed by Zooplankton. Again, both Phytoplankton and Zooplankton are consumed by different Fish species. So, the growth rates of Fish species mainly depend on the availability of Phytoplankton and Zooplankton. Fish and other aquatic organisms are very essential to be processed into various food and non-food products such as shark skin leather, pigments made from the inky secretions of cuttlefish, isinglass used for the clarification of wine and beer, fish emulsion used as a fertilizer, fish glue, fish oil and fish meal. So, Fish is very important food source in human society. Though there are different predator-prey models, but no one considers Fish population separately as a predator. Hence, the study of dynamics of Fish species with Phytoplankton and Zooplankton is necessary. This point directs us to focus on the study of interactions among three species such as Phytoplankton, Zooplankton and Fish in a predator-prey model which has biological importance on the real world.

The life of an organism has been divided into different stages in a stage structured mathematical model. Every species always experience immature stage and mature stage of their life in the real world natural ecosystems and it performs different kinds of feature at each stage of growth. So, the ecological models with stage structure is more rational than without stage structure. In anti-predator behaviour, prey groups actively defend

themselves by attacking or mobbing a predator and save their offsprings from predation. Tang et al. [230] investigated the bifurcation analysis of a predator-prey model with anti-predator behavior of prey species in which anti predator behavior had been considered for all prey members. But, in reality it is seen that only adult prey can protect their infants (juvenile prey) from predation of predator by showing anti-predator behavior. There is no research papers including stage structure on prey species and anti-predator behavior of adult prey population. This vacuum has motivated us to work on predator-prey mathematical model with stage structure and anti-predator behavior of adult prey population.

There are several motivations such as conservation of a species, control of a pest population, chaos control, disease control etc. behind the supply of additional food in a predator-prey system. Supplying additional food in a predator-prey system is the great topics to many researchers due to its eco-friendly nature. Recently, Sahoo and Poria [200] have published a research paper on the impacts of additional food in a predator-prey system with harvesting. They have considered the additional food on top predator population and top predator can consume only the middle predator. But, in reality it is seen that the top predator can consume both prey and middle predators. So, this idea has inspired us to work on the predator-prey mathematical model in the presence of additional food to the top predator.

Already, many research works have been done on the predator-prey system by taking refuge by the prey species. The using of refuge parameter decreases the predation rate of predator. Therefore, the concept of refuge by prey should have important effects on the predator-prey interaction. Many studies have suggested that refuge for prey are crucial in explaining prey persistence. Chakraborty and Das [37] have reported the impact of incorporating constant prey refuge parameter on a predator-prey system with alternative food to predators. In this model, it has been shown that the supply of alternative food to the predator has a significant effect to stabilize the predator-prey dynamics considering refuge on the prey species. But the effect of additional food on super predator and refuge on predator may provide some interesting dynamics. We like to observe such dynamics by considering a food chain model.

It is well known that, Cholera is an infectious disease caused by the bacterium Vibrio Cholerae to be contaminated with food and drinking water. The poor sanitation, contaminated drinking water, poverty etc. are the main risk factors for the Cholera disease transmission. There are many mathematical models on Cholera disease dynamics and its control strategy. Experimentally, it has been proved in the paper of Jensen et al. [110] that bacteriophages can reduce the density of bacterium Vibrio Cholerae. So, the rapid decay of bacterial culturability and the predation of Vibrio Cholerae by bacteriophages have been observed in the dynamics of Cholera model. Therefore, bacteriophage has a great importance to control the Cholera disease transmission. This fact inspires us to incorporate bacteriophage in the Cholera disease transmission model including the time periodic nature of the disease transmission rates.

There exist many research papers of the Cholera disease transmission and its control strategies. But, the parameters involved in these model are crisp in nature. In the real world, every parameter related to the disease transmission model is changing with respect to time due to human activities and natural disasters. Thus, these parameters should not be crisp in nature always i.e., these may be uncertain in nature. There are very few works on fuzzy parameters based disease transmission model. Recently, Pal et al. [172] reported the impact of taking fuzzy parameter on predator-prey harvesting model. This article intends us to consider the parameters involved in a Cholera disease transmission to be uncertain in nature.

The other important disease to study is Malaria. It is a vector borne fatal disease caused by a parasite. It spreads in human population through the bites of infected mosquitoes. There are mainly four types of Malaria parasites such as Plasmodium falciparum, P. vivax, P. ovale and P. malariae, infect humans. It causes several thousands of death in every year. It still remains global threat for humans across the whole world though the medical science has been developed. Therefore, the actual dynamics of the Malaria disease transmission and new possible effective control strategies need further investigation. Wang et al. [246] investigated a mathematical model on Malaria disease transmission in a periodic environment. According to the transmission mechanism of Malaria disease they divided mosquito population into two subpopulations such as susceptible mosquito and infected mosquito. Again, human population is also divided into three subpopulations such as susceptible human, infected human and recovered human. Here, it has been considered that the disease transmission rates are time periodic. In their model, the control parameters were not considered to eradicate the Malaria disease from human population, which are most important parameters. This idea motivated us to develop the interaction between mosquito and human population with some control parameters which are time dependent.

Japanese Encephalitis (JE) virus is a single-stranded RNA virus which belongs to the genus Flavivirus. JE virus is transmitted into humans through the bite of an infected mosquito mainly Culex species. It grows in the body of the amplifying vertebrate hosts primarily pigs and wading birds. It makes several thousands of death in every year in India and other parts in the world due to lack of investigation of proper dynamical behavior using suitable control strategies. So, the study about the authentic dynamics of this disease and its control strategies are necessary. Tapaswi et al. [232] developed a three-populations such as mosquito, reservoir and human interaction Japanese Encephalitis disease transmission model. In their model, only stability analysis of the system around different equilibrium points was discussed and no control strategies are considered to control the disease. This influenced us to develop a Japanese Encephalitis disease transmission mathematical model among mosquitoes, reservoir and human populations with some suitable control parameters.

1.4 Organization of the Thesis

In the proposed thesis, some real life Ecological and Epidemiological problems are considered and solved. The proposed thesis has been divided into following four parts and eleven chapters.

Part I: General Introduction

- **Chapter 1:** Introduction and Organization of the Thesis

Part II: Studies on Dynamics in Some Ecological Problems

- **Chapter 2:** Stability Analysis of Coexistence of Three Species Prey-Predator Model

- **Chapter 3:** Stability and Bifurcation Analysis of a Stage Structured Prey-Predator Model with Ratio-dependent Functional Response and Anti-predator Behavior of Adult Prey

- **Chapter 4:** Effects of Supplying Additional Food in a Tritrophic Food Chain Model with Harvesting Only

- **Chapter 5:** Effects of Additional Food in a Predator-Prey System Incorporating Refuge and Harvesting

Part III: Studies on Dynamics in Some Epidemiological Problems

- **Chapter 6:** Dynamics of Cholera Outbreak with Bacteriophage and Periodic Rate of Contact

- **Chapter 7:** Dynamical Study in Fuzzy Threshold Dynamics of a Cholera Epidemic Model

- **Chapter 8:** Threshold Dynamical Behaviors of a Malaria Disease in Control Parameters Based Periodic Environment

- **Chapter 9:** Stability and Bifurcation Analysis of Japanese Encephalitis Model with/without Effects of Some Control Parameters

Part IV: Summary, Extension and Bibliography

- **Chapter 10:** Summary and Future Research Work

- **Chapter 11:** Bibliography

Part I
(General Introduction)

The Part I contains only one chapter (Chapter 1).

Chapter 1: Introduction and Organization of the Thesis

This chapter contains an introduction giving an overview of the development along with the historical literature reviews on Ecological and Epidemiological problems.

Part II
(Studies on Dynamics in Some Ecological Problems)

This part is divided into four chapters (Chapter 2, 3, 4 and 5) and in those chapters different ecological models are derived and solved.

Chapter 2: Stability Analysis of Coexistence of Three Species Prey-Predator Model

In this chapter, we have proposed a prey-predator model for the study of dynamical behaviors of three species such as toxin producing Phytoplankton, Zooplankton and Fish in a Fishery system. The stability condition, existence condition of equilibrium and bifurcation have been also established. In this chapter, Holling type II functional response has been considered to analysis the proposed model. All equilibriums of the proposed system are determined and the behavior of the system is also investigated near the positive equilibrium point. Hopf bifurcation analysis has been done with respect to the consumption rate of Zooplankton (β) and releasing rate of toxin substances (ρ) produced by unit biomass of Phytoplankton. Finally, some numerical simulations has been performed to verify our theoretical results.

Chapter 3: Stability and Bifurcation Analysis of a Stage Structured Prey-Predator Model with Ratio - dependent Functional Response and Anti-predator Behavior of Adult Prey

In this chapter, a three species predator-prey model such as (i) juvenile prey and (ii) adult prey and (iii) predator population has been developed. It is considered that the growth rate of juvenile prey depends on the adult prey populations and then the juvenile prey population becomes adult. The functional responses for predator to consume both the juvenile prey and adult prey population have been considered as ratio dependent. Also, the anti-predator behavior has been considered on adult prey population. Then, the boundedness of all solutions of our proposed mathematical model has been discussed.

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Also, we determine different equilibria and there existence conditions. Then, the stability conditions of the system around these equilibria have been analyzed. Global stability analysis of the interior equilibrium has been done. After that, the Hopf bifurcation analysis with respect to the anti-predator behavior of adult prey has been discussed. Finally, some numerical simulations have been performed to test our theoretical results.

Chapter 4: Effects of Supplying Additional Food in a Tritrophic Food Chain Model with Harvesting Only

In this chapter, we propose and analyze a three species predator-prey system in presence of additional food for predators. It is assumed that the middle predator is acting as a prey as well as predator and the top predator consumes both prey as well as middle predator. It is also considered that a constant amount of additional food for the top predators exists in the ecosystem. The effects of top predator harvesting are investigated. Then the existence and stability conditions of the equilibria have been discussed analytically. The Hopf bifurcation analysis of the system with respect to predation rate of prey to the top predator and the harvesting effort have been analyzed both analytically and numerically. Pontryagins maximum principle is used to determine the optimal harvesting of top predator population to maximize the discounted net revenue. From our analysis, it is seen that the additional food has significant effects to prevent the extinction risk of top predator population and also to increase revenue collection. Finally, some numerical results have been given in support of our analytical findings.

Chapter 5: Effects of Additional Food in a Predator-Prey System Incorporating Refuge and Harvesting

In this chapter, a food chain model has been developed among three species such as prey population, predator population, super predator population. Here, it is assumed that prey population grows logistically and predator population consumes prey only. But, the predator population is consumed by a super predator population. In this model, it is assumed that the predator population shows refuge behavior to the super predator population. Due to having the refuge characteristic of the predator population, the super predator feels the lack of food. To maintain the growth of the super predator properly, a constant amount of additional food is supplied to the system. Henceforth, partially the predator population is also benefited with this additional food. On the basis of this notions, a food chain model has been derived in which the extinction conditions of super predator population has been explored. Also, stability analysis of the model has been shown along with Hopf bifurcation analysis to examine some parametric values for which the system losses its stability. To get optimal harvesting of super predator, the

Pontryagins maximum principle has been used. Finally to study the feasibility of the model, some numerical simulations have been presented.

Part III
(Studies on Dynamics in Some Epidemiological Problems)

This part is divided into four chapters (Chapter 6, 7, 8 and 9) and in those chapters different epidemiological models are derived and solved.

Chapter 6: Dynamics of Cholera Outbreak with Bacteriophage and Periodic Rate of Contact

In this chapter, a Cholera epidemic model with periodic transmission rate has been considered and discussed. It is shown that the disease free equilibrium point is globally asymptotically stable and also seen that the Cholera disease is disappeared if the basic reproduction number is less than one. When the basic reproduction number is grater than one, then the endemic equilibrium is globally asymptotically stable. Finally, numerical simulations have been given for the existence of the analytical results.

Chapter 7: Dynamical Study in Fuzzy Threshold Dynamics of a Cholera Epidemic Model

In this chapter, a fuzzy mathematical model on Cholera disease has been developed in which all parameters related to the Cholera disease have been considered as fuzzy numbers. Here, total human population is divided into three subpopulations such as susceptible human, infected human and recovered human. Also, the bacterial population is the Vibrio Cholerae in the environment. Then the existence condition and boundedness of solution of our proposed mathematical model have been discussed. Also, the different equilibrium points and the stability condition of the system around these equilibrium points have been analyzed. The global stability condition of the proposed system around the endemic equilibrium point has been also discussed. Finally, some numerical simulations have been shown to test the theoretical results of the system.

Chapter 8: Threshold Dynamical Behaviors of a Malaria Disease in Control Parameters Based Periodic Environment

In this chapter, a Malaria disease transmission model has been developed in which the transmission rates from mosquito to human as well as human to mosquito and death

rate of infected mosquito have been constituted by two variabilities: one is periodicity with respect to time and another is based on some control parameters. Here, total vector population is divided into two subpopulations such as susceptible mosquito and infected mosquito as well as the total human population is divided into three subpopulations such as susceptible human, infected human and recovered human. The dynamical behaviors of the system associated with reproduction number with respect to control parameters have been investigated theoretically and numerically both. The biologically feasible equilibria and their stability properties have been discussed and the existence condition of the disease has been illustrated numerically. At last, Hopf bifurcation analysis has been done analytically and numerically for autonomous case of our proposed model.

Chapter 9: Stability and Bifurcation Analysis of Japanese Encephalitis Model with/without Effects of Some Control Parameters

In this chapter, a mathematical model on transmission of Japanese Encephalitis disease has been developed considering some control parameters and time dependent environmental carrying capacity. Here, total vector population is divided into two subpopulations such as susceptible mosquito and infected mosquito. Here also, total reservoir population (i.e., the population in which the encephalitis virus grows) such as pig, horse etc has been considered which is divided into three subpopulations such as susceptible reservoir, infected reservoir and recovered reservoir. Total human population is also divided into three subpopulations such as susceptible human, infected human and recovered human. The dynamical behaviors of the system have been investigated. Here, the reproduction number associated with the system has been analyzed with respect to control parameters theoretically and numerically both. The biological feasible equilibria and their stability properties have been discussed and the existence condition of the disease has been illustrated numerically. For a certain set of parametric values, the effectiveness of control parameters in our proposed model has been checked numerically. At last, Hopf bifurcations have been made numerically without considering control parameters for the case of constant environmental carrying capacity of mosquito.

Part IV
(Summary, Extension and Bibliography)
This part is divided into two chapters (Chapter 10 and 11) and in those chapters summary, future research works and bibliography have been presented.

Chapter 10: Summary and Future Research Work

In this chapter, a summary of the thesis and the scope of future research work have been discussed.

Chapter 11: Bibliography

In this chapter, the references have been presented.

Part II

Studies on Dynamics in Some Ecological Problems

Chapter 2

Stability Analysis of Coexistence of Three Species Prey-Predator Model

2.1 Introduction

The study of dynamics of prey-predator systems is one of the dominant subjects in mathematical ecology due to its universal existence and importance. Thus prey-predator models have been in the focus of ecological science from the early days. It has been turned out very soon that prey-predator system can show different dynamical behaviors such as steady-states, oscillations, bifurcations [36, 68, 136, 143, 187, 241, 245] depending of the model parameters. This system has been studied by many mathematicians and ecologists [50, 168] in population dynamics.

Planktons are microscopic organisms that float freely with oceanic currents and in other bodies of water. They are made by tiny plant (called Phytoplankton) and tiny animals (called Zooplankton). It is observed that Phytoplankton are primary producer. They prepare carbohydrates using energy from sunlight, inorganic chemicals and dissolved carbon dioxide gas with the help of chlorophyll [63]. They are consumed by Zooplankton which are microscopic animals and also a most favorable food source for Fish and other aquatic animals. Phytoplankton are normally present within marine and fresh waters in low concentrations but may proliferate to form dense concentrations of cell on water surfaces referred as "blooms". The high concentrations of pigment containing Phytoplankton may impart on color to the water resulting in their description as "red tides", "brown tides" etc. Harmful Algal Blooms (HAB) [205] may also occur on the ocean bottom caused by either microscopic or macroscopic algal species. Harmful Algal Blooms (HAB) are noxious to marine ecosystems or to human health and can produce great socioeconomic damage. There are many research papers on interaction of Phytoplankton

and Zooplankton [36, 102, 114, 146, 174, 184, 253, 257] in the prey-predator models.

Among several thousands of species of Phytoplankton, a few species produce toxin, such as Alexandrium sp., Amphidinium carterae, Chrysochromulina polylepis, Cooliamonotis, Dinophysis sp., Gambierdiscus toxicus, Gynnodinium breve, Gymodinium catenatum, Pseudo-nitzschina sp., Pyrodinium bahamense, Prymnesium patelliferum and P parvum. In 2002, Chattopadhyay et al. [1, 38] investigated that the toxin substance as well as the toxin producing Phytoplankton affect the growth of Zooplankton population and it has an impact on Phytoplankton and Zooplankton interaction. After that in 2006, Gakkhar and Negi [77] constructed a mathematical model of viral infection in the toxin producing Phytoplankton-Zooplankton system and they analyzed existence condition of equilibria, stability condition, bifurcation etc. Later in 2011, Chatterjee et al. [43] talked about the bottom up and top down effect on toxin producing Phytoplankton and the formation of the planktonic blooms. There are many mathematical models on the toxin producing Phytoplankton-Zooplankton interaction such as [196, 207] etc.

It is seen that the ecological system is often deeply perturbed by human exploiting activities. During the past half century, the rapid technological advances and the significant increases in human population have been occurred. Henceforth, the amount of world fishes has been greatly reduced. The trophic interactions of food webs are significantly dependent on the capacity of predators to find, kill and consume prey population. It plays an important role in shaping of entire ecosystem. The success rate of individual predator depends on several factors which include the existence of the populations. The most important component in such models is the density of prey that determines the functional response [177, 178].

From the above literature review, it is observed that many investigations on the predator prey dynamics have been done. In spite of that, till now there exists some lacunas in the study of prey predator dynamics such as:

Generally, it is seen that Fish provides a good source of high quality protein and it also contains many vitamins and minerals. For this reason, it is consumed as a food by many species including human being throughout the world. So, the study of existence for Fish population is very much essential in our society. Again, the existence of Fish populations naturally depends on Phytoplankton and Zooplankton. From the literature survey, it is seen that there are many papers on interaction between Phytoplankton and Zooplankton. But till now, no one has studied the interaction among Phytoplankton, Zooplankton and Fish population together.

To remove above lacunas a prey-predator system for three species such as Phytoplankton, Zooplankton and Fish has been developed in this chapter. Here, it is assumed that Fish population consumes both Phytoplankton and Zooplankton. Also, it is assumed that Zooplankton consumes only Phytoplankton. Therefore, the speciality in this chap-

ter is that Zooplankton act as a predator in one side as well as a prey in another side. And Phytoplankton is purely prey and Fish is purely predator. In this chapter, our objective is to study the dynamical behaviors of these three species which are theoretically beneficial to maintain the sustainable development of a prey-predator system. The sufficient condition for stability of the system has also been established at the positive equilibrium point using Routh Hurwitz criterion and then condition of Hopf bifurcation also be investigated. Finally, the numerical results have been provided to study more realistic features of the proposed model.

2.2 Model Formulation [1]

In this chapter, the co-existence of three species such as Phytoplankton, Zooplankton and Fish as a prey and predator has been considered. To develop this model the following assumptions have been made

(i) $P(t)$, $Z(t)$ and $F(t)$ denote respectively the density of toxin producing Phytoplankton, Zooplankton and Fish at any instant of time t subject to the non-negative initial conditions $P(0){=}P_0 \geq 0$, $Z(0){=}Z_0 \geq 0$ and $F(0){=}F_0 \geq 0$.

(ii) The parameter r and K be the intrinsic growth rate and the environmental carrying capacity of Phytoplankton population and it is also assumed that the growth of Phytoplankton is logistic.

(iii) The parameter ρ denotes the rate of releasing the toxic substances produced by per unit biomass of Phytoplankton.

(iv) The constant β, β_1 and d be the maximum uptake rate, the ratio of biomass conservation and the natural death rate for Zooplankton species respectively where $0 < \beta_1 < \beta$.

(v) The constants γ and γ_1 denote the maximum uptake rate for Phytoplankton and Zooplankton by Fish population respectively.

(vi) The parameters S and S_1 be the ratio of biomass conservation of Fish population for Phytoplankton and Zooplankton respectively where $S < \gamma$ and $S_1 < \gamma_1$.

(vii) The parameter δ is the natural death rate of Fish population.

($viii$) The term $\frac{\beta P Z}{\alpha + P}$ represents the functional response for grazing of Phytoplankton by Zooplankton where α is the half saturation constant for a Holling type II functional

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[1]Published in **Nonlinear Dynamics**, Springer, vol-81, 2015, 373-382, with title *Stability analysis of coexistence of three species prey-predator model.*

response.

(ix) Since Phytoplankton is consumed by Zooplankton, hence the rate of growth of Zooplankton is proportional to consumption rate i.e., the functional response for Zooplankton is $\frac{\beta_1 PZ}{\alpha+P}$. Again since some Phytoplankton produces toxin, hence it is assumed that the rate of death of Zooplankton is proportional to rate of consumption of Phytoplankton i.e., rate of death of Zooplankton is $\frac{\rho PZ}{\alpha+P}$. Obviously for existence of population of Phytoplankton, β_1 must be greater than ρ.

(x) The terms $\frac{\gamma PF}{\alpha+P}$ and $\frac{\gamma_1 ZF}{\alpha+P}$ represent the functional responses for grazing Phytoplankton and Zooplankton by Fish population respectively.

Under the above mentioned assumptions, a mathematical model for coexistence of these three species has been developed as follows:

$$\left.\begin{array}{l} \dfrac{dP}{dt} = rP(1-\dfrac{P}{K}) - \dfrac{\beta PZ}{\alpha+P} - \dfrac{\gamma PF}{\alpha+P} \\[3mm] \dfrac{dZ}{dt} = \dfrac{\beta_1 PZ}{\alpha+P} - dZ - \dfrac{\rho PZ}{\alpha+P} - \dfrac{\gamma_1 ZF}{\alpha+P} \\[3mm] \dfrac{dF}{dt} = \dfrac{SPF}{\alpha+P} + \dfrac{S_1 ZF}{\alpha+P} - \delta F \end{array}\right\} \qquad (2.1)$$

2.3 Boundedness of Solutions

In this section, uniform boundedness of our proposed system has been presented.

Theorem 2.1 All Solutions of system (2.1) which initiate in R_+^3 are uniformly bounded for suitably chosen positive parameter ξ such that $\xi \leq min(\delta, d)$ and $\gamma_1 S \geq \gamma S_1$.

Proof. To get boundedness of solutions of system (2.1) a function W is defined as follows

$$W = P + Z + \frac{\gamma}{S}F.$$

Now, differentiating the above equation with respect to time t it is obtained that

$$\begin{aligned} \frac{dW}{dt} &= \frac{dP}{dt} + \frac{dZ}{dt} + \frac{\gamma}{S}\frac{dF}{dt} \\ &= rP(1-\frac{P}{K}) - \frac{\beta PZ}{\alpha+P} - \frac{\gamma PF}{\alpha+P} + \frac{\beta_1 PZ}{\alpha+P} - dZ - \frac{\rho PZ}{\alpha+P} - \frac{\gamma_1 FZ}{\alpha+P} + \frac{\gamma PF}{\alpha+P} \\ &\quad + \frac{\gamma S_1}{S}\frac{ZF}{\alpha+P} - \frac{\gamma}{S}\delta F. \end{aligned}$$

Here, introducing a positive constant ξ the above can be written as

$$\frac{dW}{dt} + \xi W \le \frac{K}{4r}(r+\xi)^2 - (d-\xi)Z - \frac{\gamma}{S}(\delta-\xi)F - (\gamma_1 - \frac{\gamma S_1}{S})\frac{ZF}{\alpha+P}.$$

$$i.e., \frac{dW}{dt} + \xi W \le \frac{K}{4r}(r+\xi)^2. \tag{2.2}$$

provided that $\xi \le min(\delta, d)$ and $\gamma_1 S \ge \gamma S_1$.
Now, solving the above equation (2.2) using theory of differential inequality the following is obtained

$$0 < W(P, Z, F) < \frac{M}{\xi}(1 - e^{-\xi t}) + W(P(0), Z(0), F(0))e^{-\xi t}.$$

where $M = \frac{K(r+\xi)^2}{4r}$.
Now, taking limit of the above inequality as t tends to infinity, it is obtained that

$$W(P, Z, F) < \frac{M}{\xi}.$$

From this, it is concluded that the solution of the system lies in the region

$$\Im = [(P, Z, F)\varepsilon R_+^3 : W = \frac{M}{\xi} + \epsilon, \text{ for any } \epsilon > 0].$$

2.4 Equilibrium Points

To study the stability of the proposed model the equilibrium points in the system (2.1) are necessary to calculate. Now, the possible equilibrium points of this system are given by

(i) The trivial equilibrium point $E_0 = (0, 0, 0)$.

(ii) The equilibrium point $E_1 = (K, 0, 0)$ on the boundary of the first octant.

(iii) The planer equilibrium point $E_2 = (\bar{P}, \bar{Z}, 0)$ on the $P - Z$ plane where $\bar{P} = \frac{d\alpha}{\beta_1 - \rho - d}$ and $\bar{Z} = \frac{r\alpha(\beta_1 - \rho)(K\beta_1 - K\rho - Kd - d\alpha)}{K\beta(\beta_1 - \rho - d)^2}$.

(iv) The another equilibrium point $E_3 = (P', 0, F')$ on the $P - F$ plane where
$P' = \frac{\delta\alpha}{S-\delta} > 0$ and $F' = \frac{r(\alpha+P')(1-\frac{P'}{K})}{\gamma} > 0$.

(v) The positive equilibrium point $E^* = (P^*, Z^*, F^*)$ where it is obtained as follows:

Now, P^* corresponds to a positive root of the following quadratic equation

$$f(P^*) = P^{*^2} + AP^* + B = 0$$

where $A = \frac{1}{rS_1\gamma_1}(\beta K\delta\gamma_1 - \beta KS\gamma_1 + r\alpha S_1\gamma_1 - rKS_1\gamma_1 + \gamma K\beta_1 S_1 - \gamma KdS_1 - \gamma K\rho S_1)$ and
$B = \frac{1}{rS_1\gamma_1}(\beta K\delta\alpha\gamma_1 - Kr\alpha S_1\gamma_1 - \gamma Kd\alpha S_1)$.
The roots of this quadratic equation are given by

$$P^* = \frac{-A \pm \sqrt{A^2 - 4B}}{2}$$

So, for positive equilibrium points E^*, P^* admits at least one positive value when any
one of the following cases is satisfied

(i) $A < 0$ and $B < 0$.

(ii) $A < 0$, $B > 0$ and $A^2 - 4B > 0$.

(iii) $A > 0, B < 0$.

Again, for positiveness of Z^* and F^* the following relations must be holds

$$Z^* = \frac{\delta\alpha - (S - \delta)P^*}{S_1} > 0$$

$$F^* = \frac{P^*(\beta_1 - d - \rho) - d\alpha}{\gamma_1} > 0$$

Now, from the above inequalities it is obtained that

$$\frac{d\alpha}{\beta_1 - d - \rho} < P^* < \frac{\delta\alpha}{S - \delta}$$

which is the required condition for positive equilibrium point E^*.

2.5 Stability Analysis

Let us consider the stability analysis of the deterministic differential equation (2.1) gov-
erning the evolution of the system. The stability of the equilibrium state is determined
by the nature of the eigenvalues of the variational matrix around the point $E(P, Z, F)$,
we get

$$V(P,Z,F) = \begin{pmatrix} r - \frac{2rP}{K} - \frac{\alpha\beta Z}{(\alpha+P)^2} - \frac{\gamma\alpha F}{(\alpha+P)^2} & -\frac{\beta P}{\alpha+P} & -\frac{\gamma P}{\alpha+P} \\ \frac{\beta_1\alpha Z}{(\alpha+P)^2} - \frac{\rho\alpha Z}{(\alpha+P)^2} + \frac{\gamma_1 ZF}{(\alpha+P)^2} & \frac{\beta_1 P}{\alpha+P} - d - \frac{\rho P}{\alpha+P} - \frac{\gamma_1 F}{\alpha+P} & -\frac{\gamma_1 Z}{\alpha+P} \\ \frac{S\alpha F}{(\alpha+P)^2} - \frac{S_1 ZF}{(\alpha+P)^2} & \frac{S_1 F}{\alpha+P} & \frac{SP}{\alpha+P} + \frac{S_1 Z}{\alpha+P} - \delta \end{pmatrix}$$

Theorem 2.2 The trivial equilibrium point E_0 is always unstable.

Proof. The variational matrix for E_0 is

$$V_{E_0} = \begin{pmatrix} r & 0 & 0 \\ 0 & -d & 0 \\ 0 & 0 & -\delta \end{pmatrix}$$

It has eigenvalues $r, -d$ $and -\delta$ which shows that E_0 is the saddle point. It is stable in the direction of Z and F and unstable in the S direction.

Theorem 2.3 The boundary equilibrium point E_1 is Locally asymptotically stable if there exist two critical parameters R_0 and R_1 such that $R_0 < 1$ and $R_1 < 1$ where $R_0 = \frac{K(\beta_1 - \rho - d)}{d\alpha}$ and $R_1 = \frac{SK}{\delta(\alpha + K)}$.

Proof. Now, the variational matrix V_{E_1} for the boundary equilibrium point E_1 is given by

$$V_{E_1} = \begin{pmatrix} -r & -\frac{\beta K}{\alpha + K} & -\frac{\gamma K}{\alpha + K} \\ 0 & \frac{\beta_1 K}{\alpha + K} - d - \frac{\rho K}{\alpha + K} & 0 \\ 0 & 0 & \frac{sK}{\alpha + K} - \delta \end{pmatrix}$$

Here, the eigenvalues are $\lambda_1 = -r, \lambda_2 = \frac{\beta_1 K}{\alpha + K} - d - \frac{\rho K}{\alpha + K}$ and $\lambda_3 = \frac{SK}{\alpha + K} - \delta$. Now, it is known that E_1 is locally asymptotically stable when three eigenvalues λ_1, λ_2 and λ_3 must be less than zero. Here, it is seen that $\lambda_1 = -r < 0$, since $r > 0$. So, for existence of stability for E_1, $\lambda_2 < 0$ and also $\lambda_3 < 0$

$$i.e., \frac{\beta_1 K}{\alpha + K} - d - \frac{\rho K}{\alpha + K} < 0 \text{ and } \frac{SK}{\alpha + K} - \delta < 0$$

$$i.e., \frac{K(\beta_1 - \rho - d) - d\alpha}{\alpha + K} < 0 \text{ and } \frac{KS - \delta(K + \alpha)}{\alpha + K} < 0$$

$$i.e., \frac{K(\beta_1 - \rho - d)}{d\alpha} < 1 \text{ and } \frac{KS}{\delta(\alpha + K)} < 1$$

$$i.e., R_0 < 1 \text{ and } R_1 < 1$$

where $R_0 = \frac{K(\beta_1 - \rho - d)}{d\alpha}$ and $R_1 = \frac{KS}{\delta(\alpha + K)}$ are known as critical parameters.

Theorem 2.4 The planer equilibrium point E_2 exists if $R_0 > 1$ and it is locally asymptotically stable if $B_1, B_3 > 0$ and $B_1 B_2 > B_3$ and otherwise it is unstable.

Proof. Now, the variational matrix V_{E_2} for the equilibrium point $E_2(\bar{P}, \bar{Z}, 0)$ is given by

$$V_{E_2} = \begin{pmatrix} b_1 & -b_2 & -b_3 \\ b_4 & b_5 & -b_6 \\ 0 & 0 & b_7 \end{pmatrix}$$

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where $b_1 = r - \frac{2r\bar{P}}{K} - \frac{\alpha\beta\bar{Z}}{(\alpha+\bar{P})^2}, b_2 = \frac{\beta\bar{P}}{\alpha+\bar{P}}, b_3 = \frac{\gamma\bar{P}}{\alpha+\bar{P}}, b_4 = \frac{\beta_1\alpha\bar{Z}}{(\alpha+\bar{P})^2} - \frac{\rho\alpha\bar{Z}}{(\alpha+\bar{P})^2},$
$b_5 = \frac{\beta_1\bar{P}}{\alpha+\bar{P}} - d - \frac{\rho\bar{P}}{\alpha+\bar{P}}, b_6 = \frac{\gamma_1\bar{Z}}{\alpha+\bar{P}}$ and $b_7 = \frac{S\bar{P}}{\alpha+\bar{P}} + \frac{S_1\bar{Z}}{\alpha+\bar{P}} - \delta$
Then, the characteristic equation for V_{E_2} is

$$x^3 + B_1 x^2 + B_2 x + B_3 = 0.$$

where $B_1 = -(b_1 + b_5 + b_7), B_2 = b_1 b_7 + b_5 b_7 + b_1 b_5 + b_2 b_4, B_3 = -b_1 b_5 b_7 - b_2 b_4 b_7.$
Now, E_2 exists if $R_0 > 1$ and by Routh-Hurwitz criteria, it will be locally asymptotically stable i.e., the eigenvalues of the characteristic equation will be negative real parts if $B_1, B_3 > 0$ and $B_1 B_2 - B_3 > 0$ and unstable otherwise.

Theorem 2.5 Another planer equilibrium point E_3 is locally asymptotically stable if $B'_1, B'_3 > 0$ and $B'_1 B'_2 - B'_3 > 0$ and unstable otherwise.

Proof. The variational matrix V_{E_3} for the planer equilibrium point $E_3 = (P', 0, F')$ is given by

$$V_{E_3} = \begin{pmatrix} b_{11} & -b_{12} & -b_{13} \\ 0 & b_{22} & 0 \\ b_{31} & b_{32} & b_{33} \end{pmatrix}$$

where $b_{11} = r - \frac{2rP'}{K} - \frac{\gamma\alpha F'}{(\alpha+P')^2}, b_{12} = \frac{\beta P'}{\alpha+P'}, b_{13} = \frac{\gamma P'}{\alpha+P'}, b_{22} = \frac{\beta_1 P'}{\alpha+P'} - d - \frac{\rho P'}{\alpha+P'} - \frac{\gamma_1 F'}{\alpha+P'},$
$b_{31} = \frac{S\alpha F'}{(\alpha+P')^2}, b_{32} = \frac{S_1 F'}{\alpha+P'}$ and $b_{33} = \frac{SP'}{\alpha+P'} - \delta.$
The characteristic equation for V_{E_3} is

$$y^3 + B'_1 y^2 + B'_2 y + B'_3 = 0.$$

where $B'_1 = -(b_{11} + b_{22} + b_{33}), B'_2 = b_{11}b_{33} + b_{22}b_{33} + b_{11}b_{22} + b_{13}b_{31}$ and $B'_3 = -b_{11}b_{22}b_{33} - b_{13}b_{31}b_{22}.$ Now, E_3 exists if $R_0 > 1$ and by Routh-Hurwith criteria, the system will be locally asymptotically stable i.e., the eigenvalues will be negative real parts if $B'_1, B'_3 > 0$ and $B'_1 B'_2 - B'_3 > 0$. Otherwise the system will be unstable.

Theorem 2.6 The positive equilibrium point E^* exists if $R_0 > 1$ and it is Locally asymptotically stable if $\sigma_1, \sigma_3 > 0$ and $\sigma_1 \sigma_2 > \sigma_3$ and otherwise it is unstable.

Proof. The variational matrix for $E^* = (P^*, Z^*, F^*)$ is

$$V_{E^*} = \begin{pmatrix} \sigma_{11} & -\sigma_{12} & -\sigma_{13} \\ \sigma_{21} & \sigma_{22} & -\sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} \end{pmatrix}$$

where $\sigma_{11} = r - \frac{2rP^*}{K} - \frac{\alpha\beta Z^*}{(\alpha+P^*)^2} - \frac{\gamma\alpha F^*}{(\alpha+P^*)^2}, \sigma_{12} = \frac{\gamma\alpha F^*}{(\alpha+P^*)^2}, \sigma_{13} = \frac{\gamma P^*}{\alpha+P^*},$
$\sigma_{21} = \frac{\beta_1\alpha Z^*}{(\alpha+P^*)^2} - \frac{\rho\alpha Z^*}{(\alpha+P^*)^2} + \frac{\gamma_1 Z^* F^*}{(\alpha+P^*)^2}, \sigma_{22} = \frac{\beta_1 P^*}{(\alpha+P^*)} - d - \frac{\rho P^*}{(\alpha+P^*)} - \frac{\gamma_1 F^*}{(\alpha+P^*)}, \sigma_{23} = \frac{\gamma_1 Z^*}{\alpha+P^*},$

$\sigma_{31} = \frac{S\alpha F^*}{(\alpha+P^*)^2} - \frac{S_1 Z^* F^*}{(\alpha+P^*)^2}, \sigma_{32} = \frac{S_1 F^*}{\alpha+P^*}$ and $\sigma_{33} = \frac{SP^*}{\alpha+P^*} + \frac{S_1 Z^*}{\alpha+P^*} - \delta$.
Now, the characteristic equation of the variational matrix V_{E^*} is

$$x^3 + \sigma_1 x^2 + \sigma_2 x + \sigma_3 = 0.$$

where $\sigma_1 = -(\sigma_{11} + \sigma_{22} + \sigma_{33}), \sigma_2 = \sigma_{22}\sigma_{33} + \sigma_{23}\sigma_{32} + \sigma_{11}\sigma_{22} + \sigma_{11}\sigma_{33} + \sigma_{12}\sigma_{21} + \sigma_{13}\sigma_{31}$
and $\sigma_3 = -\sigma_{11}\sigma_{22}\sigma_{33} - \sigma_{11}\sigma_{23}\sigma_{32} - \sigma_{12}\sigma_{31}\sigma_{23} - \sigma_{12}\sigma_{21}\sigma_{33} - \sigma_{31}\sigma_{22}\sigma_{13} + \sigma_{13}\sigma_{21}\sigma_{32}$. Now,
E^* exists if $R_0 > 1$ and by using Hurwitz criteria, it will be locally asymptotically stable
if:
(i) $\sigma_1, \sigma_3 > 0$ (ii) $\sigma_1 \sigma_2 - \sigma_3 > 0$ holds.
Otherwise E^* is unstable.

Lemma 2.1 If $R_0 < 1$ then E_2, E_3 and E^* does not exists. Hence existence of E_2,
E_3 and E^* implies that E_1 is unstable saddle point.

2.6 Bifurcation Analysis

In prey-predator model many parameters are used for describing the system. Prey-predator models with constant parameters are often found to approach a steady state in which the species coexist in equilibrium. But if parameters used in the model are changed, other types of dynamical behavior may occur and the critical parameter values at which such transitions happen are called bifurcation points. The purpose of this study is to determine the stability behavior of the system in presence of different density dependent factors of the prey-predator interactions. To study the transition of the system with respect to small changes in the density dependent factors. A Hopf bifurcation occurs at points where the system has a non-hyperbolic equilibrium connected with a pair of purely imaginary eigenvalues, but no zero eigenvalues. We have considered β, ρ as the bifurcation parameters and β^* and ρ^* represent the critical value or the bifurcating value of the concerned bifurcation parameter.

Theorem 2.7 The positive equilibrium E^* enters into Hopf bifurcation as β varies over $\Re$. Let $\psi : (0, \infty) \to \Re$ be the following continuously differential function of β.

$$\psi(\beta) = C_1(\beta)C_2(\beta) - C_3(\beta).$$

Let β^*be a positive root of the equation $\psi(\beta) = 0$.
Therefore, the Hopf bifurcation of the interior equilibrium $E(P^*, Z^*, F^*)$ occurs at $\beta = \beta^*$
if and only if
(i) $\psi(\beta^*) = 0$
(ii) $L_2(\beta^*)L_4(\beta^*) + L_1(\beta^*)L_3(\beta^*) \neq 0$

Proof. By the condition $\psi(\beta) = 0$, then the characteristic equation of the variational

matrix V_{E^*} from **Theorem 2.6** can be written as

$$(x^2 + \sigma_2)(x + \sigma_1) = 0$$

The roots of the above equation are ρ_1, ρ_2, ρ_3(say). Let the pair of imaginary roots at $\beta = \beta^*$ are ρ_1, ρ_2 then we have $\rho_3 = -\sigma_1$ and $\rho_1, \rho_2 = \pm i \sqrt{(\sigma_2)}$.
As $\psi(\beta^*)$ is a continuous function of all its roots so there exists an open interval $(\beta^* - \epsilon, \beta^* + \epsilon)$ where ρ_1 and ρ_2 are complex conjugate for β. Suppose that their general forms in this neighborhood are

$$\rho_1(\beta) = \chi(\beta) + i\xi(\beta)$$
$$\rho_2(\beta) = \chi(\beta) - i\xi(\beta)$$

Now, we shall verify the transversality condition $\left(\frac{d(Re\rho_j)}{d\beta}\right)_{\beta=\beta^*} \neq 0$, $j = 1, 2$.
Substituting, $\rho_j(\beta) = \chi(\beta) \pm i\xi(\beta)$ into the characteristic equation and calculating the derivative, we have

$$L_1(\beta)\chi'(\beta) - L_2(\beta)\xi'(\beta) + L_3(\beta) = 0$$
$$L_2(\beta)\chi'(\beta) + L_1(\beta)\xi'(\beta) + L_4(\beta) = 0$$

where

$$L_1(\beta) = 3\chi^2 - 3\xi^2 + 2\sigma_1\chi + \sigma_2$$
$$L_2(\beta) = 6\chi\xi + 2\sigma_1\xi$$
$$L_3(\beta) = \sigma_1'\chi^2 - \sigma_1'\xi^2 + \sigma_2'\chi + \sigma_3'$$
$$L_4(\beta) = 2\sigma_1'\chi\xi + \sigma_2'\xi$$

Solving for $\xi'(\beta)$ at $\beta = \beta^*$, we have

$$\left(\frac{d(Re\rho_j(\beta))}{d\beta}\right)_{\beta=\beta^*} = \chi'(\beta^*) = -\frac{L_2(\beta^*)L_4(\beta^*) + L_1(\beta^*)L_3(\beta^*)}{L_1^2(\beta^*) + L_2^2(\beta^*)} \neq 0$$

if $L_2(\beta^*)L_4(\beta^*) + L_1(\beta^*)L_3(\beta^*) \neq 0$. Thus the transversality condition holds and hence Hobf-bifurcation occurs at $\beta = \beta^*$. Hence the theorem.

Theorem 2.8 At its positive equilibrium point E^* for the parameter ρ the system also undergoes a bifurcation.

Proof. The proof of this theorem is similar as the proof of **Theorem 2.7.**

2.7 Global Stability Analysis

In this section, we now perform a global stability analysis of the proposed system (2.1) around the equilibrium point $E^*(P^*, Z^*, F^*)$.

Theorem 2.9 Let $U = \frac{(P-P^*)^2}{2} + \delta_1 \frac{(Z-Z^*)^2}{2} + \delta_2 \frac{(F-F^*)^2}{2}$ where $\delta_1, \delta_2 > 0$ are to be chosen properly such that $U'(E^*) = 0$ where $E^*(P^*, Z^*, F^*)$ and $U = (P, Z, F) > 0, \forall P, Z, F/E^*$. The time derivative of U is $\frac{dU}{dt} \leq 0, \forall P, Z, F \in \Gamma^+$. It then follows that $\frac{dU}{dt} = 0, \forall P^*, Z^*, F^* \in \Gamma^+$ implies that E^* of the system is Lyapunov stable and $\frac{dU}{dt} < 0 \ \forall P, Z, F \in \Gamma^+$ near E^* implies that E^* is globally stable.

Proof. Let us consider a function of the form

$$U = \frac{(P - P^*)^2}{2} + \delta_1 \frac{(Z - Z^*)^2}{2} + \delta_2 \frac{(F - F^*)^2}{2} \tag{2.3}$$

Taking time derivative of the above equation (2.3) we get

$$\frac{dU}{dt} = (P - P^*)\frac{dP}{dt} + \delta_1(Z - Z^*)\frac{dZ}{dt} + \delta_2(F - F^*)\frac{dF}{dt}$$

Now, substituting the value of $\frac{dP}{dt}, \frac{dZ}{dt}$ and $\frac{dF}{dt}$ from the model system (2.1) and putting these values in the above equation we get

$$
\begin{aligned}
\frac{dU}{dt} &= (P - P^*)\left[rP(1 - P/K) - \frac{\beta PZ}{\alpha + P} - \frac{\gamma PF}{\alpha + P} \right] \\
&+ \delta_1(Z - Z^*)\left[\frac{\beta_1 PZ}{\alpha + P} - dZ - \frac{\rho PZ}{\alpha + P} - \frac{\gamma_1 ZF}{\alpha + P} \right] \\
&+ \delta_2(F - F^*)\left[\frac{SPF}{\alpha + P} + \frac{S_1 ZF}{\alpha + P} - \delta F \right] \\
&= (P - P^*)\left[\left(r(1 - P/K) - \frac{\beta Z}{\alpha + P} - \frac{\gamma F}{\alpha + P} \right)(P - P^*) \right] \\
&+ \delta_1(Z - Z^*)\left[\left(\frac{\beta_1 P}{\alpha + P} - d - \frac{\rho P}{\alpha + P} - \frac{\gamma_1 F}{\alpha + P} \right)(Z - Z^*) \right] \\
&+ \delta_2(F - F^*)\left[\left(\frac{SP}{\alpha + P} + \frac{S_1 Z}{\alpha + P} - \delta \right)(F - F^*) \right]
\end{aligned}
$$

By rearranging, we obtain

$$
\begin{aligned}
\frac{dU}{dt} &= -(P - P^*)^2 \left[r(-1 + P/K) + \frac{\beta Z}{\alpha + P} + \frac{\gamma F}{\alpha + P} \right] \\
&- \delta_1(Z - Z^*)^2 \left[-\frac{\beta_1 P}{\alpha + P} + d + \frac{\rho P}{\alpha + P} + \frac{\gamma_1 F}{\alpha + P} \right] \\
&- \delta_2(F - F^*)^2 \left[-\frac{SP}{\alpha + P} - \frac{S_1 Z}{\alpha + P} + \delta \right]
\end{aligned}
$$

Thus, it is possible to set δ_1, δ_2 such that $\frac{dU}{dt} \leq 0$ and the equilibrium point E^* is globally asymptotically stable.

2.8   Numerical Simulation

In this section, the dynamical behavior of the proposed model (2.1) has been discussed numerically using MATLAB. Due to unavailability of real data of all parameters associated with the model, the hypothetical values to the different parameters have been considered as follows:

$r = 6, K = 10.0, \gamma = 0.3, \beta_1 = 0.2, d = 0.3, \rho = 0.2, \gamma_1 = 0.4, S = 0.28, S_1 = 0.35, \delta = 0.28, \alpha = 0.2, \beta = 0.5$.

Now, for this data it is seen that $R_0 = -50.0 < 1$ and $R_1 = -0.005 < 1$. So, according to **Theorem** 2.3 the boundary equilibrium point $E_1 = (10.0, 0.0, 0.0)$ is locally asymptotically stable. Again, Figure 2.1 also shows that there exists a stable equilibrium point E_1 and though there exists Phytoplankton in the ecosystem then due to diminishing of Zooplankton gradually Fish population is also decreasing. It is concluded that if there is only Phytoplankton in the ecosystem and no Zooplankton then the Fish population can be extinct eventually.
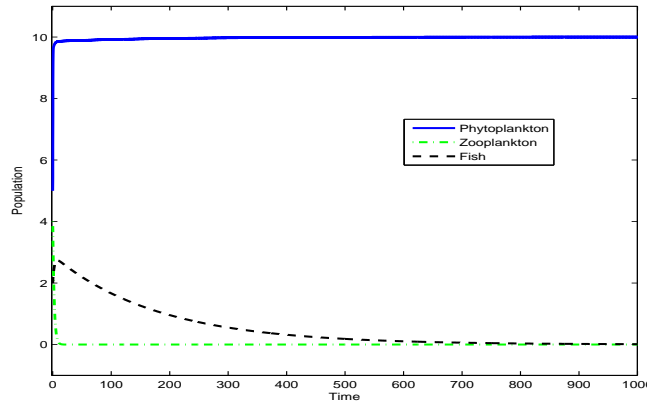

Figure 2.1: Stability of the equilibrium point E_1.

Again, for the set of parametric values $r = 8, K = 1.6, \gamma = 0.5, \beta_1 = 0.4, d = 0.1, \rho = 0.1, \gamma_1 = 0.4, S = 0.4, S_1 = 0.3, \delta = 0.2, \alpha = 1.0, \beta = 1.0$ it is seen that $R_0 = 3.2 > 1$, $B_1' = 5.8500 > 0, B_3' = 0.7050 > 0$ and $B_1'B_2' - B_3' = 49.1663 > 0$, according to **Theorem 2.4** we have a stable equilibrium point $E_3 = (1.00, 0.0, 12.0)$. From Figure 2.2, it is seen that E_3 is locally asymptotically stable and it is concluded that the Fish population can be exists and increased constantly when there is a Phytoplankton in the ecosystem and no Zooplankton in the ecosystem.
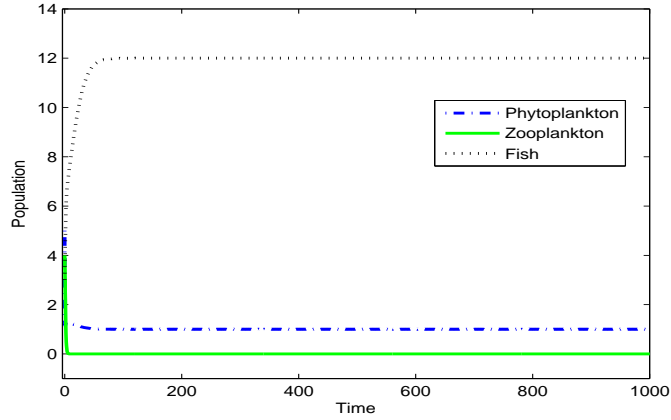
Figure 2.2: Stability of the equilibrium point E_3.

Also, it is considered the following set of parametric values such as $r = 1.5, K = 120.0, \gamma = 0.5, \beta_1 = 0.6, d = 0.3654, \rho = 0.2, \gamma_1 = 0.6, S = 0.4, S_1 = 0.5, \delta = 0.5, \alpha = 1.0, \beta_1 = 0.68$. For these parametric values, it is seen that $R_0 = 37.635 > 1$, $\sigma_1 = 1.5606 > 0$, $\sigma_3 = 0.0028 > 0$ and $\sigma_1\sigma_2 - \sigma_3 = 0.7138 > 0$, so according to the **Theorem 2.6** the positive equilibrium point E^* is locally asymptotically stable. Also, Figure 2.3 shows a locally asymptotically stable positive equilibrium point $E^* = (106.6568, 22.3314, 5.5415)$ and from this figure, it is concluded that Phytoplankton, Zooplankton and Fish population co-exist simultaneously.
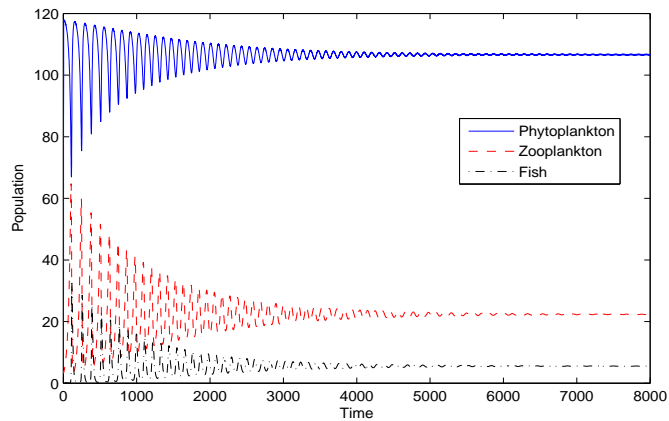

Figure 2.3: Stability of the positive equilibrium point E^*.

Again, for the parametric values $r = 3.0, K = 3.0, \gamma = 0.5, \beta_1 = 0.94, d = 0.3, \rho = 0.1, \gamma_1 = 0.6, S = 0.4, S_1 = 0.5, \delta = 0.5, \alpha = 1.0, \beta = 1.0$, the Figure 2.4 and Figure 2.5 both show phase space in which populations are tending towards the fixed point.
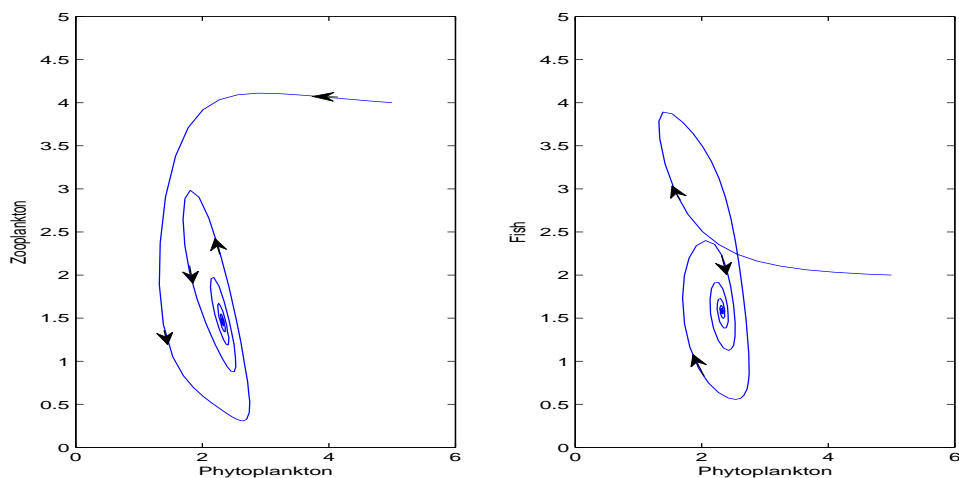
Figure 2.4: Phase space diagram of Phytoplankton-Zooplankton and Phytoplankton-Fish.
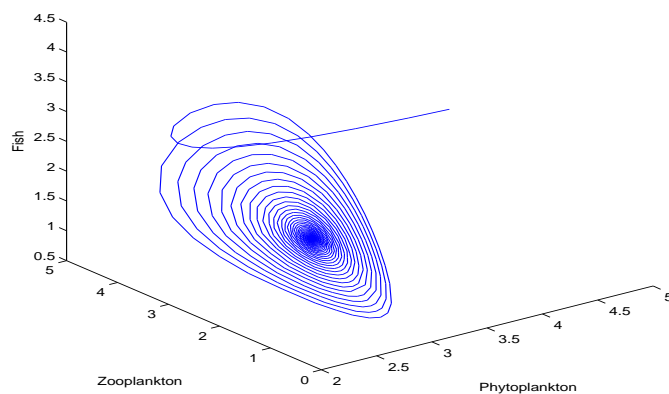
Figure 2.5: Phase space diagram of Phytoplankton-Zooplankton and Fish.

Now, the following parametric values have been considered to discuss the dynamical behavior of our proposed model taking β as a bifurcation parameter.
$r = 1.5, K = 150.0, \gamma = 0.5, \beta_1 = 0.6, d = 0.39, \rho = 0.2, \gamma_1 = 0.6, S = 0.4, S_1 = 0.5, \delta = 0.5, \alpha = 1.0$.
Using this set of values, the critical value (β^*) of the consumption rate (β) of zooplankton is obtained as $\beta^* = 0.68$. So, when $\beta > \beta^*$ we have a unstable limit cycle around the

positive equilibrium point E^* according to **Theorem 2.7** and also, it is seen in Figure 2.6.
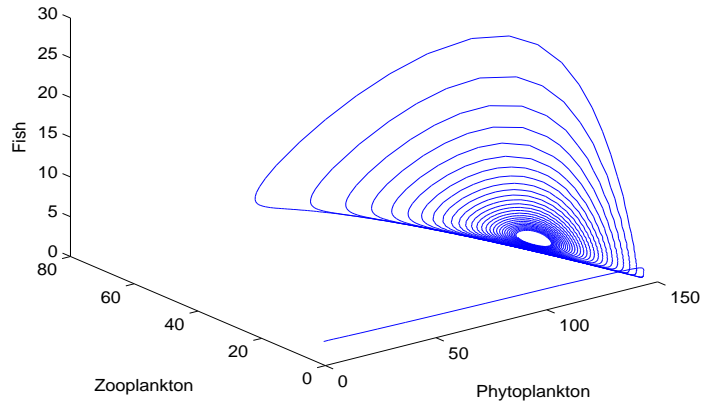
Figure 2.6: Phase space diagram of the system (2.1) for $\beta = 0.71$.

Similarly, the instability for the system (2.1) have been shown taking ρ **as the bifurcation parameter** with the help of same parametric values. Now, by **Theorem 2.8**., we can determine the critical value of ρ and it is $\rho^* = 0.21$. The system is unstable for $\rho > \rho^*$ around its interior equilibrium point E^*, taking $\rho = 0.22$ the solution curve to the system (2.1) has been shown in Figure 2.7 which indicates that the system is unstable around the interior equilibrium point E^*.

Figure 2.7: Phase space diagram of the system (2.1) for $\rho = 0.22$.

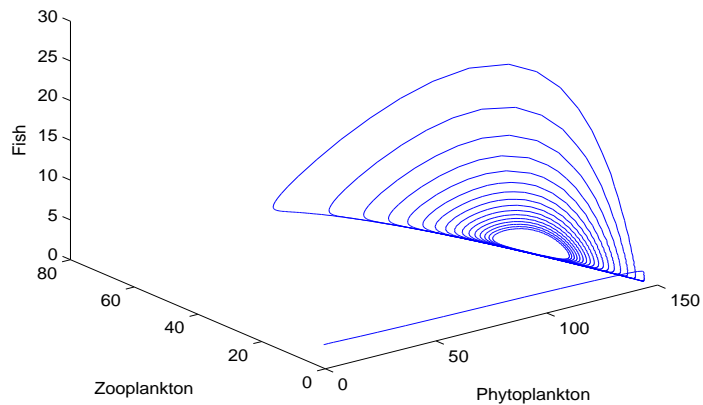Finally, for the parametric values $K = 10.0, \beta_1 = 0.2, d = 0.3, \rho = 0.2, \alpha = 0.2, \delta = 0.28, S = 0.28$, the changes of the critical parameters R_0 and R_1 with respect to the parameters associated with them have been shown in Figure 2.8. From this figure, it is observed that the value of R_0 decreases from zero when parameter K increases from zero and for this values of K, R_1 is nearly constant which is less than 1. Therefore, for all plausible values of K, R_0 and R_1 must be less than 1. Again, when parameter d increases from the initial value zero then the values of R_0 remains same which is -50 less than one. Also, it is also observed that when ρ increases from zero then R_0 decreases gradually from -16.67. But, when β_1 increases then R_0 also increases and it is seen that R_0 must be less than one for all values of β_1 which are less than 0.5. From this observation of sensitivity analysis, it is concluded that R_0 and R_1 must be less than one for all values of K, d, ρ and β_1 except β_1 is grater than equal to 0.5 i.e., the equilibrium point E_1 is locally asymptotically stable for all values of K, d, ρ and β_1 except β_1 is grater than equal to 0.5.
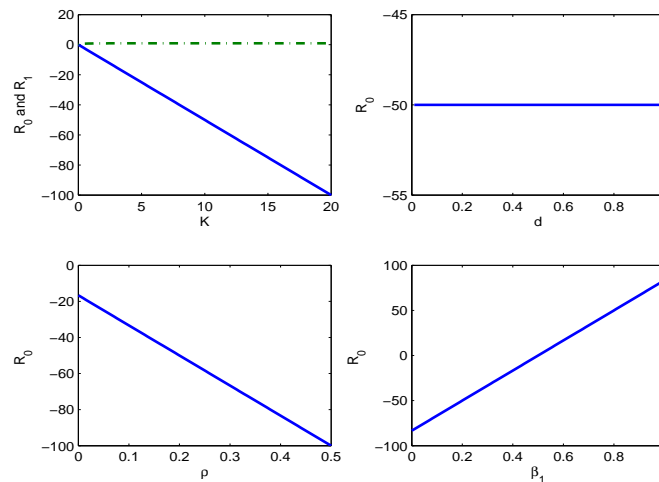
Figure 2.8: Sensitivity analysis of R_0 and R_1 where the solid line denotes R_0 and dash-dot line denotes R_1.

2.9 Conclusion

Now a days mathematical ecology becomes a highly demandable area of research for scientists, mathematician, ecologists etc. who works in the mathematical biology. They are devoted their time to find out the solutions of different ecological problems such as prey-predator interaction, existence and extinction condition of a species in natural environment and how different factors are influencing the dynamics of a population. This factors may be come due to different human activities on the nature and natural disas-

ters. In this chapter, we wanted to study a prey-predator mathematical model taking Phytoplankton, Zooplankton and Fish as separate population. This type of system can be seen in the ecosystems of ponds, lakes and marines etc.

In this chapter, we have thoroughly discussed analytically and numerically the dynamical behavior of our proposed Phytoplankton, Zooplankton and Fish interaction mathematical model. It is seen that the boundedness of solutions of our proposed system depends on death rates of both Zooplankton, Fish and also the uptake rates and conservation rates of Phytoplankton and Zooplankton by Fish species. Our study ensures the existence of five possible equilibria such as vanishing, Zooplankton and Fish free, Fish free, Zooplankton free and the coexistence one. From the stability analysis, it is seen that the trivial equilibrium is always unstable. The boundary equilibrium E_1 will be locally asymptotically stable if the two critical parameters $R_0 < 1$ and $R_1 < 1$. The planer equilibrium points E_2, E_3 and positive equilibrium E^* exists if the parametric value $R_0 > 1$. Then, we study Hopf bifurcation of the system with respect to two parametric values such as uptake rate β of Phytoplankton by Zooplankton and rate of releasing toxic substances ρ produced by unit biomass of Phytoplankton. From the numerical simulations, it is seen that for $\beta > \beta^*$ and $\rho > \rho^*$ the system shows unstable solution. So, it can be concluded that these two parameters have a significant role to stabilize the proposed system. Also, the global stability analysis of the interior equilibrium has been investigated and it will be globally asymptotically stable under certain condition. The sensitivity analysis of the two parameters R_0 and R_1 have been done. From the sensitivity analysis of the parameter R_0, it is shown that R_0 must be less than 1 when the parameters K, d, ρ and β_1 increases but R_0 must be grater than 1 when β_1 is grater than equal to 0.5. From this analysis, it can be concluded that R_0 and R_1 has important role for the local stability of equilibrium points.

Gateway from Chapter 2 to Chapter 3
In Chapter 2, we have studied some interaction dynamics among Phytoplankton, Zooplankton and Fish which is nothing but a food chain model. Stage-structure dynamics is an important phenomenon in population ecology. Chapter 3 is devoted to study the effects of anti-predator behavior of adult prey species on the dynamics of prey-predator system.

Chapter 3

Stability and Bifurcation Analysis of a Stage Structured Prey-Predator Model with Ratio-dependent Functional Response and Anti-predator Behavior of Adult Prey

3.1 Introduction

The study of interaction among different species and their surrounding natural environment is an important topic in theoretical ecology. At present not only researchers from biology but also researchers from other fields like economics, geology, applied mathematics, environmental science etc have shown their interests to investigate the interactions between two or more species. The systematic mathematical analysis can lead to better understanding of such type of interactions. Since the work of Lotka [138], various kinds of mathematical models on prey-predator interaction [77, 102, 175, 177, 250] have been explored to explain the relationship between prey and predator. It is natural that two or more species living in a common habitant are often attached to one another by

interacting in several ways. Choice of suitable growth rate is an important aspect in studying the interactions between a prey and its predator populations. However, in the existing literature, several mathematicians and theoretical ecologists have contributed their different conceptual notions about the growth rate of predator population. Normally, the rate of prey consumption by an average predator is known as functional response which can be classified as (*i*) prey dependent (*ii*) predator dependent and (*iii*) multi species dependent. In prey dependent, the functional response is affected by only prey population, in case of predator dependent, functional response can be determined by considering both predator and prey populations and in multi species dependent the species other than the focal predator and its prey influence the functional response. Traditionally in prey-predator mathematical models, the functional response has been considered depending upon density of prey population only. In 1989, Arditi and Ginzburg [10] suggested a ratio dependent functional response which is a particular type of predator dependence. Here, the response only depends on the ratio of prey population size to predator population size and it is quite better than prey dependent functional response. Although, the mathematical form of the ratio dependent functional responses are more complex than the other types, there are very few number of existing literature in prey-predator models in which these types of responses have been considered. In 2004, the ratio dependent functional response was considered by Fan and Li [66]. After that, Banerjee [21] developed a prey-predator model considering the ratio dependent functional response in 2010. In these two papers, a ratio dependency has been considered on Holling type II only. There are also some mathematical models [19, 75, 123] in which the ratio-dependent functional response has been considered to analyze those models. In 2013, Tewa et al. [234] studied the effects of Holling type II functional response on a disease induced prey-predator system. Xu and Li [254] investigated the impact of Hassell-Verley type functional response on a predator prey system in 2015. In 2016, Tripathi et al. [238] studied the effects of Crowley-Martin type functional response on a delayed induced prey-predator model.

Although biologists routinely label the animals as predators or prey, the ecological role of individuals is often far from clear. There are many examples [8, 48, 109, 182, 225] of role reversals in predators and prey, where an adult prey attacks vulnerable young predators. This implies that a juvenile prey that escapes from predation and becomes adult and then it can kill juvenile predators. The juvenile prey to adult prey results in behavioral changes later in life: after becoming adult, these prey kill juvenile predators at a faster rate than prey that had not been exposed. Anti-predator adaptations are mechanisms developed through evolution that assist prey organisms in their constant struggle against predators. Throughout the animal kingdom, adaptations have evolved for every stage of this struggle. There are very few mathematical models [230] in which anti-predator behaviors have been considered to analyze the nonlinear system.

In 2014, Pal and Mandal [173] studied a mathematical model with modified Leslie-Gower type prey-predator model with Beddington-DeAngelis functional response and strong Allee effect. After that Zhang et al. [261] investigated a diffusive prey-predator model with disease in the prey in 2014. Again, in the same year Pal et al. [171] reported the impact of omnivory and predator switching on a three species prey-predator model. Bhattacharya et al. [26] studied a disease induced prey-predator model with immune response on infected prey in 2014. In 2015, Cai et al. [30] studied the dynamics of Leslie-Gower type predator-prey model with the allee effects. Huang et al. [101] investigated the impact of environmental toxin on a prey-predator system in 2015. In the same year, M. Al-Omari [5] reported the effect of state dependent delay and harvesting on a stage structured prey-predator model. In 2016, Tang and Liu [231] studied Hopf bifurcation on a stage structured prey-predator system.

Basically there are few research articles available in the prey-predator system with anti-predator effects. But till now there exist some gaps in the literature which are as follows:

It is seen that in the existing literature, anti-predator behavior has been taken on whole prey species. But in the reality, it is not correct, because only adult prey can attack their vulnerable predators to save the infants from the predation of predator. Again, most of the research papers on mathematical modelling of prey-predator system the functional responses have been considered as prey dependent. But in many experimental works, it was showed that functional response has been depended upon both prey and predator.

To overcome these difficulties, a prey-predator model with stage-structured has been considered in this chapter. Here, we have developed a prey-predator system in which two prey species such as juvenile prey as well as adult prey and one predator species have been considered. There are very few papers on prey-predator mathematical model with anti-predator behavior. Anti-predator behavior are mechanisms developed through evolution that assist prey organisms in their constant struggle against predators. The existing paper considered the anti-predator behavior on the whole prey species [230]. But in reality, it is seen that adults prey attack their vulnerable predators and save the younger prey from predation. This motivated us to introduce the anti-predator behavior only on the adult prey population. Also, the ratio dependent functional response function has been considered along with the anti-predator behavior of adult prey population.

3.2 Model Formulation with Anti-predator Effects

In this work, we are going to discuss a prey-predator mathematical model with stage structured prey populations. In this model, it is assumed that the recruitment rate of juvenile prey is proportional to the density of existing adult prey population. It is also assumed that juvenile prey becomes adult prey after staying sometimes in the juvenile stage. But, the study of anti-predator behavior is very important in ecology due to

morphological changes and attack of adult prey. Now, according to the model developed by Tang and Xiao [230], it is seen that the growth rate of predator population has been decreased by a anti-predator behavioral term (ηxy) involving the densities of all prey populations. But from the literature survey [48, 109, 182], it is seen that only the adult prey can save itself from the attack of predator due to its morphological changes. In communal defense, adult prey groups actively defend themselves by attacking or mobbing a predator. Mobbing is the harassing of a predator by many prey animals. For example, red colobus monkeys exhibit mobbing when threatened by chimpanzees, a common predator [8, 225]. So, in this model, only the adult prey has been considered to reduce the growth rate of predators. Due to this reason, we consider a stage structured prey population with juvenile prey or immature prey and adult prey or matured prey with biomass densities $x(t)$ and $y(t)$ at any time t respectively. Here, $z(t)$ be the biomass density of predator population at time t. As we have assumed that, matured prey population is strong enough to bear an anti-predator characteristics, therefore, the anti-predator behavior is taken as a bilinear form of both the state variables y and z with η as the per capita rate of anti-predator behavior of adult prey to the predator population. In population ecology, the intra-specific competition is an interaction between the members of the same species competed for limited resource. Ruan et al. [194] investigated the effects of density dependent mortality on predator prey model. So, in this work α is taken as a death rate due of intra-specific competition between the adult prey species.

Again, in population dynamics, the functional response is very important to describe the actual nature of both prey and predator population. Now, for the traditional predator-prey model, the functional response depends upon only density of prey population. But according to Berrymen [25], the predator per capita growth rate should decline with its density also. Therefore, to satisfy the above both criteria a functional response should be a function of prey and predator both [10, 21]. In this regard, the following functional responses of predator for consuming juvenile prey and adult prey should be considered as

$$\frac{\beta_1 xz}{z + k_1 x + k_2 y} \text{ and } \frac{\beta_2 yz}{z + k_1 x + k_2 y}$$

respectively. Hence, considering above realistic criteria, a prey-predator model has been developed in this chapter as follows:

$$\left.\begin{aligned}
\frac{dx}{dt} &= \gamma y - \beta x - d_1 x - \frac{\beta_1 xz}{z + k_1 x + k_2 y} \\
\frac{dy}{dt} &= \beta x - d_2 y - \alpha y^2 - \frac{\beta_2 yz}{z + k_1 x + k_2 y} \\
\frac{dz}{dt} &= \frac{\mu \beta_1 xz}{z + k_1 x + k_2 y} + \frac{\mu_1 \beta_2 yz}{z + k_1 x + k_2 y} - d_3 z - \eta yz
\end{aligned}\right\} \tag{3.1}$$

with nonnegative initial conditions $x(0) \geq 0, y(0) \geq 0$ and $z(0) \geq 0$.

Here, the other parameters involved in the proposed model are described as follows:

- γ: recruitment rate of juvenile prey.

- β: portion of juvenile prey who becomes adult.

- β_1: predation rate of predator to the juvenile prey.

- β_2: predation rate of predator to the adult prey.

- μ: conservation rate of juvenile prey to the predator.

- μ_1: conservation rate of adult prey to the predator.

- η: rate of anti-predator behavior of adult prey to the predator.

- α: death rate of adult prey due to intra specific competition.

- d_1, d_2, d_3: natural death rates of juvenile prey, adult prey and predator respectively.

- k_1, k_2: saturation constants for the functional responses.

3.3 Boundedness of Solutions

In this section uniform boundedness of the solutions of our proposed system have been discussed.

Theorem 3.1 All solutions of the system (3.1) will be uniformly bounded in R_+^3 if $\mu \leq 1$, $\mu_1 \leq 1$ and $\delta = min\{d_1, d_3\}$.

Proof. Now, we construct a function

$$W = x + y + z.$$

Taking time derivative of W and putting the values of $\frac{dx}{dt}$, $\frac{dy}{dt}$ and $\frac{dz}{dt}$, we have

$$
\begin{aligned}
\frac{dW}{dt} &= \gamma y - \beta x - d_1 x - \frac{\beta_1 xz}{z + k_1 x + k_2 y} + \beta x - d_2 y - \alpha y^2 - \frac{\beta_2 yz}{z + k_1 x + k_2 y} \\
&+ \frac{\mu \beta_1 xz}{z + k_1 x + k_2 y} + \frac{\mu_1 \beta_2 yz}{z + k_1 x + k_2 y} - d_3 z - \eta yz. \\
\frac{dW}{dt} &= \gamma y - d_1 x - d_2 y - \alpha y^2 - d_3 z - \frac{\beta_1 xz}{z + k_1 x + k_2 y}(1 - \mu) \\
&- \frac{\beta_2 yz}{z + k_1 x + k_2 y}(1 - \mu_1) - \eta yz. \\
i.e., \frac{dW}{dt} &\leq \gamma y - d_1 x - d_2 y - \alpha y^2 - d_3 z - \eta yz, \text{ if } \mu \leq 1 \text{ and } \mu_1 \leq 1 \\
i.e., \frac{dW}{dt} &\leq \gamma y - d_1 x - d_2 y - \alpha y^2 - d_3 z.
\end{aligned}
$$

Let us introduce a positive real number δ then multiplying δ with W and adding with the above equation and applying the theory stated in [27], we have

$$
\begin{aligned}
\frac{dW}{dt} + \delta W &\leq y(\gamma - d_2 - \alpha y + \delta) - (d_1 - \delta)x - (d_3 - \delta)z. \\
i.e., \frac{dW}{dt} + \delta W &\leq y(\gamma - d_2 - \alpha y + \delta), \text{ taking } \delta = min\{d_1, d_3\} \\
i.e., \frac{dW}{dt} + \delta W &\leq \frac{(\delta + \gamma - d_2)^2}{4\alpha} = Q. \text{ (say)} \\
i.e., W &\leq \frac{Q}{\delta}(1 - e^{-\delta t}) + W(0)e^{-\delta t}.
\end{aligned}
$$

For $t \to \infty$, we have $W \leq \frac{Q}{\delta}$.

Hence, all the solutions of the system (3.1) are bounded in the region
$\sum = \{(x, y, z) \in R_+^3 : W = \frac{Q}{\delta} + \epsilon, \text{ for any } \epsilon > 0\}$.

Note: If the conservation rate of juvenile prey and adult prey to the predator are less than one. Then the solutions of the system (3.1) will be uniformly bounded.

3.4 Equilibria and Stability Analysis

In this section, we find all the possible equilibria and discuss their stability analysis.

3.4.1 Equilibria

The above model has three possible equilibria such as:

(i) The trivial equilibrium $E_0(0, 0, 0)$.

(ii) The predator free equilibrium $E_1(x_1, y_1, 0)$ exists if $\beta\gamma > d_2(\beta + d_1)$ where $x_1 = \frac{\gamma[\beta\gamma - d_2(\beta + d_1)]}{\alpha(\beta + d_1)^2}$ and $y_1 = \frac{[\beta\gamma - d_2(\beta + d_1)]}{\alpha(\beta + d_1)}$.

(iii) The positive interior equilibrium $E^*(x^*, y^*, z^*)$, where
$x^{*2}(\beta_1 d_3 k_1 - \mu\beta_1^2 - d_1\mu\beta_1) + \gamma\mu_1\beta_2 y^{*2} + \eta k_1\beta_1 x^{*2}y^* + \beta_1\eta k_2 x^* y^{*2}$
$+ x^* y^*(\mu\gamma\beta_1 - \mu_1\beta\beta_2 - d_1\mu_1\beta_2 - \mu_1\beta_1\beta_2 + \beta_1 d_3 k_2) = 0$,
$z^* = \frac{x^*(\mu\beta_1 - d_3 k_1) + y^*(\mu_1\beta_2 - d_3 k_2) - \eta k_1 x^* y^* - \eta k_2 y^{*2}}{(d_3 + \eta y^*)}$,
$A_1 y^{*2} + A_2 y^* + A_3 = 0$, i.e., $y^* = \frac{-A_2 \pm \sqrt{A_2^2 - 4A_1 A_3}}{2A_1}$, where $A_1 = (\alpha\mu_1 - \eta k_2)$, $A_2 = \mu_1 d_2 + \mu_1\beta_2 - \mu\gamma - d_3 k_2 - \eta k_1 x^*$, $A_3 = x^*(\mu\beta + \mu d_1 + \mu\beta_1 - \mu_1\beta - d_3 k_1)$.
Case 1: If $A_1 > 0$, $A_3 < 0$ then only one positive value of y^* can be obtained as $y^* = \frac{-A_2 + \sqrt{A_2^2 - 4A_1 A_3}}{2A_1}$.
Case 2: If $A_1 > 0$, $A_2 < 0$ and $A_3 > 0$ then two positive value of y^* can be found if $A_2^2 > 4A_1 A_3$.

3.4.2 Local Stability Analysis

Theorem 3.2 The trivial equilibrium E_0 will be locally asymptotically stable if $\beta\gamma < d_2(\beta + d_1)$.

Proof. Since the system is undefined at $(0, 0, 0)$ and difficult to study the behavior of the system at that point. To overcome such situation, we modify the model (3.1) as when $(x, y, z) \neq (0, 0, 0)$ and $\frac{dx}{dt} = \frac{dy}{dt} = \frac{dz}{dt} = 0$ at $E_0(0, 0, 0)$. To analyze the behavior of the system at trivial equilibrium, we follow the method developed by Arino et al. [11]. Then we rewrite the model as

$$\frac{dV}{dt} = H(V(t)) + Q(V(t)).$$

where $H(.)$ is a continuous and homogeneous function of degree one; $V = (x, y, z)$; Q is a C^1 function with $Q(V) = o(V)$. For the present problem, $H = (\lambda_1 x, \lambda_2 y, -d_3 z)$ where $\lambda_{1,2} = \frac{-(\beta + d_1 + d_2) \pm \sqrt{(\beta + d_1 + d_2)^2 - 4[d_2(\beta + d_1) - \beta\gamma]}}{2}$. Let $V(t)$ be a solution of the above such that $\liminf_{t \to \infty} ||V(t)|| = 0$ and $V(t_n)$ be the corresponding sequence which tends to zero as $t \to \infty$.
Define $y_n = (V(t_n + s)/||V(t_n + s)||)$. Then, y_n is a sequence such that $||y_n|| = 1$.
Now, by Ascoli-Arzela theorem, there should exist a subsequence of y_n that converges to a function $y(t)$ satisfying the equation

$$\frac{dy}{dt} = H(y(t)) - (y(t), H(y(t)))y(t). \tag{3.2}$$

The steady state of the above equation will be given by the vector $v(t) = (v_1, v_2, v_3)$ where $H(v) = (v, H)v$ are the solutions of the eigenvalue problem

$$H(v) = \lambda v$$
$$\lambda = (v, H(v)). \tag{3.3}$$

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From the above equation we have $(\lambda_1 - \lambda)v_1 = 0, (\lambda_2 - \lambda)v_2 = 0, (d_3 + \lambda)v_3 = 0$.
We now study the following cases:

Case I: $v_1 \neq 0, v_2 = v_3 = 0$.
In this case, the system can reach the trivial equilibrium (origin) along the x-axis with $\lambda = \lambda_1$ when $\beta\gamma < d_2(\beta + d_1)$.

Case II: $v_1 = v_3 = 0, v_2 \neq 0$.
The system will reach the origin along the y-axis with $\lambda = \lambda_2$ when $\beta\gamma < d_2(\beta + d_1)$.

Case III: $v_1 = v_2 = 0, v_3 \neq 0$.
The system will reach the origin along the z-axis with $\lambda = -d_3$.

Theorem 3.3 Predator free equilibrium E_1 is locally asymptotically stable if $\eta > \left[\frac{\mu\beta\gamma+\mu_1\beta_2(\beta+d_1)}{k_1\gamma+k_2(\beta+d_1)} - d_3\right]\alpha(\beta + d_1)/\left[\beta\gamma - d_2(\beta + d_1)\right]$ and $\beta\gamma > d_2(\beta + d_1)$.

Proof. The characteristic equation of the jacobian matrix at $E_1(x_1, y_1, 0)$ is

$$
\left(\frac{1}{(k_1x_1 + k_2y_1)}[\mu\beta_1x_1 + \mu_1\beta_2y_1] - d_3 - \eta y_1 - \lambda\right)[\lambda^2 + \lambda(\beta + d_1 + d_2 + 2\alpha y_1)
$$
$$
+\beta\gamma - d_2(\beta + d_1)] = 0.
$$

Hence, E_1 is locally asymptotically stable if
$\eta > \left[\frac{\mu\beta\gamma+\mu_1\beta_2(\beta+d_1)}{k_1\gamma+k_2(\beta+d_1)} - d_3\right]\alpha(\beta + d_1)/\left[\beta\gamma - d_2(\beta + d_1)\right]$ and $\beta\gamma > d_2(\beta + d_1)$.

Note: When the anti-predator behavior of adult prey is greater than the difference of predation of juvenile prey, adult prey by predator and death rate of predator population, divided by the equilibrium biomass of adult prey. Then the predator free equilibrium will be locally asymptotically stable.

Observation 1 Suppose that $\eta > \left[\frac{\mu\beta\gamma+\mu_1\beta_2(\beta+d_1)}{k_1\gamma+k_2(\beta+d_1)} - d_3\right]\alpha(\beta + d_1)/\left[\beta\gamma - d_2(\beta + d_1)\right]$ holds. Then for the parametric condition $\gamma = d_2(\beta + d_1)/\beta$ the model system (1) undergoes through a transcritical bifurcation around the trivial equilibrium point E_0 as when $\gamma < d_2(\beta + d_1)/\beta$ then the trivial equilibrium becomes asymptotically stable where as for $\gamma > d_2(\beta + d_1)/\beta$ not only the trivial equilibrium E_0 becomes unstable but also a new (predator free) equilibrium forms and becomes locally asymptotically stable.

Theorem 3.4 The interior equilibrium E^* of the system (3.1) is locally asymptotically stable if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$ holds where σ_1, σ_2 and σ_3 are given within the proof.

Proof. The characteristic equation of the jacobian matrix at $E^*(x^*, y^*, z^*)$ is

$$
\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0.
$$

where $\sigma_1 = -(M_{11} + M_{22} + M_{33})$,
$\sigma_2 = (M_{11}M_{22} - M_{12}M_{21}) + (M_{11}M_{33} - M_{13}M_{31}) + (M_{22}M_{33} - M_{23}M_{32})$,

$\sigma_3 = M_{11}\left(M_{23}M_{32} - M_{22}M_{33}\right) + M_{12}\left(M_{21}M_{33} - M_{23}M_{31}\right) + M_{13}\left(M_{31}M_{22} - M_{21}M_{32}\right),$

$\sigma_1\sigma_2 - \sigma_3 = -M_{11}^2(M_{22} + M_{33}) - M_{22}^2(M_{11} + M_{33}) - M_{33}^2(M_{11} + M_{22})$

$+M_{11}(M_{12}M_{21} + M_{13}M_{31}) + M_{22}(M_{12}M_{21} + M_{23}M_{32}) + M_{33}(M_{13}M_{31} - 2M_{11}M_{22}),$

$M_{11} = -\beta - d_1 - \frac{\beta_1 z^*(z^* + k_2 y^*)}{(z^* + k_1 x^* + k_2 y^*)^2}, \; M_{12} = \gamma + \frac{\beta_1 k_2 x^* z^*}{(z^* + k_1 x^* + k_2 y^*)^2}, \; M_{13} = -\frac{\beta_1 x^*(k_1 x^* + k_2 y^*)}{(z^* + k_1 x^* + k_2 y^*)^2},$

$M_{21} = \beta + \frac{\beta_2 k_1 y^* z^*}{(z^* + k_1 x^* + k_2 y^*)^2}, \; M_{22} = -d_2 - 2\alpha y^* - \frac{\beta_2 z^*(z^* + k_1 x^*)}{(z^* + k_1 x^* + k_2 y^*)^2}, \; M_{23} = -\frac{\beta_2 y^*(k_1 x^* + k_2 y^*)}{(z^* + k_1 x^* + k_2 y^*)^2},$

$M_{31} = \frac{(\mu\beta_1 z^*(z^* + k_2 y^*) - k_1\mu_1\beta_2 y^* z^*)}{(z^* + k_1 x^* + k_2 y^*)^2}, \; M_{32} = \frac{(\mu_1\beta_2 z^*(z^* + k_1 x^*) - \mu\beta_1 k_2 x^* z^*)}{(z^* + k_1 x^* + k_2 y^*)^2} - \eta z^*,$

$M_{33} = \frac{k_1 x^* + k_2 y^*}{(z^* + k_1 x^* + k_2 y^*)^2}\left(\mu\beta_1 x^* + \mu_1\beta_2 y^*\right) - d_3 - \eta y^*.$

By using the Routh-Hurwitz criteria, it is observed that the interior equilibrium point will be locally asymptotically stable if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$ holds.

3.4.3 Global Stability Analysis

Here, we consider an autonomous dynamical system:

$$\dot{x} = f(x) \tag{3.4}$$

where $f : D \to R^n, D \subset R^n$ be an open set and simply connected and $f \in C^1(D)$ which is the space of continuously differentiable function on domain D. Each solution $x(t)$ of this differential equation (3.4) is uniquely determined by its initial value $x(0) = x_0$. Let x^* be an equilibrium of (3.4). Then we can say that x^* is said to be globally asymptotically stable in D if it is locally asymptotically stable and all trajectories in D converge to x^*. It is assumed that the following hypothesis hold:

(i) There exists a compact absorbing set $K \subset D$

(ii) The equation (3.4) has unique equilibrium x^* in D.

The basic idea of this method is that if the equilibrium x^* is locally asymptotically stable, then the global stability is assured provided that (i) and (ii) hold and the equation (3.4) has no periodic solution.

Now, a matrix $P(x)$ is choosen in such a way that it will be a nonsingular $\binom{n}{2} \times \binom{n}{2}$ matrix valued function $x \to P(x)$ that is defined in C^1 on D and it is considered that

$$B = P_f P^{-1} + PJ^{[2]}P^{-1}.$$

where the matrix P_f is $(P_{ij}(x))_f$ which is given by

$$(P_{ij}(x))_f = \nabla P_{ij}.f(x)$$

and the jacobian matrix $J^{[2]}$ is the second additive compound matrix of the jacobian matrix J. Generally for a $n \times n$ matrix $J = (J_{i,j})_n = \left(\frac{\partial f_i}{\partial x_j}\right)_n$, $J^{[2]}$ is a $\binom{n}{2} \times \binom{n}{2}$ matrix and in a special case for $n = 3$, it is as follows:

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

Now, it is considered that the Lozinskii measure Γ of B with respect to a vector norm $|.|$ in R^N where $N = \binom{n}{2}$, is defined by

$$\Gamma(B) = \lim_{h \to 0} \frac{|I + hB| - 1}{h}.$$

Now, it is proved in [131] that if (i) and (ii) hold as well as the condition

$$\lim_{t \to \infty} \sup \sup \frac{1}{t} \int_0^t \Gamma(B(x(s, x_0)))ds < 0. \tag{3.5}$$

then it guarantees that there are no orbits giving rise to a simple rectifiable curve in D which is invariant for (3.4). Therefore, Li and Muldowney [131] states that if the conditions (i) and (ii) hold, then the interior equilibrium x^* is globally asymptotically stable in D provided that there exist a nonsingular matrix valued function $P(x)$ and a Lozinskii measure μ such that the condition (3.5) holds. Using this results we have proved that our proposed system (3.1) is globally asymptotically stable around its interior equilibrium as follows:

Theorem 3.5 The system (3.1) is globally asymptotically stable around its interior equilibria if $\eta < \{\frac{(\mu\beta_1 + \mu_1\beta_2)}{(1 + k_1 + k_2)} - d_3 - \mu_5\}/\mu_2$ where the expression of μ_2 and μ_5 are given within the proof of this theorem.

Proof. The system (3.1) can be expressed as

$$\frac{dX}{dt} = f(X).$$

where $f(X) = \begin{pmatrix} \gamma y - \beta x - d_1 x - \frac{\beta_1 xz}{z + k_1 x + k_2 y} \\ \beta x - d_2 y - \alpha y^2 - \frac{\beta_2 yz}{z + k_1 x + k_2 y} \\ \frac{\mu\beta_1 xz}{z + k_1 x + k_2 y} + \frac{\mu_1 \beta_2 yz}{z + k_1 x + k_2 y} - d_3 z - \eta yz \end{pmatrix}$ and $X = \begin{pmatrix} x \\ y \\ z \end{pmatrix}$.

Then the jacobian matrix (J) of the system (3.1) is

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

$J_{11} = -\beta - d_1 - \frac{\beta_1 z(z + k_2 y)}{(z + k_1 x + k_2 y)^2}$, $J_{12} = \gamma + \frac{\beta_1 k_2 xz}{(z + k_1 x + k_2 y)^2}$, $J_{13} = -\frac{\beta_1 x(k_1 x + k_2 y)}{(z + k_1 x + k_2 y)^2}$,

$J_{21} = \beta + \frac{\beta_2 k_1 yz}{(z + k_1 x + k_2 y)^2}$, $J_{22} = -d_2 - 2\alpha y - \frac{\beta_2 z(z + k_1 x)}{(z + k_1 x + k_2 y)^2}$, $J_{23} = -\frac{\beta_2 y(k_1 x + k_2 y)}{(z + k_1 x + k_2 y)^2}$,

$J_{31} = \frac{(\mu\beta_1 z(z + k_2 y) - k_1\mu_1\beta_2 yz)}{(z + k_1 x + k_2 y)^2}$, $J_{32} = \frac{(\mu_1\beta_2 z(z + k_1 x) - \mu\beta_1 k_2 xz)}{(z + k_1 x + k_2 y)^2} - \eta z$,

$J_{33} = \frac{k_1 x + k_2 y}{(z + k_1 x + k_2 y)^2}(\mu\beta_1 x + \mu_1\beta_2 y) - d_3 - \eta y$.

If $J^{[2]}$ be the second additive compound matrix jacobian matrix of the J, then

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

Next, we consider $P(X)$ in $C^1(D)$ in such a way that $P = diag\left\{\frac{x}{z}, \frac{x}{z}, \frac{x}{z}\right\}$ and then we have $P^{-1} = diag\left\{\frac{z}{x}, \frac{z}{x}, \frac{z}{x}\right\}$.

Now, $P_f = \frac{dP}{dX} = diag\left\{\frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}, \frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}, \frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}\right\}$, $P_f P^{-1} = diag\left\{\frac{\dot{x}}{x} - \frac{\dot{z}}{z}, \frac{\dot{x}}{x} - \frac{\dot{z}}{z}, \frac{\dot{x}}{x} - \frac{\dot{z}}{z}\right\}$ and $PJ^{[2]}P^{-1} = J^{[2]}$.

Also, we have

$$B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$$

where $B_{11} = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + J_{11} + J_{22}$, $B_{12} = \begin{pmatrix} J_{23} & -J_{13} \end{pmatrix}$, $B_{21} = \begin{pmatrix} J_{32} & -J_{31} \end{pmatrix}^t$ and $B_{22} = \begin{pmatrix} J_{11} + J_{33} & J_{12} \\ J_{21} & J_{22} \end{pmatrix}$.

Let us introduce the following vector norm in R^3 of the form

$$|(u, v, w)| = max\{|u|, |v| + |w|\}.$$

where (u, v, w) is the vector in R^3 and Lozinskii measure with respect to this norm is denoted by Γ.

So, $\Gamma(B) \leq sup\{p_1, p_2\}$ where $p_1 = \Gamma_1(B_{11}) + |B_{12}|$ and $p_2 = \Gamma_1(B_{22}) + |B_{21}|$, where $|B_{12}|$, $|B_{21}|$ are the matrix norms with respect to the L^1 vector norm and Γ_1 is the Lozinskii measure with respect to that norm. Then, the required values can be obtained as

$$\Gamma_1(B_{11}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + J_{11} + J_{22}, |B_{12}| = max\left\{|J_{23}|, |-J_{13}|\right\},$$
$$|B_{21}| = max\left\{|J_{32}|, |-J_{31}|\right\}.$$
$$\Gamma_1(B_{22}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + max\left\{J_{11} + J_{33} + J_{21}, J_{12} + J_{22}\right\}.$$

From the third equation of system (3.1), we have

$$\frac{\dot{z}}{z} = \frac{\mu\beta_1 x}{z + k_1 x + k_2 y} + \frac{\mu_1\beta_2 y}{z + k_1 x + k_2 y} - d_3 - \eta y.$$

Then, using the above values we have

$$p_1 = \frac{\dot{x}}{x} - \frac{\mu\beta_1 x}{z + k_1 x + k_2 y} - \frac{\mu_1\beta_2 y}{z + k_1 x + k_2 y} + d_3 + \eta y + J_{11} + J_{22} + max\left\{|J_{23}|, |-J_{13}|\right\}.$$
$$p_2 = \frac{\dot{x}}{x} - \frac{\mu\beta_1 x}{z + k_1 x + k_2 y} - \frac{\mu_1\beta_2 y}{z + k_1 x + k_2 y} + d_3 + \eta y + max\left\{J_{11} + J_{33} + J_{21}, J_{12} + J_{22}\right\}$$
$$+ max\left\{|J_{32}|, |-J_{31}|\right\}.$$

Therefore, from the above we have

$$\begin{aligned} \Gamma(B) \leq & \frac{\dot{x}}{x} - \frac{\mu\beta_1 x}{z + k_1 x + k_2 y} - \frac{\mu_1\beta_2 y}{z + k_1 x + k_2 y} + d_3 + \eta y \\ & + max[max\left\{J_{11} + J_{22} + |J_{23}|, J_{11} + J_{22} + |-J_{13}|\right\}, \\ & max\left\{J_{11} + J_{33} + J_{21} + |J_{32}|, J_{12} + J_{22} + |-J_{31}|\right\}]. \end{aligned} \tag{3.6}$$

It is assume that there exists a positive real number μ_2 and $t_1 > 0$ such that $\mu_2 = inf\{x(t), y(t), z(t)\}$ when $t > t_1$. Also, we take

$$\mu_3 = max\{J_{11} + J_{22} + |J_{23}|, J_{11} + J_{22} + |-J_{13}|\} \text{ at } \mu_2 = inf\{x(t), y(t), z(t)\}.$$

$$i.e., \mu_3 = max\{-\beta - d_1 - \frac{\beta_1(1 + k_2)}{(1 + k_1 + k_2)^2} - d_2 - 2\alpha\mu_2 - \frac{\beta_2(1 + k_1)}{(1 + k_1 + k_2)^2}$$

$$+ \frac{\beta_2(k_1 + k_2)}{(1 + k_1 + k_2)^2}, -\beta - d_1 - \frac{\beta_1(1 + k_2)}{(1 + k_1 + k_2)^2} - d_2 - 2\alpha\mu_2 - \frac{\beta_2(1 + k_1)}{(1 + k_1 + k_2)^2}$$

$$+ \frac{\beta_1(k_1 + k_2)}{(1 + k_1 + k_2)^2}\}.$$

$$\mu_4 = max\{J_{11} + J_{33} + J_{21} + |J_{32}|, J_{12} + J_{22} + |-J_{31}|\}_{\mu_2=inf\{x(t),y(t),z(t)\}}.$$

$$i.e., \mu_4 = max\{-\beta - d_1 - \frac{\beta_1(1 + k_2)}{(1 + k_1 + k_2)^2} + \frac{k_1 + k_2}{(1 + k_1 + k_2)^2}(\mu\beta_1 + \mu_1\beta_2) - d_3 - \eta\mu_2$$

$$+ \beta + \frac{\beta_2 k_1}{(1 + k_1 + k_2)^2} + |\frac{(\mu_1\beta_2(1 + k_1) - \mu\beta_1 k_2)}{(1 + k_1 + k_2)^2} - \eta\mu_2|, \gamma + \frac{\beta_1 k_2}{(1 + k_1 + k_2)^2}$$

$$- d_2 - 2\alpha\mu_2 - \frac{\beta_2(1 + k_1)}{(1 + k_1 + k_2)^2} + |\frac{(\mu\beta_1(1 + k_2) - k_1\mu_1\beta_2)}{(1 + k_1 + k_2)^2}|\}$$

and $\mu_5 = max\{\mu_3, \mu_4\}$.

Therefore, from equation (3.6) we have

$$\Gamma(B) \leq \frac{\dot{x}}{x} - \frac{\mu\beta_1}{1 + k_1 + k_2} - \frac{\mu_1\beta_2}{1 + k_1 + k_2} + d_3 + \eta\mu_2 + \mu_5.$$

$$i.e., \Gamma(B) \leq \frac{\dot{x}}{x} - \left(\frac{\mu\beta_1}{1 + k_1 + k_2} + \frac{\mu_1\beta_2}{1 + k_1 + k_2} - d_3 - \eta\mu_2 - \mu_5\right).$$

Now, integrating the above equation in $[0, t]$ we have

$$\int_0^t \Gamma(B)ds \leq log\frac{x(t)}{x(0)} - \left(\frac{\mu\beta_1}{1 + k_1 + k_2} + \frac{\mu_1\beta_2}{1 + k_1 + k_2} - d_3 - \eta\mu_2 - \mu_5\right)t.$$

$$\frac{1}{t}\int_0^t \Gamma(B)ds \leq \frac{1}{t}log\frac{x(t)}{x(0)} - \left(\frac{\mu\beta_1}{1 + k_1 + k_2} + \frac{\mu_1\beta_2}{1 + k_1 + k_2} - d_3 - \eta\mu_2 - \mu_5\right).$$

$$\lim_{t\to\infty} sup \, sup \, \frac{1}{t}\int_0^t \Gamma(B)ds < -\left(\frac{\mu\beta_1}{1 + k_1 + k_2} + \frac{\mu_1\beta_2}{1 + k_1 + k_2} - d_3 - \eta\mu_2 - \mu_5\right) < 0.$$

Hence, the system (3.1) will be globally asymptotically stable around the interior equilibria E^* if $\eta < \{\frac{(\mu\beta_1 + \mu_1\beta_2)}{(1 + k_1 + k_2)} - d_3 - \mu_5\}/\mu_2$.

3.4.4 Hopf bifurcation Analysis

In this section, when any parameter value changed then the stability of the system is determined. If we change a parameter for a critical value of this parameter then different dynamical behavior except stability may occur. The critical value of a parameter for

which the dynamical behavior of a system is changed, is called the critical point. The critical value of the parameter is called the bifurcation point. In this chapter, we have considered (η) (rate of anti-predator behavior of adult prey) as the bifurcation parameter.

Theorem 3.6 The necessary and sufficient conditions for the occurrence of Hopf bifurcation at $\eta = \eta^*$ are stated as follows:
(i) $\sigma_i(\eta^*) > 0, i = 1, 2, 3$
(ii) $\sigma_1(\eta^*)\sigma_2(\eta^*) - \sigma_3(\eta^*) = 0,$
(iii) $Re\left(\frac{d\lambda_i}{d\eta}\right)_{\eta=\eta^*} \neq 0, i = 1, 2, 3$
where λ_i are the roots of the characteristic equation corresponding to the interior equilibria and σ_1, σ_2 and σ_3 are defined within the **Theorem 3.4**.

Proof. When $\eta = \eta^*$ then the characteristic equation in **Theorem 3.4**

$$\lambda^3 + \sigma_1\lambda + \sigma_2\lambda + \sigma_3 = 0. \tag{3.7}$$

becomes

$$(\lambda^2 + \sigma_2)(\lambda + \sigma_1) = 0.$$

since at $\eta = \eta^*$, $\sigma_1(\eta^*)\sigma_2(\eta^*) - \sigma_3(\eta^*) = 0$.
So, the above characteristic equation has three roots such as $\lambda_1 = i\sqrt{\sigma_2}, \lambda_2 = -i\sqrt{\sigma_2}$ and $\lambda_3 = -\sigma_1$.
Now, for $\eta \in (\eta^* - \epsilon, \eta^* + \epsilon)$, the roots of the characteristic equation are

$$\lambda_1(\eta) = \phi_1(\eta) + i\phi_2(\eta),$$
$$\lambda_2(\eta) = \phi_1(\eta) - i\phi_2(\eta),$$
$$\lambda_3(\eta) = -\sigma_1.$$

Now, we verify the transversality condition $Re\left(\frac{d\lambda_i}{d\eta}\right)_{\eta=\eta^*} \neq 0, i = 1, 2, 3$.
Substituting $\lambda_1(\eta) = \phi_1(\eta) + i\phi_2(\eta)$ in equation (3.7) and calculating the derivatives we have

$$P(\eta)\phi_1'(\eta) - Q(\eta)\phi_2(\eta) + U(\eta) = 0,$$
$$Q(\eta)\phi_1'(\eta) + P(\eta)\phi_2(\eta) + V(\eta) = 0,$$

where

$$P(\eta) = 3\phi_1^2(\eta) + 2\sigma_1(\eta)\phi_1(\eta) + \sigma_2(\eta) - 3\phi_2^2(\eta),$$
$$Q(\eta) = 6\phi_1(\eta)\phi_2(\eta) + 2\sigma_1(\eta)\phi_2(\eta),$$
$$U(\eta) = \phi_1^2(\eta)\sigma_1'(\eta) + \sigma_2'(\eta)\phi_1(\eta) + \sigma_3'(\eta) - \sigma_1'(\eta)\phi_2^2(\eta),$$
$$V(\eta) = 2\phi_1(\eta)\phi_2(\eta)\sigma_1'(\eta) + \sigma_2'(\eta)\phi_2(\eta),$$

Now,

$$\left(\frac{d}{d\eta}(Re(\lambda_i(\eta)))\right)_{\eta=\eta^*} = -\frac{Q(\eta^*)V(\eta^*) + P(\eta^*)U(\eta^*)}{P^2(\eta^*) + Q^2(\eta^*)} \neq 0, \text{ for } i = 1, 2$$

since $Q(\eta^*)V(\eta^*) + P(\eta^*)U(\eta^*) \neq 0$ and $\lambda_3(\eta^*) = -\sigma_1$.
Hence, the theorem is proved.

3.5   The Model without Anti-predator Effects

Taking the rate of anti-predator behavior $\eta = 0$ then system (3.1) reduces to the following form

$$\left.\begin{aligned}
\frac{dx}{dt} &= \gamma y - \beta x - d_1 x - \frac{\beta_1 xz}{z + k_1 x + k_2 y} \\
\frac{dy}{dt} &= \beta x - d_2 y - \alpha y^2 - \frac{\beta_2 yz}{z + k_1 x + k_2 y} \\
\frac{dz}{dt} &= \frac{\mu\beta_1 xz}{z + k_1 x + k_2 y} + \frac{\mu_1\beta_2 yz}{z + k_1 x + k_2 y} - d_3 z
\end{aligned}\right\} \tag{3.8}$$

System (3.8) has three possible equilibria as

(i) The trivial equilibria $\hat{E}_0(0,0,0)$.

(ii) The predator free equilibria $\hat{E}_1(x_2, y_2, 0)$ exists if $\beta\gamma > d_2(\beta + d_1)$
where $x_2 = \frac{\gamma[\beta\gamma - d_2(\beta+d_1)]}{\alpha(\beta+d_1)^2}$ and $y_2 = \frac{[\beta\gamma - d_2(\beta+d_1)]}{\alpha(\beta+d_1)}$.

(iii) The positive interior equilibria $\hat{E}_*(\hat{x}^*, \hat{y}^*, \hat{z}^*)$, where
$\hat{x}^{*2}(\beta_1 d_3 k_1 - \mu\beta_1^2 - d_1\mu\beta_1) + \gamma\mu_1\beta_2\hat{y}^{*2} + \hat{x}^*\hat{y}^*(\mu\gamma\beta_1 - \mu_1\beta\beta_2 - d_1\mu_1\beta_2 - \mu_1\beta_1\beta_2 + \beta_1 d_3 k_2) = 0$, $\hat{z}^* = \frac{\hat{x}^*(\mu\beta_1 - d_3 k_1) + \hat{y}^*(\mu_1\beta_2 - d_3 k_2)}{d_3}$,
$\mu_1\alpha\hat{y}^{*2} + A_4\hat{y}^* + A_5 = 0$ i.e., $\hat{y}^* = \frac{-A_4 \pm \sqrt{A_4^2 - 4\mu_1\alpha A_5\hat{x}^*}}{2\mu_1\alpha}$,
$A_4 = \mu_1 d_2 + \mu_1\beta_2 - \mu\gamma - d_3 k_2$, $A_5 = \mu\beta + \mu d_1 + \mu\beta_1 - \mu_1\beta - d_3 k_1$.
Case 1: If $A_5 < 0$ then only one positive value of $\hat{y}^*$ can be found and its value
becomes $\hat{y}^* = \frac{-A_4 + \sqrt{A_4^2 - 4\mu_1\alpha A_5\hat{x}^*}}{2\mu_1\alpha}$.
Case 2: If $A_4 < 0$ and $A_5 > 0$ then two positive value of $\hat{y}^*$ can be found if
$A_4^2 - 4\mu_1\alpha A_5\hat{x}^* > 0$.

Lemma 3.1 The predator free equilibria $\hat{E}_1$ of system (3.8) will be locally asymptotically stable if $\beta\gamma > d_2(\beta + d_1)$ and $d_3 > \frac{\mu\beta_1\gamma + \mu_1\beta_2(\beta+d_1)}{k_1\gamma + k_2(\beta+d_1)}$.

Lemma 3.2 The interior equilibria $\hat{E}_*$ will be locally asymptotically stable if $\sigma_1' > 0$, $\sigma_3' > 0$ and $\sigma_1'\sigma_2' - \sigma_3' > 0$, where σ_1', σ_2' and σ_3' can be obtained by putting $\eta = 0$ in the expression of σ_1, σ_2 and σ_3 respectively given in **Theorem 3.4**.

3.6 Comparison Between Two Models

From the model (3.1), we see that the predator free equilibrium's locally asymptotically stability depends on the anti-predator behavior of the adult prey population. Again, it is also seen from the model (3.1) that for interior equilibrium, the equilibrium biomass of predator population depends on the equilibrium biomass of juvenile prey and adult prey population. Also, the equilibrium biomass of predator population decreases with the increases of the anti-predator behavior of adult prey.

But from the model (3.8), we see that the predator free equilibrium's locally asymptotically stability depends on the death rate of the predator population. If death rate is greater than some value then predator population vanishes and locally asymptotically stable. Also, for the interior equilibrium of the model (3.8), the equilibrium biomass of predator population depends on the biomass of prey and adult prey population but not depends upon the anti-predator behavior.

3.7 Numerical Simulation

In this section, the dynamical behavior of the proposed model (3.1) has been discussed numerically using MATLAB. Let us consider a set of parametric values as: $\gamma = 1.5$, $\beta = 0.3$, $d_1 = 0.25$, $\beta_1 = 0.1$, $k_1 = 0.1$, $k_2 = 0.5$, $d_2 = 0.13$, $\alpha = 0.14$, $\beta_2 = 0.2$, $\mu = 0.3$, $\mu_1 = 0.4$, $d_3 = 0.2$, $\eta = 0.2$. For this set of parametric values Figure 3.1 has been drawn which shows that the predator free equilibrium $E_1(13.41, 4.911, 0)$ is locally asymptotically stable.
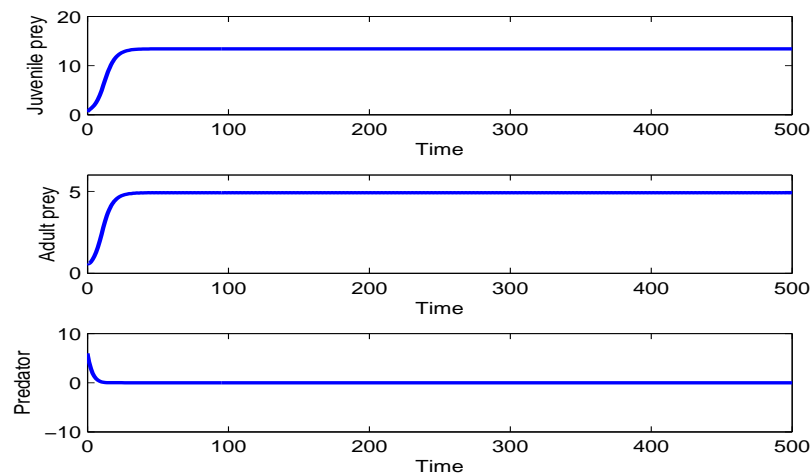

Figure 3.1: Local stability of the equilibrium point E_1.

Again, we consider another set of parametric values as $\gamma = 1.5$, $\beta = 0.29$, $d_1 = 0.25$, $\beta_1 = 0.1$, $k_1 = 0.01$, $k_2 = 0.02$, $d_2 = 0.13$, $\alpha = 0.14$, $\beta_2 = 0.2$, $\mu = 0.75$, $\mu_1 = 0.8$, $d_3 = 0.25$, $\eta = 0.01$. Using the above set of parametric values Figure 3.2 has been drawn. From this figure, it is seen that the interior equilibrium $E^*(6.076, 2.579, 3.034)$ is locally asymptotically stable.
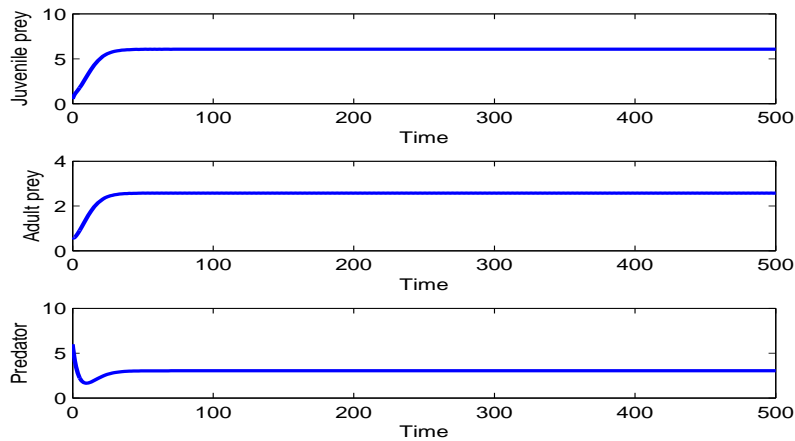

Figure 3.2: Local stability of the equilibrium point E^*.

Also, another set of parametric values have been considered as $\gamma = 0.5, \beta = 2.5, d_1 = 0.25, \beta_1 = 0.5, k_1 = 1.0, k_2 = 1.0, d_2 = 0.23, \alpha = 0.1, \beta_2 = 0.29, \mu = 0.2, \mu_1 = 0.7, d_3 = 0.11, \eta = 0.0001$. For this set of parametric values Fig.3.3 has been drawn with different initial conditions. From this figure, it is observed that the interior equilibrium is globally asymptotically stable.
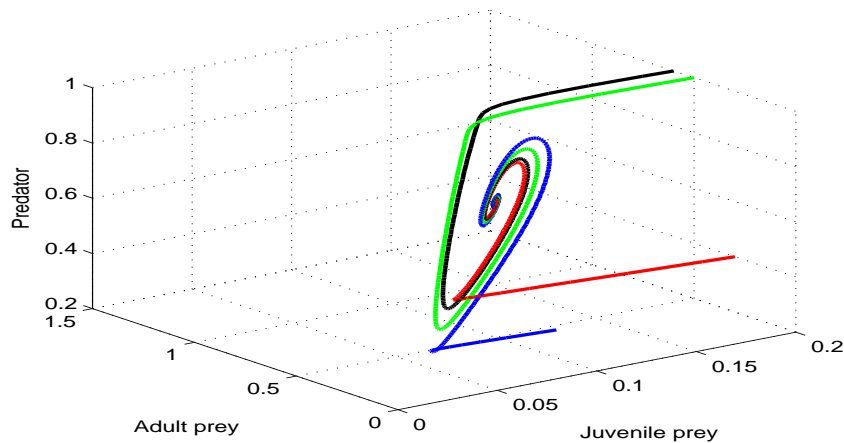

Figure 3.3: Global stability of interior equilibrium.

For the set of parametric values $\gamma = 0.5, \beta = 2.5, d_1 = 0.25, \beta_1 = 0.5, k_1 = 1.0, k_2 = 1.0, d_2 = 0.23, \alpha = 0.09, \beta_2 = 0.29, \mu = 0.2, \mu_1 = 0.98, d_3 = 0.11$, Figure 3.4 has been drawn. From this figure, it is observe that when η, the anti-predator behavior of adult prey crosses 0.00105 then the value of $(\sigma_1\sigma_2 - \sigma_3)$ becomes negative. So, here $\eta^* = 0.00105$ is the critical point or bifurcation point and the unstable solution curve with respect to time and the corresponding Hopf bifurcation diagram of the system have been shown in Figure 3.5 and Figure 3.6 respectively. From these figure, it is observed that the interior equilibrium losses its stability as the value of $\eta < \eta^*$.
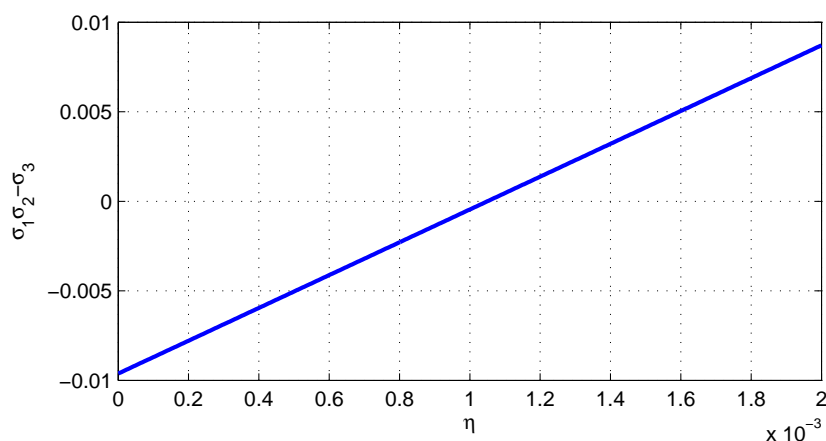

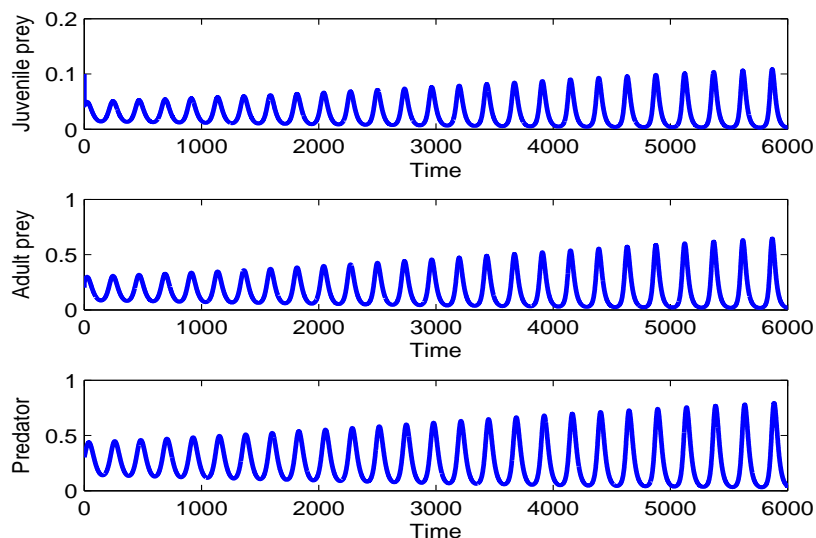Figure 3.4: Represents the value of $(\sigma_1\sigma_2 - \sigma_3)$ with respect to η.

Figure 3.5: Unstable interior equilibrium for $\eta(= 0.001) < \eta^*$.
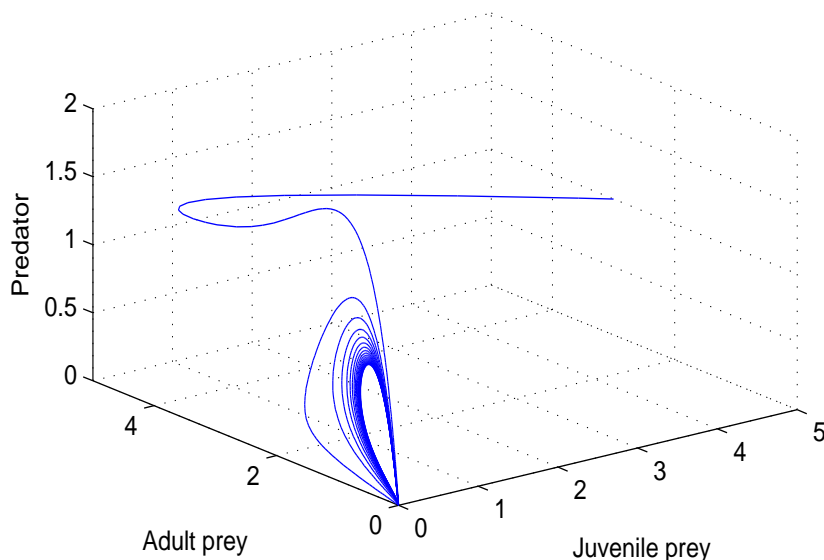
Figure 3.6: Bifurcation diagram for $\eta(= 0.001) < \eta^*$.

Using the same set of parametric values used in Figure 3.4 except $\eta = 0.01$, the Figure 3.7 and Figure 3.8 have been drawn. From these figure, it is seen that the unstable system becomes stable i.e., the interior equilibrium is asymptotically stable. From these figure, it can be concluded that the anti-predator behavior may act as a control of instability or chaos.

Figure 3.7: Stable solution of the system (3.1) when $\eta(= 0.01) > \eta^*$.

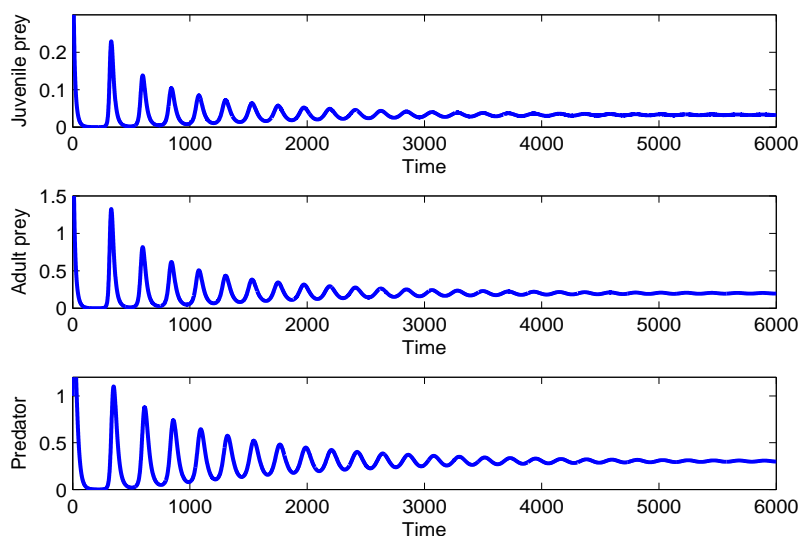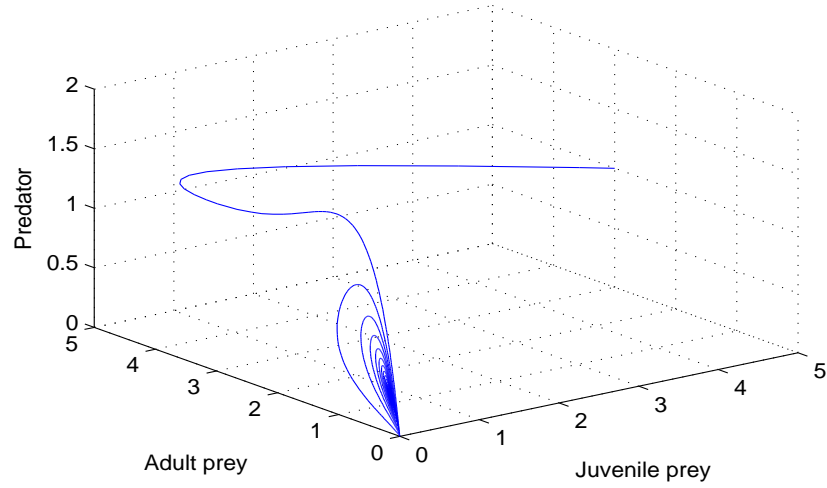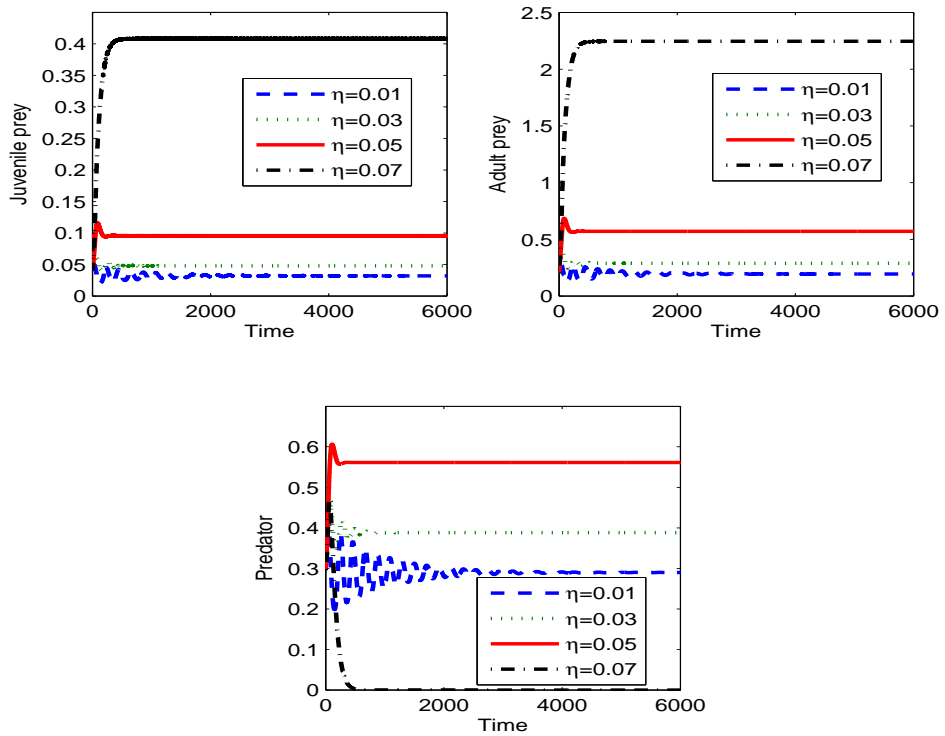Figure 3.8: Stable solution of the system (3.1) when $\eta(= 0.01) > \eta^*$.

Figure 3.9: Sensitivity analysis with respect to η.

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For the same set of parametric values used in Figure 3.5 with different values of η, Figure 3.9 has been drawn. From this figure, it is observed that as the value of anti-predator behavior increases then the number of juvenile prey and adult prey increases. Because due to the increase of anti-predator behavior of adult prey, the predation rate of adult prey to the predator decreases and also adult prey struggle to save the juvenile prey from the predation. Again, for the increase of anti-predator behavior of adult prey, predator population first increases due to availability of juvenile prey but for more higher values of η predator population gradually deceases and it may extinct.

To compare the results of original system (3.1) and the system (3.8), the following parametric values has been considered as: $\gamma = 0.5, \beta = 2.5, d_1 = 0.25, \beta_1 = 0.5, k_1 = 1.0, k_2 = 1.0, d_2 = 0.23, \alpha = 0.09, \beta_2 = 0.29, \mu = 0.21, \mu_1 = 0.97, d_3 = 0.11, \eta = 0.01$. Using the above parametric values Figure 3.10 and Figure 3.11 have been drawn. From these figure, it is seen that for the same set of parametric values the interior equilibrium is stable for the original system (3.1) but the interior equilibrium is unstable for the system without anti-predator effects (3.8).
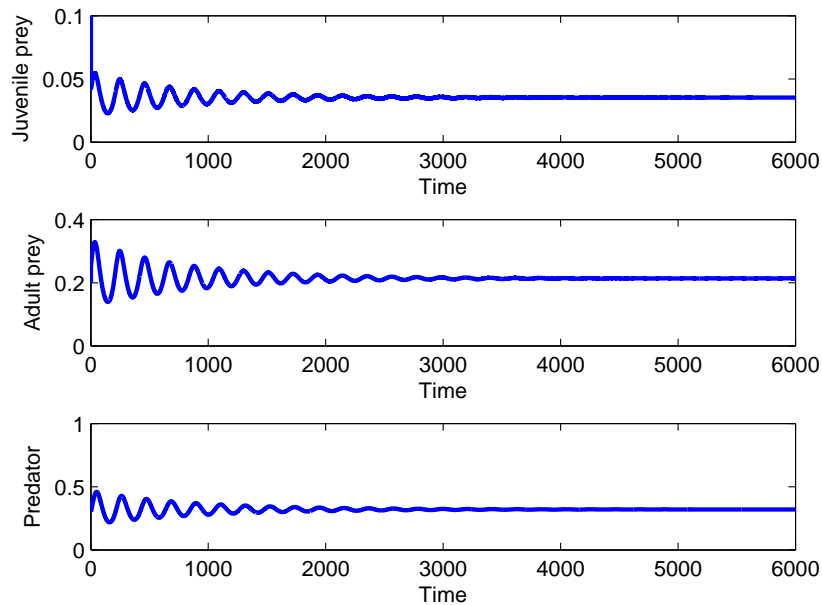

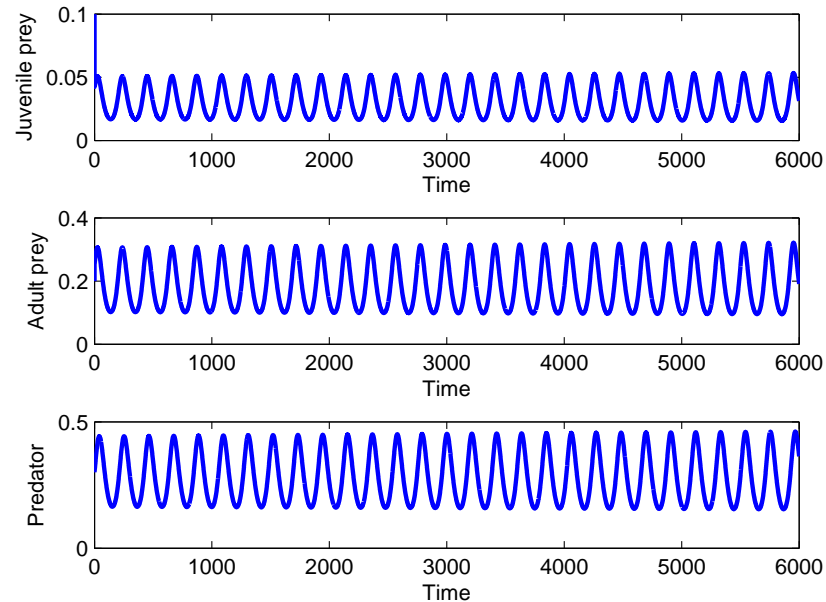Figure 3.10: Stable interior equilibrium for the original system (3.1).

Figure 3.11: Unstable interior equilibrium for the system (3.8).

3.8 Conclusion

Of late mathematical ecology has become a demanding area of research to the scientists and researchers of different specializations including mathematics, biology, economics etc. They are engaged to find out the solution of various ecological problems which evolve with prey-predator interactions, inter and intra-specific competitions, biological conversations and bio-diversity, extinction and coexistence of populations of different communities in different environment etc. In this chapter, we intend to study a mathematical model on prey-predator system with some anti-predator behavior of the adult prey populations. This type of system can be observed in red colobus monkeys exhibit mobbing when threatened by chimpanzees.

In this chapter, we have thoroughly described the dynamical behavior of our proposed model through both theoretical and numerical experiments. Our investigation ensures the existence of three possible equilibria including vanishing, predator free and the co-existence one. The asymptotic local stability analysis of the equilibria shows that all the equilibria are not only conditionally asymptotically stable but also there is a possibility of occurring transcritical bifurcation around the trivial equilibrium. Thus, it can be concluded that the recruitment rate of juvenile prey populations (γ) plays an important role regarding the existence of entire prey species in the system as a comparative less

value of γ would tend to the system asymptotically stable around trivial equilibrium (see Theorem 3.2) where as comparative higher value of γ assures the system existence of entire prey species (see Theorem 3.3).

We mainly study the behavior of the system around co-existing equilibrium due to its natural importance. Apart from locally asymptotically stability criteria, we investigate the existence of Hopf bifurcation and global asymptotic stability criteria of the interior equilibrium. A geometric method has been applied to investigate the global asymptotic stability of the proposed system around the interior equilibrium. There are very few papers on prey predator mathematical model with anti-predator behavior. The existing paper considered the anti-predator behavior on the whole prey species [230]. But in reality, it is seen that adults prey attacks their vulnerable predators and save the younger prey from predation. So, in this mathematical model we introduce the anti-predator behavior term only on the adult prey population. Also, the ratio dependent functional response function has been considered along with the anti-predator behavior of adult prey population which are newly introduced in this chapter. From the numerical simulation, it is seen that if the anti-predator behavior (η) of adult prey is less (or greater) than a critical value (η^*) then the system (3.1) becomes unstable (or stable). Also, from the sensitivity analysis (see Figure 3.9) of system (3.1) with respect to η, it is observed that as the value of η increases then the density of juvenile prey and adult prey increases but the density of predator decreases. It is biologically meaningful, because it helps to protect juvenile prey from predation. Again, from Figure 3.10 and Figure 3.11, it can be concluded that η has an important role to stabilize a system. So, from our analysis, it can be said that the anti-predator behavior has a significant role in a prey-predator dynamics.

Gateway from Chapter 3 to Chapter 4
Species extinction is a great problem for our globe. Extinction of top predator is a great threat. The next chapter is devoted to study the effects of supplying additional food to prevent the extinction of top predator. We also study the dynamics of such system taking into account harvesting effect on top predator.

Chapter 4

Effects of Additional Food in Tritrophic Food Chain Model with Harvesting Only

4.1 Introduction

Modelling of predator prey interaction is an important topic in mathematical biology due to its universal existence and importance. It was started with the pioneer work of Lotka and Volterra [139] in 1956. After Lotka and Volterra, many researchers [98, 148, 160] investigated several mathematical models to understand predator prey dynamics. The growth rate of prey and the functional response are the most important factors for modelling of predator prey ecological system. There are many types of growth rates among them the logistic growth is the mostly used prey growth rate. Holling type functional responses are commonly used in prey predator interactions. Most of the considered prey predator models are either two species or three species model. Dynamics of three species prey predator model are much more complex than a two species model. There are many unexplored things still there. Three species food chain models are investigated by mathematical biologists and ecologists for the last four decades. Hastings and Powell [92] found existence of chaos in a three species food chain model. Persistence in models of three interacting predator-prey populations was reported by Freedman and Waltman [73]. Nonlinear aspects of competition between three species was investigated by May and Leonard [149]. Aziz-Alaoui et al. [13,14,128] analyzed a three species predator prey model with two types of functional responses. Fan and Kuang [67] reported the effects of Beddington-DeAngelis functional response on a predator prey system. Recently, Panja and Mondal [175] investigated the stability and existence conditions of

three species predator prey model with Holling type II functional response.

Now, the continuous harvesting may cause the extinction of predator population and that makes the ecosystem unstable by loosing bio-diversity. Additional food may be very useful component in ecosystem to prevent the species extinction. It is shown that the supply of additional food to predator may be useful to control infectious disease of prey [197]. Again, the effects of additional food may be helpful to stabilize a delayed type predator prey system [199]. So, the additional food can change the shape of an ecological system. Biological control of predator population through the supply of additional food to the predator was reported by Srinivasu et al. [223] in 2007. Kar and Chattopadhyay [117] investigated a long run sustain-ability of a predator to be harvested in the prey-predator system in the presence of alternative food. Chakraborty and Das [37] reported the effects of supply additional food in a prey predator system with constant prey refuge. Most of the previous investigations are done to study the effects of presence of additional food either in two species prey predator model or in a three species model with additional food for top predator only.

Realistically it is seen that, the harvesting is an important issue for the conservation biologist. Determination of optimal harvesting rate is of great importance to avoid species extinction. Good harvesting strategy is helpful for economic developments of a country. In 1992, Myerscough et al. [162] reported the effects of predator harvesting and stoking in a two species predator prey model. Impact of harvesting on two competing fish species in presence of toxicity was discussed by Kar and Chaudhuri [112]. Chakraborty et al. [34] reported the effects of prey harvesting in a ratio dependent eco-epidemiological system. Jana et al. [108] investigated the effects of harvesting and infection on predator in a prey-predator system. Gupta et al. [86] studied the impact of nonlinear predator harvesting in a predator prey system. Sahoo and Poria [200] reported the effects of supplying alternative food in a prey predator model with top predator harvesting. In their model [200], the top predator can consume only the middle predator and the middle predator can consume only the prey species. But in many real world ecological systems [118, 171], the top predator can consume both the middle predator as well as the prey.

From the above literature review, it is observed that many investigations have been made on the predator prey system. But till now, there exists some lacunas such as:

There exists few number of mathematical models on predator prey dynamics where it has been assumed that the top predator consumes both prey and middle predator. Also, the effects of supplying additional food to the predators have not been studied extensively by mathematical biologists. Again, there exists very few research papers on the study of effects of additional food to the predator prey system to control chaos or to increase harvesting of predator population. But, there is no work considering additional food to the top predator including the fact that top predator can consume both the

middle predator as well as the prey and harvesting on top predator.

To overcome these lacunas, a predator prey model in the presence of additional food has been developed in this chapter. To investigate the effects of additional food and harvesting, we propose a three species prey predator model where the top predator consume both prey and middle predator. In fact, we have investigated the impacts of top predator to be harvested in the presence of additional food for top predator only. The effects of variation of additional food and harvesting rate are reported in this study. Bifurcation analysis is done with respect to harvesting rate and with respect to consumption rate of prey to the top predator. The optimal harvesting rate is calculated and the effects of supplying additional food on the system are analyzed.

4.2 Model Formulation

Now, we consider three species food chain model of Hastings and Powell [92] as follows:

$$
\left.
\begin{aligned}
\frac{dX}{dT} &= R_0 X\left(1 - \frac{X}{K_0}\right) - \frac{C_1 A_1 XY}{B_1 + X} \\[2mm]
\frac{dY}{dT} &= \frac{A_1 XY}{B_1 + X} - \frac{A_2 YZ}{B_2 + Y} - D_1 Y \\[2mm]
\frac{dZ}{dT} &= \frac{C_2 A_2 YZ}{B_2 + Y} - D_2 Z
\end{aligned}
\right\}
\tag{4.1}
$$

where T is the time, R_0 and K_0 are the intrinsic growth rates and the carrying capacity of prey population X respectively. The conservation rate of prey (X) and middle predator (Y) to the top predator (Z) are C_1^{-1} and C_2 respectively. The death rate for middle predator and top predator are D_1 and D_2 respectively. Also, the constants A_1, A_2 and B_1, B_2 are the predation rates and half saturation constants respectively. Sahoo and Poria [200] extended this model (4.1) by introducing additional food to the top predator only.
Then, Pal et al. [171] extended the model (4.1) by assuming that the top predator consumes both prey and middle predator. According to their assumption, the model (4.1) is reduced to the following form

$$
\left.
\begin{aligned}
\frac{dX}{dT} &= R_0 X\left(1 - \frac{X}{K_0}\right) - \frac{C_1 A_1 XY}{B_1 + X} - \frac{A_3 XZ}{B_3 + X} \\[2mm]
\frac{dY}{dT} &= \frac{A_1 XY}{B_1 + X} - \frac{A_2 YZ}{B_2 + Y} - D_1 Y \\[2mm]
\frac{dZ}{dT} &= \frac{C_2 A_2 YZ}{B_2 + Y} + \frac{C_3 A_3 XZ}{B_3 + X} - D_2 Z
\end{aligned}
\right\}
\tag{4.2}
$$

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where A_3, B_3 and C_3 are the predation rates of top predator, half saturation constant and conservation rate of prey to the top predator respectively. This type of mathematical model is the real example of the interaction among Cod fish, Herring fish and Sprat fish [118].

First, we extend the model (4.2) considering the effects of constant harvesting of top predator. Since the continuous harvesting may cause the extinction of top predator population, so, we apply additional food (A) $(0 \leq A \leq 1)$ to the top predator because it helps the top predator for faster growth and hence it can reduce the predation pressure of both the prey population and middle predator population. Supplying of alternative food to top predator helps to increase its harvesting and henceforth that will be profitable for economic growth. Incorporating this idea, we modify the model (4.2) into the following form:

$$
\left.
\begin{aligned}
\frac{dX}{dT} &= R_0 X(1 - \frac{X}{K_0}) - \frac{C_1 A_1 XY}{B_1 + X} - \frac{(1-A)A_3 XZ}{B_3 + X} \\[2mm]
\frac{dY}{dT} &= \frac{A_1 XY}{B_1 + X} - \frac{(1-A)A_2 YZ}{B_2 + Y} - D_1 Y \\[2mm]
\frac{dZ}{dT} &= \frac{(1-A)C_2 A_2 YZ}{B_2 + Y} + \frac{(1-A)C_3 A_3 XZ}{B_3 + X} + (C_2 A_2 + C_3 A_3)AZ - D_2 Z - EZ.
\end{aligned}
\right\}
\tag{4.3}
$$

It is easy to check that for $A = 0$ the model (4.3) is converted to model (4.2) and for $A = 1$ the prey and middle predator have no connection to the top predator. In this case, the top predator growth depends only on the additional food.

Now, we non-dimensionalize the model (4.3) by taking $x = \frac{X}{K_0}, y = \frac{C_1 Y}{K_0}$, $z = \frac{C_1 Z}{C_2 K_0}$, $t = R_0 T$ and obtain the following system:

$$
\left.
\begin{aligned}
\frac{dx}{dt} &= x(1-x) - \frac{a_1 xy}{1 + b_1 x} - \frac{a_3(1-A)xz}{1 + b_3 x} \\[2mm]
\frac{dy}{dt} &= \frac{a_1 xy}{1 + b_1 x} - \frac{a_2(1-A)yz}{1 + b_2 y} - d_1 y \\[2mm]
\frac{dz}{dt} &= \frac{a_2(1-A)yz}{1 + b_2 y} + \frac{c_1 a_3(1-A)xz}{1 + b_3 x} + (\frac{a_3}{b_3}c_1 + a_2 c_2)Az - d_2 z - ez
\end{aligned}
\right\}
\tag{4.4}
$$

where $a_1 = \frac{A_1 K_0}{R_0 B_1}$, $a_2 = \frac{A_2 K_0 C_2}{B_2 R_0 C_1}$, $a_3 = \frac{A_3 C_2 K_0}{B_3 C_1 R_0}$, $b_1 = \frac{K_0}{B_1}$, $b_2 = \frac{K_0}{B_2 C_1}$, $b_3 = \frac{K_0}{B_3}$, $d_1 = \frac{D_1}{R_0}$, $d_2 = \frac{D_2}{R_0}$, $e = \frac{E}{R_0}$, $c_1 = \frac{C_1 C_3}{C_2}$, $c_2 = \frac{C_1 B_2}{K_0}$. We analyze the system (4.4) with initial conditions $x(0) \geq 0$, $y(0) \geq 0$, $z(0) \geq 0$.

4.3 Positivity and Boundedness of Solutions

In this section, we have discussed about the positivity and boundedness of solutions of the proposed system.

Theorem 4.1 All the solutions of the system (4.4) will be nonnegative.

Proof. The first equation of system (4.4) can be rewritten as

$$\frac{dx}{x} = \left\{ (1 - x) - \frac{a_1 y}{1 + b_1 x} - \frac{a_3(1 - A)z}{1 + b_3 x} \right\} dt,$$

which is of the form $\frac{dx}{x} = \phi(x, y, z)$, where $\phi(x, y, z) = (1 - x) - \frac{a_1 y}{1 + b_1 x} - \frac{a_3(1-A)z}{1+b_3 x}$.
Then integrating, the above equation from $[0, t]$, we have

$$x(t) = x(0)e^{\int_0^t \phi(x,y,z)dt} > 0, \forall t$$

Again, from the second equation of system (4.4), we have

$$\frac{dy}{y} = \left\{ \frac{a_1 x}{1 + b_1 x} - \frac{a_2(1 - A)z}{1 + b_2 y} - d_1 \right\} dt$$

which is of the form $\frac{dy}{y} = \psi(x, y, z)$, where $\psi(x, y, z) = \frac{a_1 x}{1+b_1 x} - \frac{a_2(1-A)z}{1+b_2 y} - d_1$.
Integrating, the above equation from $[0, t]$, we have

$$y(t) = y(0)e^{\int_0^t \psi(x,y,z)dt} > 0, \forall t$$

Also, from the last equation of system (4.4), we have

$$\frac{dz}{z} = \left\{ \frac{a_2(1 - A)y}{1 + b_2 y} + \frac{c_1 a_3(1 - A)x}{1 + b_3 x} + (\frac{a_3}{b_3}c_1 + a_2 c_2)A - d_2 - e \right\} dt$$

which is of the form $\frac{dz}{z} = \chi(x, y, z)$,
where $\chi(x, y, z) = \frac{a_2(1-A)y}{1+b_2 y} + \frac{c_1 a_3(1-A)x}{1+b_3 x} + (\frac{a_3}{b_3}c_1 + a_2 c_2)A - d_2 - e$.
Integrating above from $[0, t]$, we have

$$z(t) = z(0)e^{\int_0^t \chi(x,y,z)dt} > 0, \forall t$$

Hence, all the solutions of system (4.4) are non-negative.

Theorem 4.2 All solutions of the system (4.4) will be bounded in R_+^3 if $c_1 \leq 1$ and $d_2 + e > A(a_2 c_2 + \frac{a_3}{b_3}c_1)$.

Proof. Let us construct a function

$$W = x + y + z.$$

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Taking time derivative of above function, we have

$$
\begin{aligned}
\frac{dW}{dt} &= \frac{dx}{dt} + \frac{dy}{dt} + \frac{dz}{dt} \\
i.e., \frac{dW}{dt} &= x(1-x) - \frac{a_1 xy}{1+b_1 x} - \frac{a_3(1-A)xz}{1+b_3 x} + \frac{a_1 xy}{1+b_1 x} - \frac{a_2(1-A)yz}{1+b_2 y} - d_1 y \\
&\quad + \frac{a_2(1-A)yz}{1+b_2 y} + \frac{c_1 a_3(1-A)xz}{1+b_3 x} + (\frac{a_3}{b_3}c_1 + a_2 c_2)Az - d_2 z - ez \\
&= x(1-x) - d_1 y - \frac{a_3(1-A)xz}{1+b_3 x}(1-c_1) - d_2 z - ez + (a_2 c_2 + \frac{a_3}{b_3}c_1)Az \\
&\leq x(1-x) - d_1 y - \left\{ d_2 + e - A(a_2 c_2 + \frac{a_3}{b_3}c_1) \right\} z, \text{ since } c_1 \leq 1 \\
&\leq -x - (x-1)^2 + 1 - d_1 y - \left\{ d_2 + e - A(a_2 c_2 + \frac{a_3}{b_3}c_1) \right\} z \\
&\leq -\alpha(x+y+z) - (x-1)^2 + 1
\end{aligned}
$$

where $\alpha = min\{1, d_1, d_2 + e - A(a_2 c_2 + \frac{a_3}{b_3}c_1)\}$, provided that $d_2 + e > A(a_2 c_2 + \frac{a_3}{b_3}c_1)$.

$$
\text{Therefore, } \frac{dW}{dt} + \alpha W \leq 1
$$

Applying the theory of differential inequality, we have

$$
0 < W < \frac{1 - e^{-\alpha t}}{\alpha} + W(0)e^{-\alpha t}.
$$

Now, for $t \to \infty$, we have $0 < W < \frac{1}{\alpha}$. Hence all solutions of system (4.4) will be bounded in the region $\Lambda = \{(x, y, z) \in R^3 : W = \frac{1}{\alpha} + \epsilon, \text{ for } \epsilon > 0\}$.

Note: Biological meaning of the above condition can be interpreted as the following. When the conservation rate of top-predator population is less than or equal to one and $\frac{d_2 + e}{(a_2 c_2 + \frac{a_3}{b_3}c_1)} > A$, then the solutions of the system (4.4) will be bounded.

4.4 Different Equilibria and Their Stability Analysis

The system (4.4) has five possible equilibria as

(i) The trivial equilibrium $E_0(0,0,0)$.

(ii) The axial equilibrium $E_1(1,0,0)$.

(iii) The top predator free equilibrium $E_2(\bar{x}, \bar{y}, 0)$ where $\bar{x} = \frac{d_1}{(a_1 - b_1 d_1)}, \bar{y} = \frac{a_1 - (b_1+1)d_1}{(a_1 - b_1 d_1)^2}$ exists if $a_1 > (b_1 + 1)d_1$.

(iv) The middle predator free equilibrium $E_3(\hat{x}, 0, \hat{z})$ where $\hat{x} = \frac{\{(d_2+e)-(\frac{a_3}{b_3}c_1+a_2c_2)A\}}{c_1a_3-b_3(d_2+e)+a_2c_2b_3A}$, $\hat{z} = \frac{(1-\hat{x})(1+b_3\hat{x})}{a_3(1-A)}$.

(v) The interior equilibrium $E^*(x^*, y^*, z^*)$ where
$$(1 - x^*) - \frac{a_1y^*}{1+b_1x^*} - \frac{a_3(1-A)z^*}{1+b_3x^*} = 0$$
$$y^* = \frac{(d_2+e)-(\frac{a_3}{b_3}c_1+a_2c_2)A-\frac{c_1a_3(1-A)x^*}{1+b_3x^*}}{a_2(1-A)-b_2\left[(d_2+e)-(\frac{a_3}{b_3}c_1+a_2c_2)A-\frac{c_1a_3(1-A)x^*}{1+b_3x^*}\right]}$$, exists if $(d_2 + e) > (\frac{a_3}{b_3}c_1 + a_2c_2)A - \frac{c_1a_3(1-A)x^*}{1+b_3x^*}$ and $a_2(1 - A) > b_2\left[(d_2 + e) - (\frac{a_3}{b_3}c_1 + a_2c_2)A - \frac{c_1a_3(1-A)x^*}{1+b_3x^*}\right]$.
$$z^* = \frac{\left(\frac{a_1x^*}{1+b_1x^*}-d_1\right)(1+b_2y^*)}{a_2(1-A)}, \text{ exists if } \left(\frac{a_1x^*}{1+b_1x^*} > d_1\right).$$

Theorem 4.3 The trivial equilibrium of system (4.4) is always unstable.

Proof. The characteristic equation of the system (4.4) at the trivial equilibrium is given by

$$(\lambda - 1)(\lambda + d_1)\left(\lambda - [(c_1\frac{a_3}{b_3} + a_2c_2)A - d_2 - e]\right) = 0$$

Eigenvalues of this equation are $1, -d_1$ and $[(c_1\frac{a_3}{b_3} + a_2c_2)A - d_2 - e]$. Since the two eigenvalues 1 and $-d_1$ are opposite in sign, so the trivial equilibrium is unstable.

Theorem 4.4 The axial equilibrium $E_1(1, 0, 0)$ of system (4.4) will be locally asymptotically stable if $\frac{a_1}{1+b_1} < d_1$ and $A < \frac{(d_2+e)-\frac{c_1a_3}{1+b_3}}{a_2c_2+c_1\frac{a_3}{b_3}-\frac{c_1a_3}{1+b_3}}$.

Proof. The characteristic equation of system (4.4) at $E_1(1, 0, 0)$ is given by

$$(\lambda + 1)\left(\lambda - \frac{a_1}{1 + b_1} + d_1\right)\left(\lambda - \frac{c_1a_3(1 - A)}{1 + b_3} - a_2c_2A + d_2 + e\right) = 0$$

Eigenvalues of the above equation are $\lambda_1 = -1, \lambda_2 = \frac{a_1}{1+b_1} - d_1$ and $\lambda_3 = \frac{c_1a_3(1-A)}{1+b_3} + a_2c_2A - d_2 - e$. So the equilibrium E_1 will be locally asymptotically stable if $\frac{a_1}{1+b_1} < d_1$ and $A < \frac{(d_2+e)-\frac{c_1a_3}{1+b_3}}{a_2c_2+c_1\frac{a_3}{b_3}-\frac{c_1a_3}{1+b_3}}$.

Theorem 4.5 Top predator free equilibrium $E_2(\bar{x}, \bar{y}, 0)$ of system (4.4) will be locally asymptotically stable if $B_{33} < 0$, $B_{11} + B_{22} < 0$ and $B_{11}B_{22} - B_{12}B_{21} > 0$ where B_{11}, B_{12}, B_{21} and B_{22} are given within the proof.

Proof. The characteristic equation of system (4.4) at $E_2(\bar{x}, \bar{y}, 0)$ is given by
$$(\lambda - B_{33})\left(\lambda^2 - \lambda(B_{11} + B_{22}) + B_{11}B_{22} - B_{12}B_{21}\right) = 0$$

where $B_{11} = 1 - 2\bar{x} - \frac{a_1\bar{y}}{(1+b_1\bar{x})^2}$, $B_{12} = -\frac{a_1\bar{x}}{1+b_1\bar{x}}$, $B_{21} = \frac{a_1\bar{y}}{(1+b_1\bar{x})^2}$, $B_{22} = \frac{a_1\bar{x}}{1+b_1\bar{x}} - d_1$, $B_{33} = \frac{a_2(1-A)\bar{y}}{1+b_2\bar{y}} + \frac{c_1a_3(1-A)\bar{x}}{1+b_3\bar{x}} + (c_1\frac{a_3}{b_3} + a_2c_2)A - d_2 - e$.

So, the equilibrium E_2 will be locally asymptotically stable if $B_{33} < 0$, $B_{11} + B_{22} < 0$ and $B_{11}B_{22} - B_{12}B_{21} > 0$. From the above conditions for local stability of top predator free equilibrium E_2, we have $A < \dfrac{[d_2 + e - \frac{a_2\bar{y}}{1+b_2\bar{y}} - \frac{c_1 a_3 \bar{x}}{1+b_3\bar{x}}]}{[(\frac{a_3}{b_3}c_1 + a_2 c_2) - \frac{a_2\bar{y}}{1+b_2\bar{y}} - \frac{c_1 a_3 \bar{x}}{1+b_3\bar{x}}]}$, $d_1 > 1 - 2\bar{x} - \dfrac{a_1\bar{y}}{(1+b_1\bar{x})^2} + \dfrac{a_1\bar{x}}{1+b_1\bar{x}}$

and $(1 - 2\bar{x})(\frac{a_1\bar{x}}{(1+b_1\bar{x})} - d_1) + \frac{a_1 d_1\bar{y}}{(1+b_1\bar{x})^2} > 0$.

Theorem 4.6 Middle predator free equilibrium $E_3(\hat{x}, 0, \hat{z})$ of system (4.4) will be locally asymptotically stable if $M_{22} < 0$, $M_{11} + M_{33} < 0$ and $M_{11}M_{33} - M_{13}M_{31} > 0$ where M_{11}, M_{13}, M_{31} and M_{33} are given within the proof.

Proof. The characteristic equation of system (4.4) at $E_3(\hat{x}, 0, \hat{z})$ is given by

$$(\lambda - M_{22})\left(\lambda^2 - \lambda(M_{11} + M_{33}) + M_{11}M_{33} - M_{13}M_{31}\right) = 0$$

where $M_{11} = 1 - 2\hat{x} - \dfrac{a_3(1-A)\hat{z}}{(1+b_3\hat{x})^2}$, $M_{13} = -\dfrac{a_3(1-A)\hat{x}}{1+b_3\hat{x}}$, $M_{22} = \dfrac{a_1\hat{x}}{1+b_1\hat{x}} - a_2(1-A)\hat{z} - d_1$,
$M_{31} = \dfrac{c_1 a_3(1-A)\hat{z}}{(1+b_3\hat{x})^2}$, $M_{33} = \dfrac{c_1 a_3(1-A)\hat{x}}{1+b_3\hat{x}} + \left(c_1\frac{a_3}{b_3} + a_2 c_2\right)A - d_2 - e$.
So, the equilibrium E_3 will be locally asymptotically stable if $M_{22} < 0$, $(M_{11} + M_{33}) < 0$ and $M_{11}M_{33} - M_{13}M_{31} > 0$.
From the conditions of local stability of middle predator free equilibrium E_3, we have
$\dfrac{\left[1 - 2\hat{x} - d_2 - e - \frac{a_3\hat{z}}{(1+b_3\hat{x})^2} + \frac{c_1 a_3\hat{x}}{(1+b_3\hat{x})}\right]}{\left[\frac{c_1 a_3\hat{x}}{(1+b_3\hat{x})} - \frac{a_3\hat{z}}{(1+b_3\hat{x})^2} - \left(c_1\frac{a_3}{b_3} + a_2 c_2\right)\right]} < A < \left[1 - \frac{1}{a_2\hat{z}}\left(\frac{a_1\hat{x}}{1+b_1\hat{x}} - d_1\right)\right]$ and
$\left\{\left(c_1\frac{a_3}{b_3} + a_2 c_2\right) - d_2 - e\right\}\left[1 - 2\hat{x} - \frac{a_3(1-A)\hat{z}}{(1+b_3\hat{x})^2}\right] + (1 - 2\hat{x})\frac{c_1 a_3(1-A)\hat{x}}{(1+b_3\hat{x})} > 0$.

Theorem 4.7 Interior equilibrium $E^*(x^*, y^*, z^*)$ of system (4.4) will be locally asymptotically stable if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$.

Proof. The characteristic equation of system (4.4) at $E^*(x^*, y^*, z^*)$ is given by

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0$$

where $\sigma_1 = -(N_{11} + N_{22} + N_{33})$, $\sigma_2 = N_{11}N_{22} + N_{11}N_{33} + N_{22}N_{33} - N_{12}N_{21} - N_{13}N_{31} - N_{23}N_{32}$, $\sigma_3 = N_{11}N_{23}N_{32} + N_{12}N_{21}N_{33} + N_{13}N_{31}N_{22} - N_{11}N_{22}N_{33} - N_{12}N_{31}N_{23} - N_{13}N_{21}N_{32}$,
$N_{11} = 1 - 2x^* - \dfrac{a_1 y^*}{(1+b_1 x^*)^2} - \dfrac{a_3(1-A)z^*}{(1+b_3 x^*)^2}$, $N_{12} = -\dfrac{a_1 x^*}{1+b_1 x^*}$, $N_{13} = -\dfrac{a_3(1-A)x^*}{1+b_3 x^*}$, $N_{21} = \dfrac{a_1 y^*}{(1+b_1 x^*)^2}$,
$N_{22} = \dfrac{a_1 x^*}{1+b_1 x^*} - \dfrac{a_2(1-A)z^*}{(1+b_2 y^*)^2} - d_1$, $N_{23} = -\dfrac{a_2(1-A)y^*}{1+b_2 y^*}$, $N_{31} = \dfrac{c_1 a_3(1-A)z^*}{(1+b_3 x^*)^2}$, $N_{32} = \dfrac{a_2(1-A)z^*}{(1+b_2 y^*)^2}$ and
$N_{33} = \dfrac{a_2(1-A)y^*}{(1+b_2 y^*)^2} + \dfrac{c_1 a_3(1-A)x^*}{1+b_3 x^*} + \left(c_1\frac{a_3}{b_3} + a_2 c_2\right)A - d_2 - e$.
Hence, according to Routh-Hurwith criteria the interior equilibrium E^* will be locally asymptotically stable if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$.

Theorem 4.8 The system (4.4) is globally asymptotically stable around its interior equilibria if $\mu_2 > 0$ and the expression of μ_2 is given within the proof of this theorem.

Proof. The system (4.4) can be expressed as

$$\frac{dX}{dt} = f(X)$$

where $f(X) = \begin{pmatrix} x(1-x) - \frac{a_1 xy}{1+b_1 x} - \frac{a_3(1-A)xz}{1+b_3 x} \\ \frac{a_1 xy}{1+b_1 x} - \frac{a_2(1-A)yz}{1+b_2 y} - d_1 y \\ \frac{a_2(1-A)yz}{1+b_2 y} + \frac{c_1 a_3(1-A)xz}{1+b_3 x} + \left(\frac{a_3}{b_3}c_1 + a_2 c_2\right)Az - d_2 z - ez \end{pmatrix}$ and $X = \begin{pmatrix} x \\ y \\ z \end{pmatrix}$.

Then the jacobian matrix (J) of the system (4.4) is

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

where $J_{11} = 1 - 2x - \frac{a_1 y}{(1+b_1 x)^2} - \frac{a_3(1-A)z}{(1+b_3 x)^2}$, $J_{12} = -\frac{a_1 x}{1+b_1 x}$, $J_{13} = -\frac{a_3(1-A)x}{1+b_3 x}$,
$J_{21} = \frac{a_1 y}{(1+b_1 x)^2}$, $J_{22} = \frac{a_1 x}{1+b_1 x} - \frac{a_2(1-A)z}{(1+b_2 y)^2} - d_1$, $J_{23} = -\frac{a_2(1-A)y}{1+b_2 y}$,
$J_{31} = \frac{c_1 a_3(1-A)z}{(1+b_3 x)^2}$, $J_{32} = \frac{a_2(1-A)z}{(1+b_2 y)^2}$,
$J_{33} = \frac{a_2(1-A)y}{1+b_2 y} + \frac{c_1 a_3(1-A)x}{1+b_3 x} + \left(\frac{a_3}{b_3}c_1 + a_2 c_2\right)A - d_2 - e$.
If $J^{[2]}$ be the second additive compound matrix [131] jacobian matrix of the J, then

$$J^{[2]} = \begin{pmatrix} J_{11} + J_{22} & J_{23} & -J_{13} \\ J_{32} & J_{11} + J_{33} & J_{12} \\ -J_{31} & J_{21} & J_{22} + J_{33} \end{pmatrix} = \begin{pmatrix} L_1 & J_{23} & -J_{13} \\ J_{32} & L_2 & J_{12} \\ -J_{31} & J_{21} & L_3 \end{pmatrix}$$

where $L_1 = J_{11} + J_{22}, L_2 = J_{11} + J_{33}$ and $L_3 = J_{22} + J_{33}$.
Next, we consider $P(X)$ in $C^1(D)$ in such a way that $P = diag\left\{\frac{x}{z}, \frac{x}{z}, \frac{x}{z}\right\}$ and then we have $P^{-1} = diag\left\{\frac{z}{x}, \frac{z}{x}, \frac{z}{x}\right\}$.
Now, $P_f = \frac{dP}{dX} = diag\left\{\frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}, \frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}, \frac{\dot{x}}{z} - \frac{x}{z^2}\dot{z}\right\}$, $P_f P^{-1} = diag\left\{\frac{\dot{x}}{x} - \frac{\dot{z}}{z}, \frac{\dot{x}}{x} - \frac{\dot{z}}{z}, \frac{\dot{x}}{x} - \frac{\dot{z}}{z}\right\}$
and $PJ^{[2]}P^{-1} = J^{[2]}$.
Also, we have

$$B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$$

where $B_{11} = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + L_1$, $B_{12} = \begin{pmatrix} J_{23} & -J_{13} \end{pmatrix} = \begin{pmatrix} -\frac{a_2(1-A)y}{1+b_2 y} & \frac{a_3(1-A)x}{1+b_3 x} \end{pmatrix}$,

$B_{21} = \begin{pmatrix} J_{32} & -J_{31} \end{pmatrix}^t = \begin{pmatrix} \frac{a_2(1-A)z}{(1+b_2 y)^2} \\ -\frac{c_1 a_3(1-A)z}{(1+b_3 x)^2} \end{pmatrix}$.

and $B_{22} = \begin{pmatrix} \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + J_{11} + J_{33} & J_{12} \\ J_{21} & \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + J_{22} + J_{33} \end{pmatrix} = \begin{pmatrix} \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + L_2 & -\frac{a_1 x}{1+b_1 x} \\ \frac{a_1 y}{(1+b_1 x)^2} & \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + L_3 \end{pmatrix}$.

Let us introduce the following vector norm in R^3 of the form

$$|(u, v, w)| = max\{|u|, |v| + |w|\}$$

where (u, v, w) is the vector in R^3 and Lozinskii measure with respect to this norm is denoted by Γ.

So, $\Gamma(B) \leq sup\{q_1, q_2\}$ where $q_1 = \Gamma_1(B_{11}) + |B_{12}|$ and $q_2 = \Gamma_1(B_{22}) + |B_{21}|$, where $|B_{12}|$, $|B_{21}|$ are the matrix norms with respect to the L^1 vector norm and Γ_1 is the Lozinskii measure with respect to that norm. Then, the required values can be obtained as

$$\Gamma_1(B_{11}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + L_1, |B_{12}| = max\left\{|-\frac{a_2(1-A)y}{1+b_2y}|, |\frac{a_3(1-A)x}{1+b_3x}|\right\},$$

$$|B_{21}| = max\left\{|\frac{a_2(1-A)z}{(1+b_2y)^2}|, |-\frac{c_1a_3(1-A)z}{(1+b_3x)^2}|\right\}$$

$$\Gamma_1(B_{22}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + max\left\{L_2 + \frac{a_1y}{(1+b_1x)^2}, L_3 - \frac{a_1x}{1+b_1x}\right\}$$

From the third equation of system (4.4) we have

$$\frac{\dot{z}}{z} = \frac{a_2(1-A)y}{1+b_2y} + \frac{c_1a_3(1-A)x}{1+b_3x} + (\frac{a_3}{b_3}c_1 + a_2c_2)A - (d_2 + e)$$

Then, using the above values we have

$$
\begin{aligned}
q_1 &= \frac{\dot{x}}{x} - \frac{a_2(1-A)y}{1+b_2y} - \frac{c_1a_3(1-A)x}{1+b_3x} - (\frac{a_3}{b_3}c_1 + a_2c_2)A + (d_2 + e) + L_1 \\
&+ max\left\{|-\frac{a_2(1-A)y}{1+b_2y}|, |\frac{a_3(1-A)x}{1+b_3x}|\right\} \\
q_2 &= \frac{\dot{x}}{x} - \frac{a_2(1-A)y}{1+b_2y} - \frac{c_1a_3(1-A)x}{1+b_3x} - (\frac{a_3}{b_3}c_1 + a_2c_2)A + (d_2 + e) \\
&+ max\left\{L_2 + \frac{a_1y}{(1+b_1x)^2}, L_3 - \frac{a_1x}{1+b_1x}\right\} + max\left\{|\frac{a_2(1-A)z}{(1+b_2y)^2}|, |-\frac{c_1a_3(1-A)z}{(1+b_3x)^2}|\right\}
\end{aligned}
$$

Therefore, from the above we have

$$
\begin{aligned}
\Gamma(B) &\leq sup\{q_1, q_2\} \\
&\leq \frac{\dot{x}}{x} - \frac{a_2(1-A)y}{1+b_2y} - \frac{c_1a_3(1-A)x}{1+b_3x} - (\frac{a_3}{b_3}c_1 + a_2c_2)A + (d_2 + e) + L_1 \\
&+ max\left\{L_2 + \frac{a_1y}{(1+b_1x)^2} + \frac{a_2(1-A)z}{(1+b_2y)^2} + |-\frac{a_2(1-A)y}{1+b_2y}|,\right. \\
&\left. L_3 - \frac{a_1x}{1+b_1x} + \frac{a_3(1-A)x}{1+b_3x} + |-\frac{c_1a_3(1-A)z}{(1+b_3x)^2}|\right\}
\end{aligned}
$$

Substituting the values of L_1, L_2 and L_3, the above reduces to the form

$$
\begin{aligned}
\Gamma(B) \quad \leq \quad & sup\left\{q_1, q_2\right\} \\
\leq \quad & \frac{\dot{x}}{x} - \left[2x + \frac{a_1 y}{(1+b_1 x)^2} + \frac{a_3(1-A)z}{(1+b_3 x)^2} + \frac{a_2(1-A)z}{(1+b_2 y)^2} - \frac{a_1 x}{1+b_1 x} + d_1 - 1 \right. \\
- \quad & max\left\{1 - 2x - \frac{a_3(1-A)z}{(1+b_3 x)^2} + \frac{a_2(1-A)z}{(1+b_2 y)^2} + |-\frac{a_2(1-A)y}{1+b_2 y}|, \right. \\
& \left. \left. \frac{a_3(1-A)x}{1+b_3 x} - \frac{a_2(1-A)z}{(1+b_2 y)^2} - d_1 + |-\frac{c_1 a_3(1-A)z}{(1+b_3 x)^2}|\right\}\right]
\end{aligned}
\tag{4.5}
$$

It is assume that there exists a positive real number μ_1 and $t_1 > 0$ such that $\mu_1 = inf\{x(t), y(t), z(t)\}$ when $t > t_1$. Also, let us take

$$
\begin{aligned}
\mu_2 \quad = \quad & 2\mu_1 + \frac{a_1 \mu_1}{(1+b_1 \mu_1)^2} + \frac{a_3(1-A)\mu_1}{(1+b_3 \mu_1)^2} + \frac{a_2(1-A)\mu_1}{(1+b_2 \mu_1)^2} - \frac{a_1 \mu_1}{1+b_1 \mu_1} + d_1 - 1 \\
- \quad & max\left\{1 - 2\mu_1 - \frac{a_3(1-A)\mu_1}{(1+b_3 \mu_1)^2} + \frac{a_2(1-A)\mu_1}{(1+b_2 \mu_1)^2} + |-\frac{a_2(1-A)\mu_1}{1+b_2 \mu_1}|, \right. \\
& \left. \frac{a_3(1-A)\mu_1}{1+b_3 \mu_1} - \frac{a_2(1-A)\mu_1}{(1+b_2 \mu_1)^2} - d_1 + |-\frac{c_1 a_3(1-A)\mu_1}{(1+b_3 \mu_1)^2}|\right\}
\end{aligned}
$$

Therefore, from equation (4.5) we have

$$
\Gamma(B) \leq \frac{\dot{x}}{x} - \mu_2
$$

Now, integrating the above equation in $[0, t]$ we have

$$
\int_0^t \Gamma(B)ds \leq log\frac{x(t)}{x(0)} - \mu_2 t
$$
$$
\frac{1}{t}\int_0^t \Gamma(B)ds \leq \frac{1}{t}log\frac{x(t)}{x(0)} - \mu_2
$$
$$
\lim_{t\to\infty} \ sup \ sup \ \frac{1}{t}\int_0^t \Gamma(B)ds < -\mu_2 < 0 \ \text{if} \ \mu_2 > 0
$$

Hence, the system (4.4) will be globally asymptotically stable around the interior equilibria E^* if $\mu_2 > 0$.

4.5 Hopf bifurcation Analysis

Hopf bifurcation is a critical point where a system's stability switches and a periodic solution arises. It is a local bifurcation in which a fixed point of a dynamical system losses its stability as a pair of complex conjugate eigenvalues crosses the complex plane

imaginary axis [91, 260]. In this chapter, we have considered a_3 (predation rate of prey to the top predator) and e (harvesting effort) as the bifurcation parameters.

Theorem 4.9 The necessary and sufficient conditions for the occurrence of Hopf bifurcation at $a_3 = a_3^*$ are stated as follows:
(i) $\sigma_i(a_3^*) > 0, i = 1, 2, 3$
(ii) $\sigma_1(a_3^*)\sigma_2(a_3^*) - \sigma_3(a_3^*) = 0$
(iii) $Re\left(\frac{d\lambda_i}{da_3}\right)_{a_3=a_3^*} \neq 0, i = 1, 2, 3$
where λ_i are the roots of the characteristic equation corresponding to the interior equilibria and σ_1, σ_2 and σ_3 are defined within the **Theorem 4.7**.

Proof. At the point $a_3 = a_3^*$, the characteristic equation in **Theorem 4.7**

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0. \tag{4.6}$$

becomes

$$(\lambda^2 + \sigma_2)(\lambda + \sigma_1) = 0,$$

since at $a_3 = a_3^*$, $\sigma_1(a_3^*)\sigma_2(a_3^*) - \sigma_3(a_3^*) = 0$.
So, the above characteristic equation has three roots such as $\lambda_1 = i\sqrt{\sigma_2}, \lambda_2 = -i\sqrt{\sigma_2}$ and $\lambda_3 = -\sigma_1$.
Now, for $a_3 \in (a_3^* - \epsilon, a_3^* + \epsilon)$, the roots of the characteristic equation are

$$\lambda_1(a_3) = \phi_1(a_3) + i\phi_2(a_3),$$
$$\lambda_2(a_3) = \phi_1(a_3) - i\phi_2(a_3),$$
$$\lambda_3(a_3) = -\sigma_1.$$

Next, we want to verify the transversality condition $Re\left(\frac{d\lambda_i}{da_3}\right)_{a_3=a_3^*} \neq 0, i = 1, 2, 3$.
Substituting $\lambda_1(a_3) = \phi_1(a_3) + i\phi_2(a_3)$ in equation (4.6) and calculating the derivatives we have

$$P(a_3)\phi_1'(a_3) - Q(a_3)\phi_2(a_3) + U(a_3) = 0,$$
$$Q(a_3)\phi_1'(a_3) + P(a_3)\phi_2(a_3) + V(a_3) = 0,$$

where

$$P(a_3) = 3\phi_1^2(a_3) + 2\sigma_1(a_3)\phi_1(a_3) + \sigma_2(a_3) - 3\phi_2^2(a_3),$$
$$Q(a_3) = 6\phi_1(a_3)\phi_2(a_3) + 2\sigma_1(a_3)\phi_2(a_3),$$
$$U(a_3) = \phi_1^2(a_3)\sigma_1'(a_3) + \sigma_2'(a_3)\phi_1(a_3) + \sigma_3'(a_3) - \sigma_1'(a_3)\phi_2^2(a_3),$$
$$V(a_3) = 2\phi_1(a_3)\phi_2(a_3)\sigma_1'(a_3) + \sigma_2'(a_3)\phi_2(a_3)$$

Now, $P(a_3^*) = -2\sigma_2(a_3^*)$, $Q(a_3^*) = 2\sigma_1(a_3^*)\sqrt{\sigma_2(a_3^*)}$, $U(a_3^*) = \sigma_3'(a_3^*) - \sigma_1'(a_3^*)\sigma_2(a_3^*)$, $V(a_3^*) = \sigma_2'(a_3^*)\sqrt{\sigma_2(a_3^*)}$. Therefore,

$$\left(\frac{d}{da_3}(Re(\lambda_i(a_3)))\right)_{a_3=a_3^*} = -\frac{Q(a_3^*)V(a_3^*) + P(a_3^*)U(a_3^*)}{P^2(a_3^*) + Q^2(a_3^*)} \neq 0, \text{ for } i = 1, 2$$

if $Q(a_3^*)V(a_3^*) + P(a_3^*)U(a_3^*) \neq 0$ and $\lambda_3(a_3^*) = -\sigma_1 \neq 0$.

Therefore, transversality conditions hold. This indicates that Hopf bifurcation occurs at $a_3 = a_3^*$. Hence the theorem is proved.

4.6 Optimal Control Theory Applied in Harvesting

In commercial exploitation of renewable resources, our main target is to determine the optimal trade off between the present and future harvest. In this section, we study the optimal harvesting policy by considering the profit earned by harvesting. Here, quadratic cost is used and the main reason behind this is to find the analytical expression for the optimal harvesting. It is assumed that the price is a function which is inversely proportional to the available biomass of top predator population. Let h_1, p_1 and w_1 be the constant harvesting cost per unit effort, the constant price per unit biomass of predator and economic constant respectively. Thus to maximize the total discounted net revenues from the model, the optimal control problem can be formulated as follows:

$$J(e) = \int_{t_0}^{t_1} e^{-\delta_1 t} \left[(p_1 - w_1 ez)ez - h_1 e\right] dt \tag{4.7}$$

where δ_1 is the instantaneous annual discount rate. Here, the control e is bounded in $0 \leq e \leq e_{max}$ and our target is to find out an optimal control e_0 such that $J(e_0) = max_{e \in U} J(e)$ where U is the control set defined by $U = \{e$: e is measurable and $0 \leq e \leq e_{max}$, for all $t\}$. The problem (4.7) is solved by Pontryagin's maximum principle [183] subject to the initial condition system (4.4) and to optimize the objective function $J(e)$, we construct the Hamiltonian H as follows:

$$H = (p_1 - w_1 ez)ez - h_1 e + \lambda_1 \left\{x(1-x) - \frac{a_1 xy}{1 + b_1 x} - \frac{a_3(1-A)xz}{1 + b_3 x}\right\}$$

$$+ \lambda_2 \left\{\frac{a_1 xy}{1 + b_1 x} - \frac{a_2(1-A)yz}{1 + b_2 y} - d_1 y\right\}$$

$$+ \lambda_3 \left\{\frac{a_2(1-A)yz}{1 + b_2 y} + \frac{c_1 a_3(1-A)xz}{1 + b_3 x} + (\frac{a_3}{b_3}c_1 + a_2 c_2)Az - d_2 z - ez\right\}.$$

where λ_1, λ_2 and λ_3 are the adjoint variables and the transversality conditions are $\lambda_i(t_1) = 0$, for $i = 1, 2, 3$. Using the optimality condition $\frac{\partial H}{\partial e} = 0$, the optimal effort e_{δ_1} can be obtained as follows:

$$e_{\delta_1} = \frac{p_1 z - h_1 - \lambda_3 z}{2w_1 z^2}.$$

Now, the adjoint equations are

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= \delta_1\lambda_1 - \frac{\partial H}{\partial x}. \\
&= \left(\delta_1 + 2x + \frac{a_1 y}{(1+b_1 x)^2} + \frac{a_3(1-A)z}{(1+b_3 x)^2} - 1\right)\lambda_1 - \frac{a_1 y}{(1+b_1 x)^2}\lambda_2 \\
&\quad - \frac{c_1 a_3(1-A)z}{(1+b_3 x)^2}\lambda_3.
\end{aligned}
\tag{4.8}
$$

$$
\begin{aligned}
\frac{d\lambda_2}{dt} &= \delta_1\lambda_2 - \frac{\partial H}{\partial y}. \\
&= \left(\frac{a_1 x}{1+b_1 x}\right)\lambda_1 + \left(\delta_1 + d_1 + \frac{a_2(1-A)z}{(1+b_2 y)^2} - \frac{a_1 x}{1+b_1 x}\right)\lambda_2 - \frac{a_2(1-A)z}{(1+b_2 y)^2}\lambda_3.
\end{aligned}
\tag{4.9}
$$

$$
\begin{aligned}
\frac{d\lambda_3}{dt} &= \delta_1\lambda_3 - \frac{\partial H}{\partial z}. \\
&= \left(\frac{a_3(1-A)x}{1+b_3 x}\right)\lambda_1 + \left(\frac{a_2(1-A)y}{1+b_2 y}\right)\lambda_2 \\
&\quad + \left(\delta_1 + d_2 + e - \frac{a_2(1-A)y}{1+b_2 y} - \frac{c_1 a_3(1-A)x}{1+b_3 x} - \left(\frac{a_3}{b_3}c_1 + a_2 c_2\right)A\right)\lambda_3 - p_1 e + 2w_1 e^2 z.
\end{aligned}
\tag{4.10}
$$

The above results is described in the following theorem.

Theorem 4.10 There exists an optimal control e_{δ_1} and the corresponding solutions of the system (4.4) $(x_{\delta_1}, y_{\delta_1}, z_{\delta_1})$ which optimize the objective functional J over the region U. Then, there exists adjoint variables λ_1, λ_2 and λ_3 which satisfies the equations $(4.8 - 4.10)$ with transversality conditions $\lambda_i(t_1) = 0$ for $i = 1, 2, 3$, where at the optimal harvesting level the values of the state variables x, y and z are $x_{\delta_1}, y_{\delta_1}$ and z_{δ_1} and the optimal control is given by $e_{\delta_1} = \frac{p_1 z_{\delta_1} - h_1 - \lambda_3 z_{\delta_1}}{2w_1 z_{\delta_1}^2}$.

4.7 Numerical Simulation

We have solved the system (4.4), numerically using MATLAB to get better insight of the proposed model. Numerical simulations are done choosing biologically meaningful parameters from Table 4.1.

Table 4.1: Estimation of parameters

Parameter name	Value	Reference
a_1	5.0	[92]
a_2	0.1	[92]
a_3	$(0.0 - 0.4)$	[171]
b_1	3.0	[92]
b_2	2.0	[92]
b_3	2.0	[92]
d_1	0.4	[92]
d_2	0.01	[92]
c_1	0.2835	Assumed
c_2	0.28	Assumed

Using the above set of parametric values, time evolution of prey, middle predator and top predator are drawn in Figure 4.1. From this figure, it is observed that the system (4.4) is locally asymptotically stable around interior equilibrium $(0.6211, 0.06288, 7.945)$. Also, the eigenvalues of the Jacobian matrix at the interior points are $-0.2577, -0.0202 - 0.0761i, -0.0202 + 0.0761i$ that is the conditions of local stability are satisfied.
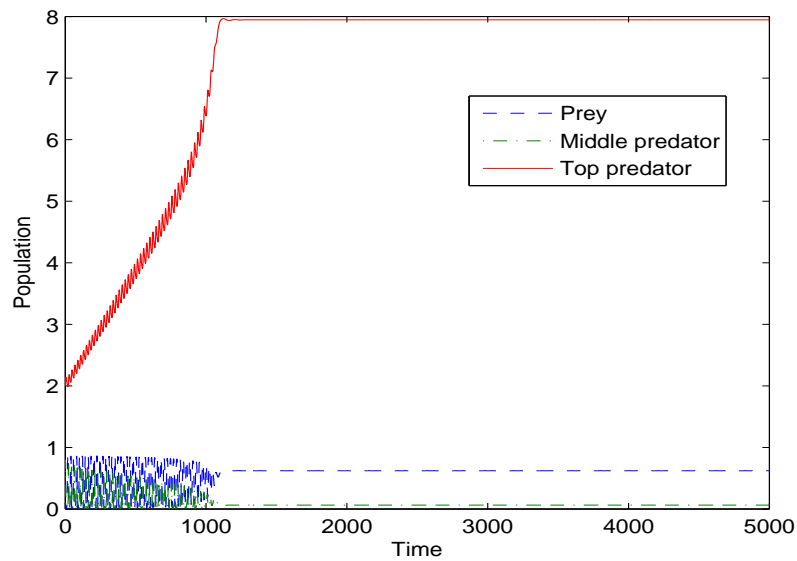

Figure 4.1: Time evolution of prey, middle predator and top predator.

Taking different initial conditions and parametric values given in Table 4.1, Figure 4.2 has been drawn. From this figure, it is observed that all the solutions converges to the interior equilibrium point. Also from **Theorem 4.8**, we have calculated $\mu_2 = 0.87 > 0$ and so it can be concluded that the interior equilibrium is globally asymptotically stable.
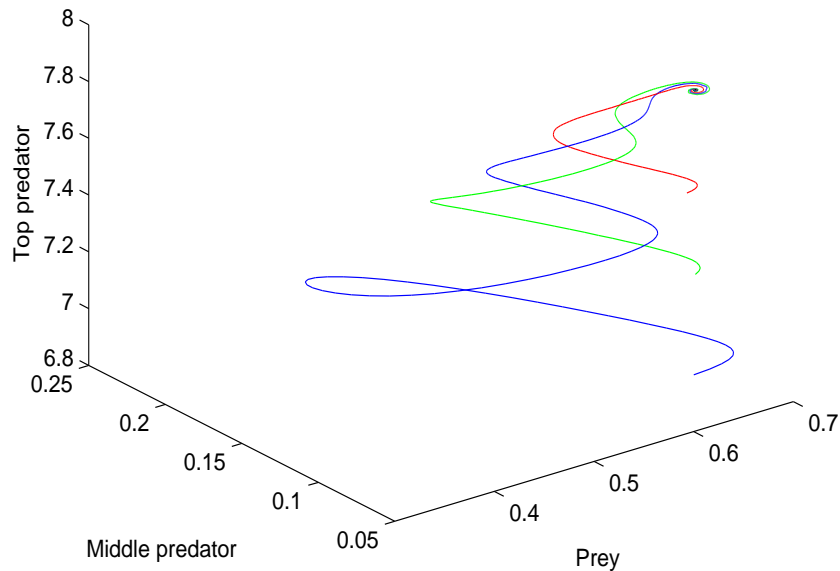

Figure 4.2: Global stability of interior equilibrium point is shown.

Form Figure 4.3, it is seen that when we change the predation rate a_3 of the prey to the top predator from 0.0 to 0.4 then the system (4.4) undergoes bifurcation. According to **Theorem** 4.9, we have calculated the critical value of the predation rate a_3 is 0.088. So, in the absence of alternative food the predation rate (a_3) makes the system unstable. Also, the system (4.4) shows complex dynamics in the range $0 \leq a_3 \leq 0.03$ which is observed in Figure 4.3. In higher predation rate of prey to the top predator ($0.32 \leq a_3 \leq 0.4$) then prey population may extinct. The system shows stable steady state for prey population in ($0.08 \leq a_3 \leq 0.21$). Again, the system shows stable steady state for middle predator population in ($0.09 \leq a_3 \leq 0.13$). But in higher predation rate ($0.13 < a_3 \leq 0.4$) middle predator population may extinct. Also, the system shows stable steady state for top predator population in ($0.08 \leq a_3 \leq 0.21$). Oscillatory behaviour of top predator population is observed in the range ($a_3 > 0.21$).
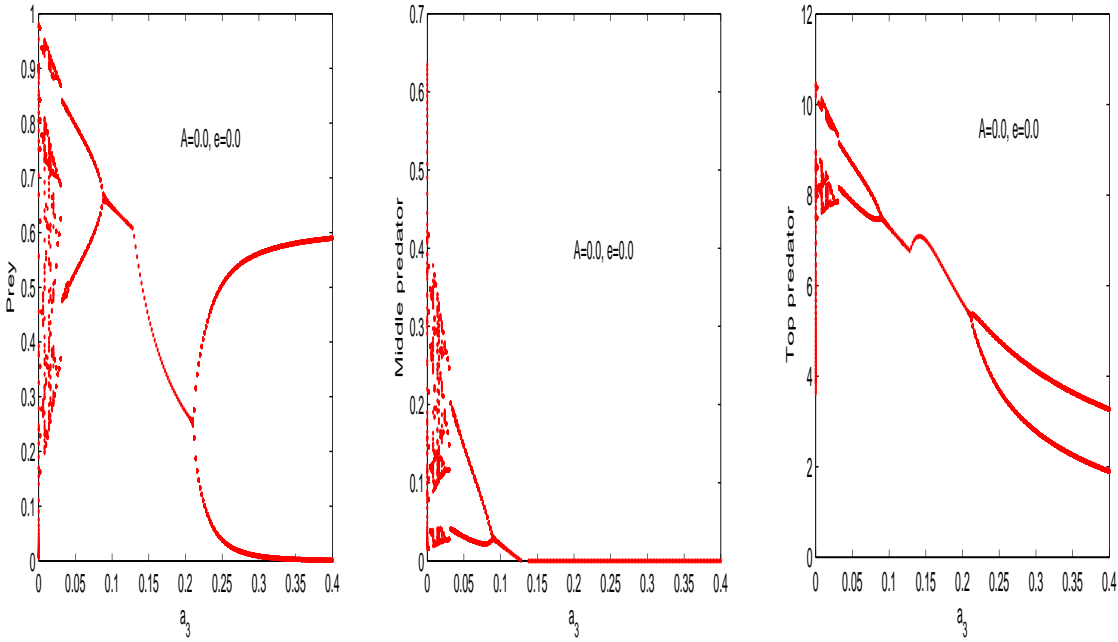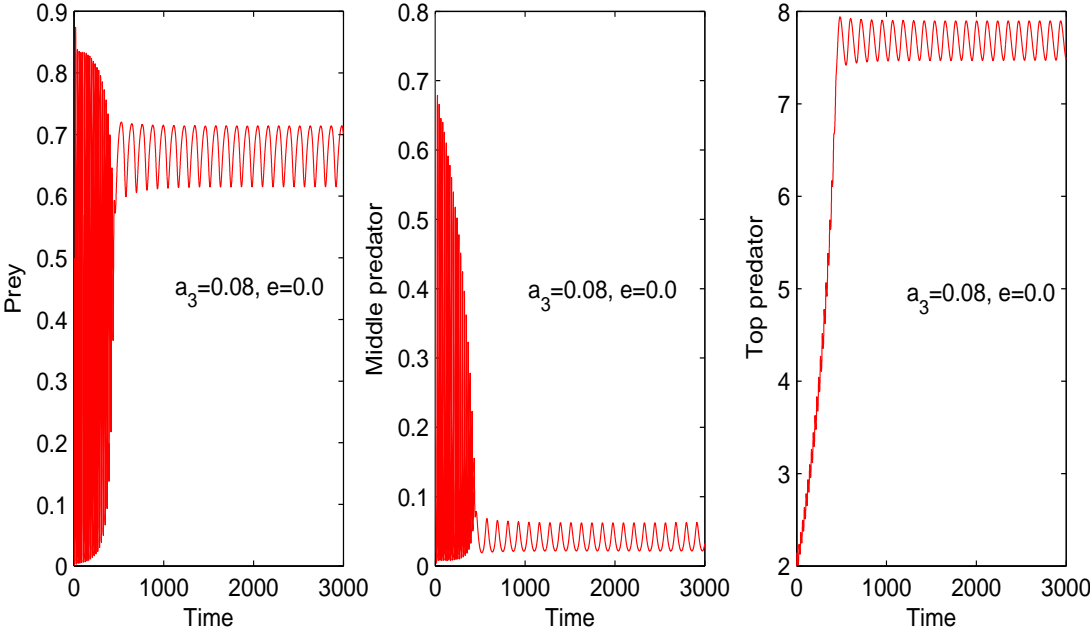
Figure 4.3: Bifurcation diagram with respect to a_3.

Figure 4.4: Time evolution of prey, middle predator and top predator.

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Using the same set of parametric values given in Table 4.1 except $a_3 = 0.08$ the unstable solution curve of top-predator population with respect time in the absence of additional food has been shown in Figure 4.4. But supplying additional food ($A = 0.13$) to top predator makes the system stable which is also shown in Figure 4.5. So, it is concluded that additional food may act as controller of the dynamics of the model.
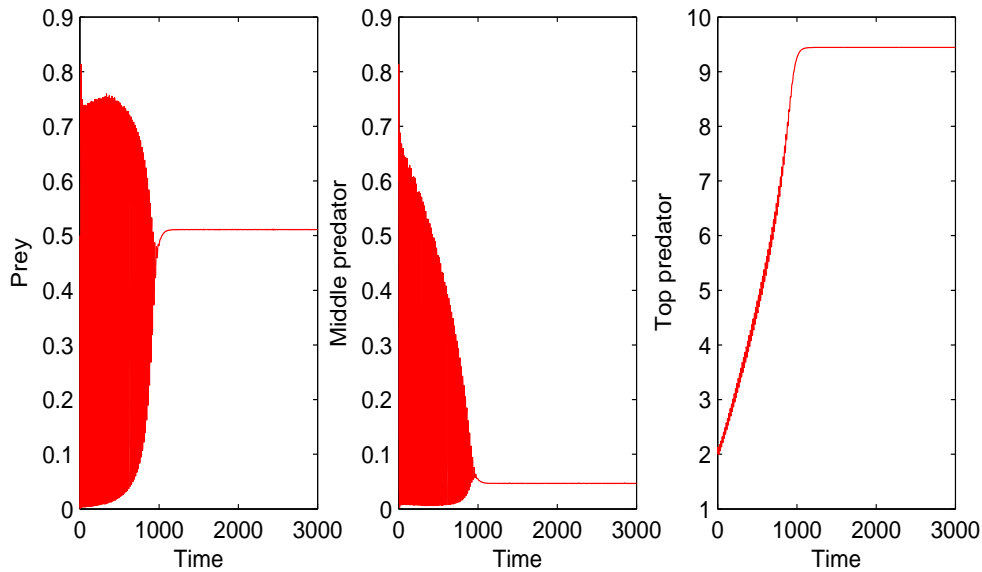

Figure 4.5: Stable solution of system (4.4) in the presence of additional food.

If the consumption rate of prey to the predator (i.e., a_3) is 0.001 then the change of harvesting effort (e) from 0 to 0.01 in the absence of additional food makes the system unstable which have been shown in Figure 4.6. We observe from this figure that prey, middle predator and top predator shows coexistence with complex dynamical behaviour, especially in spite of danger or hardship if $e < 0.0028$, oscillatory behaviour in $0.0028 \leq e < 0.003375$ and shows stable steady state for $0.003375 \leq e < 0.006675$ and prey and top predator population will be extinct if $e \geq 0.006675$.

In the presence of additional food the change of prey population have been presented in Figure 4.7 by using same set of parametric values used in Figure 4.6. From this figure when $A = 0.1$, it is observed that the prey population shows oscillatory behaviour in ($0 \leq e \leq 0.00036$ and $0.0043 < e < 0.00475$), struggle of existence in $0.0016 \leq e < 0.0043$ and this figure shows stable coexistence for $0.00036 < e < 0.0016$, $0.00476 < e < 0.00772$ and for $e \geq 0.00772$ prey population will extinct. But, when the source or amount of additional food increases then the prey population able to survive even if the harvesting effort is high.
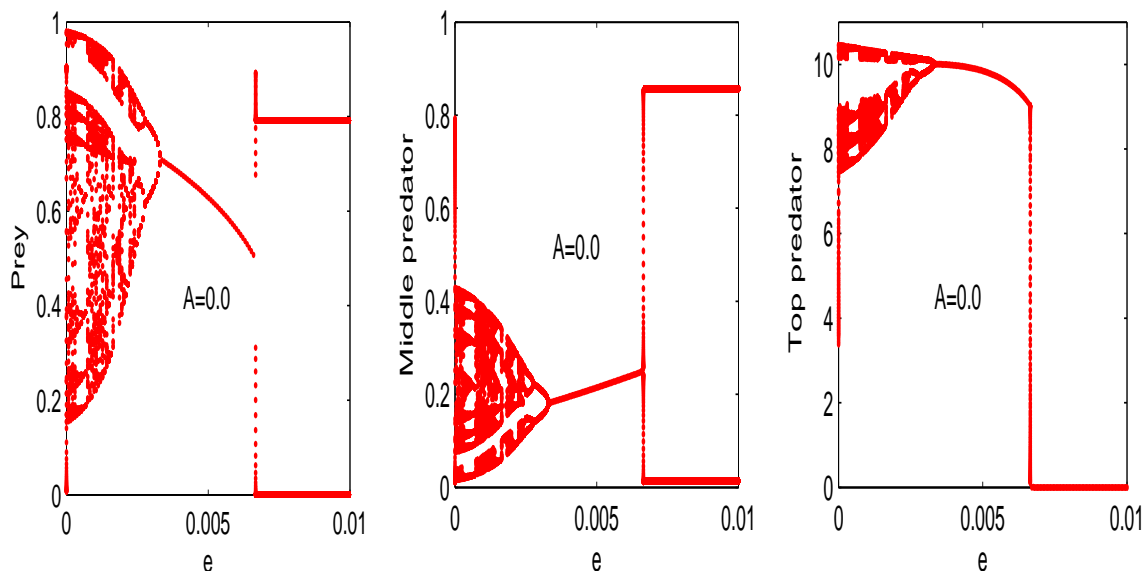
Figure 4.6: Bifurcation diagram of the system (4.4) with respect to e.
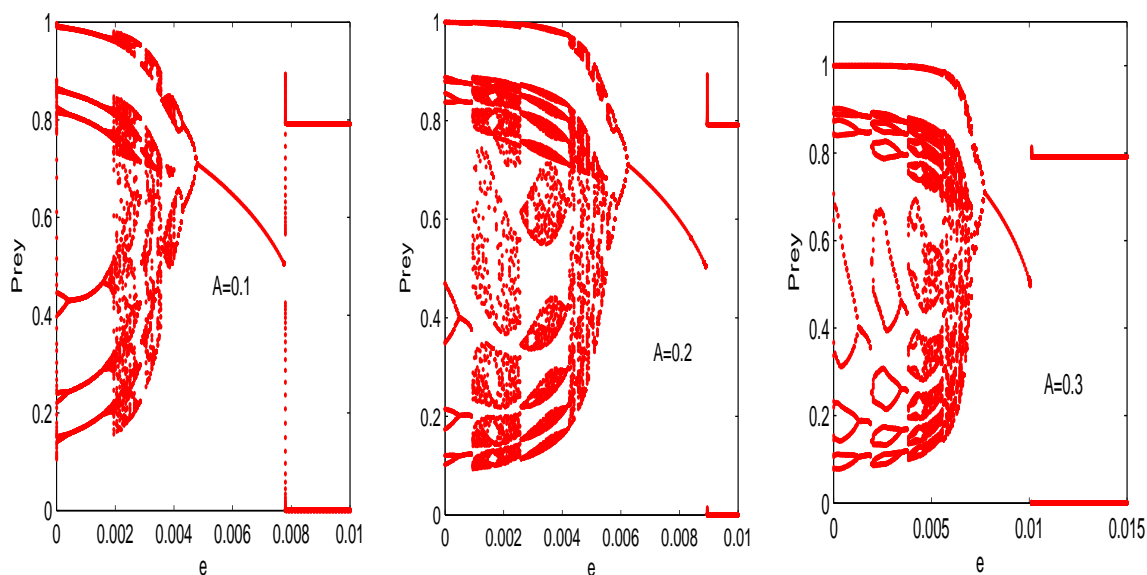
Figure 4.7: Bifurcation diagram of prey with respect to harvesting effort (e).

Again, in the presence of additional food the change of middle predator population have been shown in Figure 4.8. From this figure when $A = 0.1$, it is seen that the middle predator population shows oscillatory behaviour in ($0.00036 < e < 0.0016$ and $0.0043 < e < 0.00475$), struggle for existence for $0.0016 \leq e < 0.0043$ and this figure

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shows stable solution for and for $e > 0.00476$ middle predator population cannot be extinct. But when the value of source of additional food increases then the critical value of harvesting effort increases and there will be no extinction risk for middle predator population. So, the supply of additional food to the top predator prevent the extinction risk of middle predator and also decrease the predation pressure of middle predator.
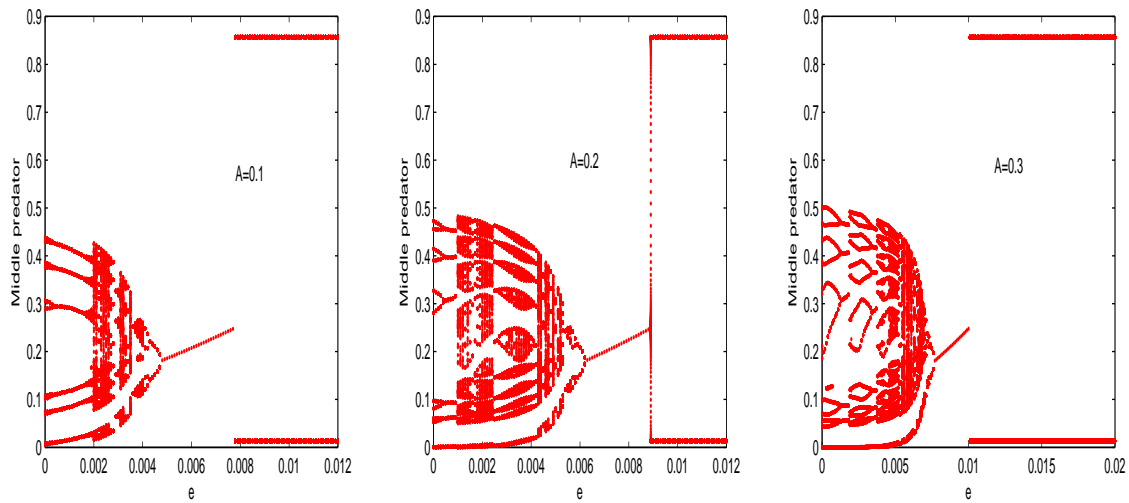

Figure 4.8: Bifurcation diagram of middle predator with respect to harvesting effort (e).
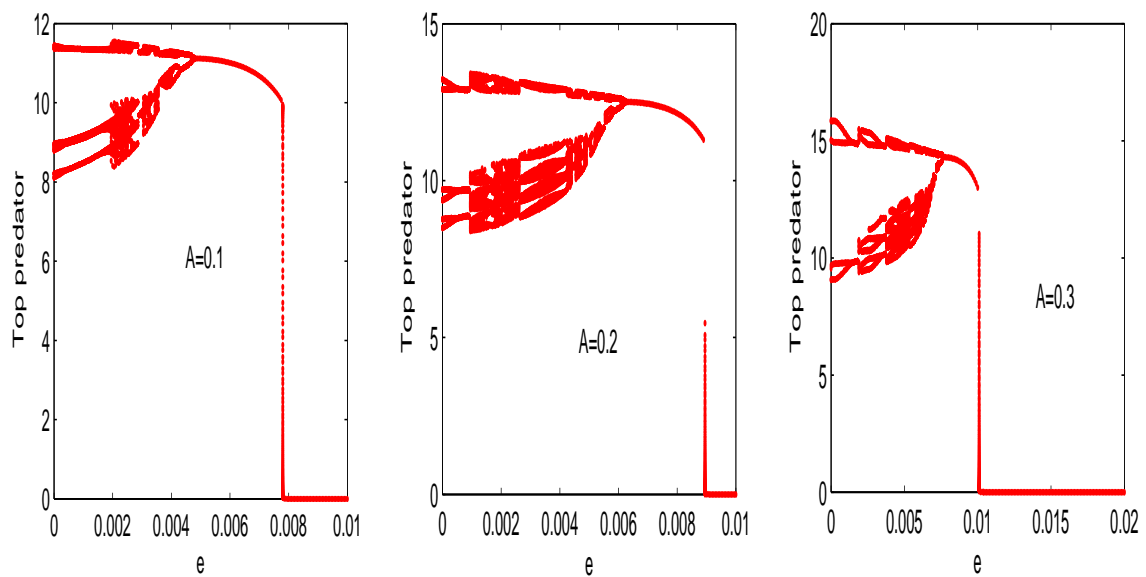

Figure 4.9: Bifurcation diagram of top predator with respect to harvesting effort (e).

Also, in the presence of additional food the change of top predator population have been also given in Figure 4.9. From this figure when $A = 0.1$, it is seen that the top predator population shows oscillatory behaviour in $(0.00036 < e < 0.0016$ and $0.0043 < e < 0.00475)$, struggle for existence for $0.0016 \leq e < 0.0043$ and this figure shows stable solution for $0.00475 \leq e < 0.00772$ and for $e \geq 0.00772$ top predator population will extinct. But, when the value of source of additional food increases then the critical value of harvesting effort increases and the top predator population extinct for more higher value of the harvesting effort. Thus, the supply of additional food to the top predator may help to exist even if harvesting effort is high.

Here, we have considered a set of parametric values to solve optimal control problem as $\delta_1 = 0.01, p_1 = 3.5, w_1 = 7.5, h_1 = 0.1$ and other values are same as Figure 4.1. To find the solution of the optimal control problem, first we use forward forth order Runge-Kutta method to solve the state variables of equation (4.4) with non-negative initial conditions. Then we solve the adjoint variables equations from $(4.8 - 4.10)$ using backward forth order Runge-Kutta method with the conditions $\lambda_i(t_1) = 0.0$ for $i = 1, 2, 3$. For the said parametric values, Figure 4.10 has been drawn which are the state variables in the presence of optimal harvesting. Figure 4.11 shows the variation of optimal harvesting with the change of additional food. From this figure, it is seen that the harvesting rate increases as the supply of additional food increases.
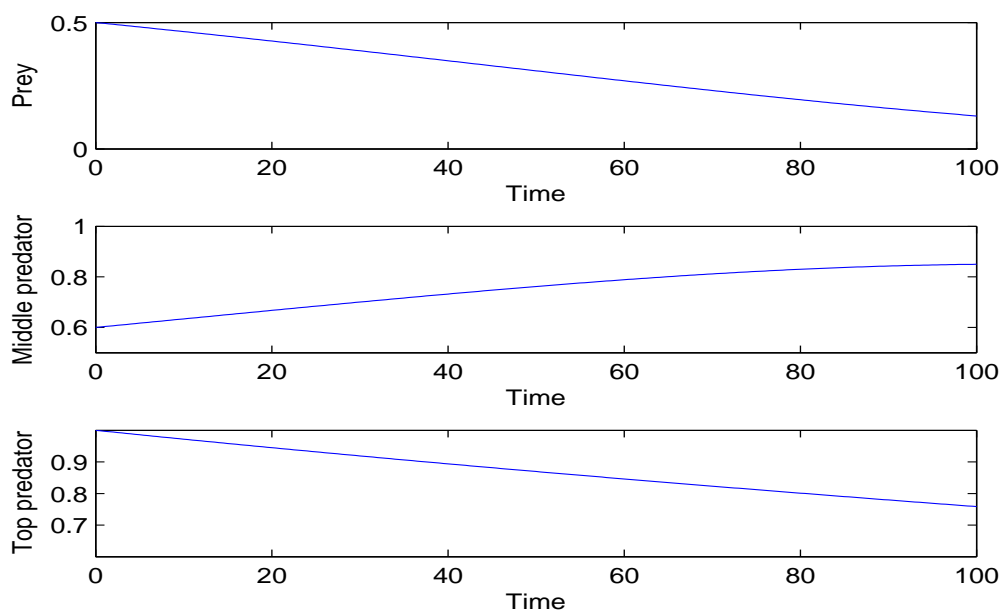

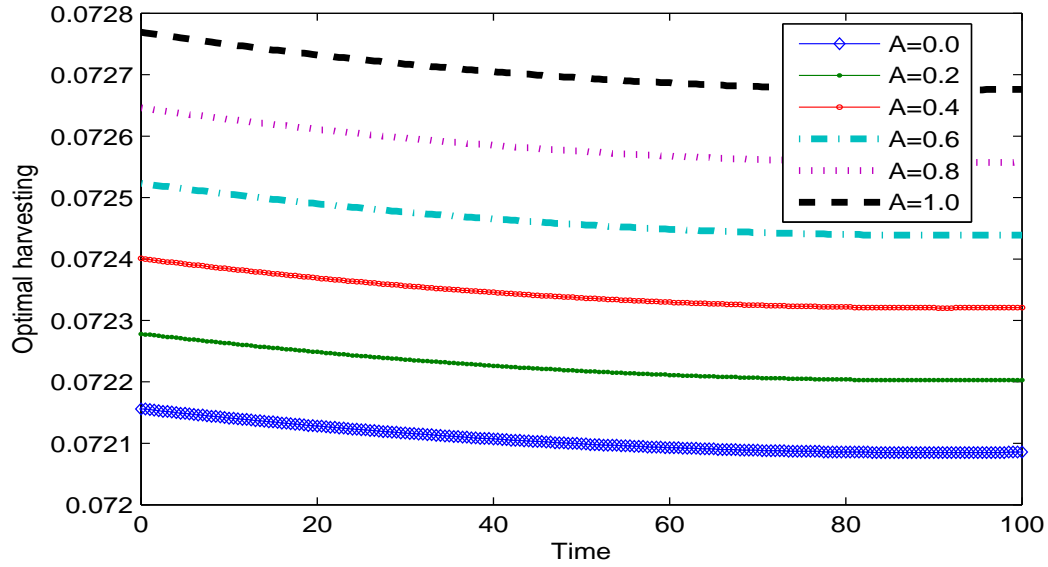Figure 4.10: Represents state variables.

Figure 4.11: Variation of optimal harvesting with additional food.
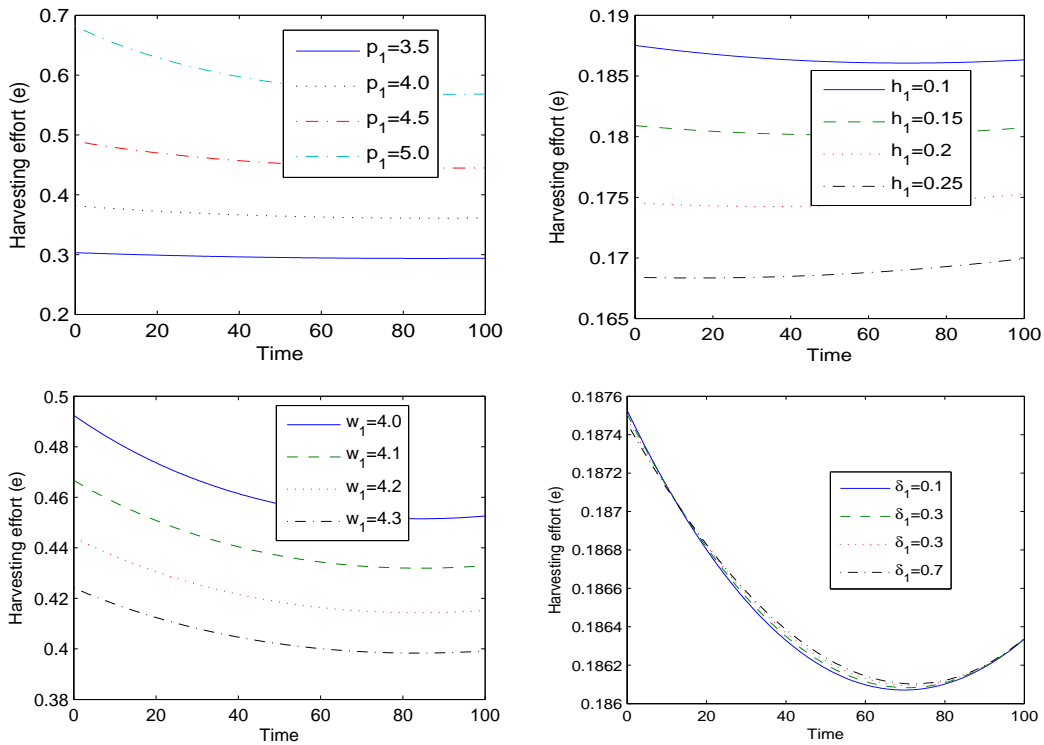
Figure 4.12: Variation of harvesting effort with respect to different cost.

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Again, from Figure 4.12, it is observed that the harvesting effort (e) increases if the constant price per unit biomass of top predator (p_1) and the instantaneous annual discount rate δ_1 increases. Also, it is observed that constant harvesting cost per unit effort (h_1) and economic constant (w_1) increases then the harvesting effort gradually decreases. Next Figure 4.13 represents the adjoint variables of the optimal control problem and it is seen that ultimately their values becomes zero at the end of the time.
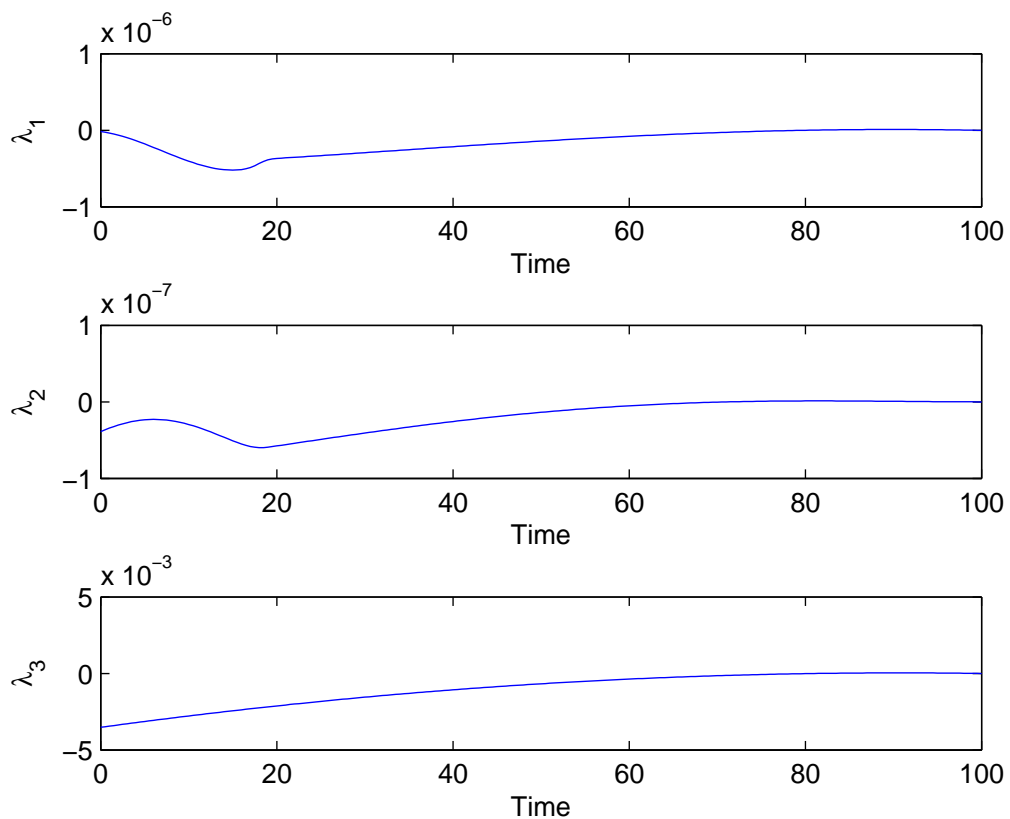
Figure 4.13: Adjoint variables.

4.8   Conclusion

In this work, we intend to study a mathematical model on prey predator system to be consisted of prey, middle predator and top predator in which some additional food is supplied to the top predator population. This type of mathematical model is the real example of the interaction between Cod fish, Herring fish and Sprat fish. Here, we have considered the ecosystem where the middle predator acts as a prey as well as predator and the top predator consumes both prey and middle predator. The non-negativity and

boundedness of solutions of the system have been shown theoretically. Different equilibrium points are determined and their local stability analysis have been done. Hopf bifurcation analysis has been done with respect to the predation rate (a_3) of prey to the top predator and the harvesting effort (e) of the top predator. Also, Pontryagins maximum principle is used to obtain the optimal harvesting effort such that the discounted net revenue will be maximum and top predator population will not be extincted.

From this study, it is observed that the supply of some additional food to the top predator may be very useful to stabilize the system dynamics which was originally unstable in absence of additional food. From this result, it is concluded that an additional food may control the instability or chaos of the system (4.4). Also, from numerical results, it is concluded that the supply of additional food to the top predator decrease the predation pressure on the prey as well as middle predator. The extinction risk of the top predator population has been effectively reduced due to the additional food. Again, from the optimal control problem, it can be concluded that for a constant value of additional food, if we increase the cost of unit biomass of top predator (p_1) and annual instantaneous discount rate (δ_1), then the harvesting effort gradually increases. But if the economic constant (w_1) and cost of harvesting effort per unit biomass (h_1) increases, then the harvesting effort gradually decreases. This study may be useful for conservation biologists for conservation of species. Another application of this model may be found in a fishery to increase the harvesting rate by supplying suitable additional food.

Gateway from Chapter 4 to Chapter 5
Apart from additional food, the role of refuge is of great concern in population dynamics. In the next chapter, the effects of refuge of predator population and the supply of additional food for super predator have been studied including the harvesting of super predator.

Chapter 5

Effects of Additional Food in a Predator-Prey System Incorporating Refuge and Harvesting

5.1 Introduction

In the present days, the ecological modelling is highly demandable area of research to the applied mathematicians and theoretical ecologists due to its universal existence and importance. Mathematical models help better understanding of dynamical behaviors of a real life problems. It is seen that the functional response has great importance on ecological modelling. In predator-prey model, it is the intake rate of a predator as a function of prey density. A predator prey model with predator interference and Holling type I functional response was explored by Gunog and DeAngelis [85]. Madhusudanan and Vijaya [145], Panja and Mondal [175], Liu et al. [135] investigated the effects of Holling type II functional response on predator-prey model. Morozov [156] studied the impact of Holling type III functional response on predator-prey interaction. Then, Baek [16] investigated the influence of Holling type IV functional response and impulsive perturbation on predator-prey dynamics. From these literature survey, it is observed that, these functional responses are dependent on only prey density. But, in reality the intake rate may depend on prey and predator both. Including the effects of prey and predator both, Flores and Gonzlez-Olivares [71] reported the effects of ratio dependent functional response on predator-prey dynamics. Again, the different types of functional responses and stability of a predator-prey system was studied by Oaten and Murdoch [167]. There are also many other types of functional responses such as Bedington-DeAngalis [88], Crowley-Martin [239], Non-monotonic [193] etc.

It is also noted that refuge plays a vital role on predator prey dynamics. It is described as a place where an organism can escape from predation. In 1988, Sih et al. [216] investigated the impact of prey refuge on sunfish and salamander larvae. Then Huang et al. [100] studied a predator-prey model considering a constant prey refuge. After that Kar [113] worked on the effects of a prey refuge on a harvested predator-prey system. Again, Jana et al. [107] reported the impact of prey refuge on a predator-prey system including stability and Hopf bifurcation. An example of a constant prey refuge is the gypsy moth which consumes bark flaps and any other suitable objects as a protection against predation by mice and birds [31]. Except these, there are also many researchers [55, 58, 157, 208, 209, 214] who studied the dynamics of predator-prey system considering refuge. Now, from the literature survey it is seen that the refuge concept has been utilized in two dimensional predator-prey systems. So, our main interest is to investigate the effects of refuge on a three dimensional predator-prey system.

Since prey refuge can make the crisis of food for the predator population, so the additional food may be necessary for the existence of predator in a predator-prey system. Already, some works have been done considering additional food. In 2007, Srinivasu et al. [223] studied biological control of extinction of a predator-prey system. Then, Samanta et al. [204] investigated how the supply of additional food to the predator prevents the disease of prey population. Then, Sahoo and Poria [198] reported the control of chaos of a three species predator-prey system through the supply of additional food. Chakraborty and Das [37] explored the effects of supplying additional food to a predator-prey system with prey refuge. Then, Kumar et al. [124] studied the dynamics of a ratio dependent predator-prey model with and without the supply of additional food.

To helps the economic developments of a country the study of harvesting is very essential. In this regard, it is an important research topic for conservation biologists and ecologists. But, the continuous harvesting of a species leads the extinction of that species. So, the investigation about a suitable optimal harvesting policy is very necessary. Choudhury and Ray [44] reported the impacts of combined harvesting on a predator-prey model. Then, Gakkhar and Singh [78] explored the effects of predator harvesting on a two prey one predator system. Chakraborty et al. [34] studied the influence of prey harvesting in a ratio dependent predator-prey system. Then, Gupta et al. [86] explored the effects of nonlinear predator harvesting on a complex predator-prey system. After that, the impact of harvesting on a reserved area for prey in the presence of toxicity has been investigated by Yang and Jia [255] in 2016.

From the literature review, it is observed that many investigations have been done on predator-prey dynamical system. But till now, there exists some vacuums in the literature as follows:

It is seen that there exists some research papers in which prey dependent functional response has been considered and also, in some papers prey and predator dependent

functional response has been considered. Again, there exists very few number of articles where the effects of supplying additional food to the predator has been considered in the presence of refuge. But, there is no work considering additional food to the super predator in the presence of refuge on predator and harvesting on super predator including prey, predator dependent functional response.

To conquer these vacuums a three species predator prey model has been developed in this chapter. Here, it is considered that the super predator consumes only the predator and also the predator consumes only prey. It is also assumed that predator population shows a refuge behavior when they are attacked by the super predator. Due to the presence of refuge behavior, some constant amount of additional food has been assumed to be supplied to the super predator. With this consideration, the predator population is also benefited by this additional food. Here, also Beddington-DeAngelis type functional response and harvesting of super predator have been considered.

5.2 Model Formulation

In this chapter, a three species predator-prey model such as prey (x), predator (y) and super predator (z) have been considered. It is assumed that predator and super predator population take the prey and predator respectively. Though now and then preys are captured by super predator but that is very small. Henceforth, this is not considered here. Here, the functional responses for predator and super predator are considered as Beddington-DeAngelis type. Also a constant rate of refuge (m_1) has been considered by the predator population (y) to the super predator population (z). Due to refuge of predator population, super predator (z) cannot consume the sufficient amount of food. For this reason, it is assumed that the some additional food A is supplied to the super predator (z). Predator population is also benefited by some portion of the additional food $(1-\rho)A$ where ρA is applied to the super predator (z) and $0 < \rho < 1$. It is assumed that the super predator (z) will be harvested with the catchability coefficient (q_1) and harvesting effort (E). Here, r, k, a_1 and c_1 be the intrinsic growth rate, carrying capacity, consumption rate and conservation rate of prey population respectively. Also, a_2, c_2, d_1 and d_2 be consumption rate of predator population, conservation rate of predator population, death rate of predator population and death rate of super predator population respectively. The constants $b_1, b_2, \alpha_1, \alpha_2, \alpha_3$ and α_4 are the half saturation constants for the functional responses. In view of these considerations, the system is governed by the

following differential equations

$$
\left.\begin{array}{rcl}
\dfrac{dx}{dt} & = & rx(1 - \dfrac{x}{k}) - \dfrac{c_1 a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} \\[3mm]
\dfrac{dy}{dt} & = & \dfrac{a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} - \dfrac{a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + (1 - \rho)Ay - d_1 y \\[3mm]
\dfrac{dz}{dt} & = & \dfrac{c_2 a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + \rho Az - d_2 z - q_1 Ez
\end{array}\right\} \tag{5.1}
$$

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

5.3 Boundedness of Solutions

Theorem 5.1 All solutions of system (5.1) in R_+^3 are uniformly bounded if $A < min\{d_1/(1 - \rho), (d_2 + q_1 E)/\rho\}$.

Proof. Let us construct a function

$$
W = x + c_1 y + \frac{c_1}{c_2} z. \tag{5.2}
$$

Taking time derivative of equation (5.2) we have

$$
\frac{dW}{dt} = \frac{dx}{dt} + c_1 \frac{dy}{dt} + \frac{c_1}{c_2} \frac{dz}{dt}.
$$

$$
i.e., \frac{dW}{dt} = rx(1 - \frac{x}{k}) - \frac{c_1 a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} + \frac{c_1 a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} - \frac{c_1 a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z}
$$
$$
+ c_1(1 - \rho)Ay - c_1 d_1 y + \frac{c_1 a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + \frac{c_1}{c_2}\rho Az - \frac{c_1}{c_2}d_2 z - \frac{c_1}{c_2}q_1 Ez.
$$

$$
i.e., \frac{dW}{dt} = rx(1 - \frac{x}{k}) + c_1(1 - \rho)Ay - c_1 d_1 y + \frac{c_1}{c_2}\rho Az - \frac{c_1}{c_2}d_2 z - \frac{c_1}{c_2}q_1 Ez.
$$

$$
i.e., \frac{dW}{dt} = rx(1 - \frac{x}{k}) - c_1 y\left[d_1 - A(1 - \rho)\right] - \frac{c_1}{c_2}\left[d_2 + q_1 E - \rho A\right]. \tag{5.3}
$$

Now, we introduce a positive number σ, then multiply σ with equation (5.2) and adding this to the both side of equation (5.3), we have

$$
\frac{dW}{dt} + \sigma W = rx(1 - \frac{x}{k}) + \sigma x - c_1 y\left[d_1 - A(1 - \rho) - \sigma\right] - \frac{c_1}{c_2}\left[d_2 + q_1 E - \rho A - \sigma\right].
$$

If $d_1 - A(1 - \rho) - \sigma > 0$ and $d_2 + q_1 E - \rho A - \sigma > 0$ holds that is $\sigma = min\{d_1 - A(1 - \rho), d_2 + q_1 E - \rho A\}$ then the above equation can be written as

$$
\frac{dW}{dt} + \sigma W \leq rx(1 - \frac{x}{k}) + \sigma x.
$$

$$
i.e., \frac{dW}{dt} + \sigma W \leq \frac{k(r + \sigma)^2}{4r}, \text{ since } max\left\{rx(1 - \frac{x}{k}) + \sigma x\right\} = \frac{k(r + \sigma)^2}{4r}.
$$

Therefore, solving above we have

$$W \leq \frac{k(r+\sigma)^2}{4r\sigma} + k_1 e^{-\sigma t}.$$

Now, taking $t \to \infty$ we have $W \leq \frac{k(r+\sigma)^2}{4r\sigma}$. Hence all solutions of system (5.1) are bounded within the region

$$\Phi = \{(x, y, z) \in R_+^3 : W = \frac{k(r+\sigma)^2}{4r\sigma} + \epsilon, \text{ for } \epsilon > 0\}.$$

Note: From the above analysis, it is observed that the boundedness of solutions of system (5.1) depends on the supply of additional food.

Lemma 5.1 Super predator population will be extinct if $m_1 < 1$ and $E > \frac{1}{q_1}\left[\frac{c_2 a_2}{\alpha_3} + \rho A - d_2\right]$.

Proof. From the third equation of system (5.1) we have

$$\frac{dz}{dt} = \frac{c_2 a_2 (1 - m_1) yz}{b_2 + \alpha_3 (1 - m_1) y + \alpha_4 z} + \rho Az - d_2 z - q_1 Ez.$$

$$i.e., \frac{dz}{dt} = \frac{c_2 a_2 (1 - m_1) z}{\alpha_3 (1 - m_1)\left(1 + \frac{\alpha_4 z}{\alpha_3 (1 - m_1) y} + \frac{b_2}{\alpha_3 (1 - m_1) y}\right)} + \rho Az - d_2 z - q_1 Ez.$$

$$i.e., \frac{dz}{dt} = \left[\frac{\frac{c_2 a_2}{\alpha_3}}{\left(1 + \frac{\alpha_4 z}{\alpha_3 (1 - m_1) y} + \frac{b_2}{\alpha_3 (1 - m_1) y}\right)} + \rho A - d_2 - q_1 E\right] z.$$

$$i.e., \frac{dz}{z} \leq \left[\frac{c_2 a_2}{\alpha_3} + \rho A - d_2 - q_1 E\right] dt$$

This is possible if $m_1 < 1$ and $\left(1 + \frac{\alpha_4 z}{\alpha_3 (1 - m_1) y} + \frac{b_2}{\alpha_3 (1 - m_1) y}\right) > 1$.

Therefore, integrating the above equation from $[t_0, t]$ we have

$$z(t) \leq z(t_0) exp\left(\int_{t_0}^{t}\left(\frac{c_2 a_2}{\alpha_3} + \rho A - d_2 - q_1 E\right) ds\right). \tag{5.4}$$

Now, if $E > \frac{1}{q_1}\left[\frac{c_2 a_2}{\alpha_3} + \rho A - d_2\right]$, then from equation (5.4), we have $\lim_{t \to \infty} z(t) = 0$. Hence the lemma.

Note: Conclusion of Lemma 5.1 is that higher rate of harvesting make the system free from super predator.

5.4 Equilibrium Points and Stability Analysis

In this section, we study the dynamical behavior of the model system. For this purpose all the feasible equilibria with their existence criteria are obtained and they are as follows:

The possible equilibria are

(i) The trivial equilibrium $E_0(0,0,0)$.

(ii) The axial equilibrium $E_1(k,0,0)$.

(iii) Super predator free equilibrium $E_2(\hat{x}, \hat{y}, 0)$ where $\hat{y} = \frac{b_1(1-\rho)A - b_1 d_1 + \hat{x}(a_1 + \alpha_1(1-\rho)A - \alpha_1 d_1)}{\alpha_2[d_1 - (1-\rho)A]}$ and $P\hat{x}^2 + Q\hat{x} + R = 0$ where $P = a_1 r \alpha_2$, $Q = a_1 k(a_1 c_1 - r\alpha_2) + kc_1 a_1 \alpha_1((1-\rho)A - d_1)$ and $R = kc_1 a_1 b_1((1-\rho)A - d_1)$. Now, $\hat{x}$ will be positive if $Q < 0, R > 0$ and $Q^2 \geq 4PR$.

(iv) The interior equilibrium $E^*(x^*, y^*, z^*)$ where $y^* = \frac{r(1-\frac{x^*}{k})(b_1 + \alpha_1 x^*)}{c_1 a_1 - \alpha_2 r(1-\frac{x^*}{k})}$,

$z^* = \frac{(1-m_1)[c_2 a_2 - d_2 \alpha_3 - q_1 E\alpha_3 + \rho A\alpha_3]r(1-\frac{x^*}{k})(b_1 + \alpha_1 x^*) + (b_2 \rho A - b_2 d_2 - b_2 q_1 E)(c_1 a_1 - \alpha_2 r(1-\frac{x^*}{k}))}{\alpha_4(c_1 a_1 - \alpha_2 r(1-\frac{x^*}{k}))[d_2 + q_1 E - \rho A]}$ and

x^* satisfies the equations $L_1 x^3 + L_2 x^2 + L_3 x + L_4 = 0$. Now, according to Descarte's rule this equation have one positive real root if any one of the following conditions holds

$(a) L_1 < 0, L_2 > 0, L_3 > 0, L_4 > 0$, $(b) L_1 > 0, L_2 > 0, L_3 > 0, L_4 < 0$,
$(c) L_1 < 0, L_2 < 0, L_3 > 0, L_4 > 0$, $(d) L_1 > 0, L_2 > 0, L_3 < 0, L_4 < 0$.
where $L_1 = r\alpha_2 N_{12} N_{18} - \alpha_1 N_{14}$, $L_2 = r\alpha_2 N_{11} N_{18} + kN_{12} N_{17} N_{18} - k\alpha_1 N_{14} N_{15}$
$-b_1 N_{14} - \alpha_1 N_{13} - r\alpha_2 N_{14} N_{16} - rd_1 \alpha_2 N_{14} N_{18} + (1-\rho)Ar\alpha_2 N_{14} N_{18}$,
$L_3 = kN_{11} N_{17} N_{18} - b_1 kN_{14} N_{15} - k\alpha_1 N_{13} N_{15} - b_1 N_{13} - kN_{14} N_{16} N_{17} - r\alpha_2 N_{13} N_{16} + r\alpha_2 d_1 N_{13} N_{18} - (1-\rho)Ar\alpha_2 N_{13} N_{18} + kd_1 N_{14} N_{17} N_{18} - (1-\rho)AkN_{14} N_{17} N_{18}$,
$L_4 = k(1-\rho)AN_{13} N_{17} N_{18} - kd_1 N_{13} N_{17} N_{18} - kN_{13} N_{16} N_{17} - kb_1 N_{13} N_{15}$,
$N_{11} = a_1(c_1 a_1 - r\alpha_2)$, $N_{12} = \frac{a_1 r\alpha_2}{k}$, $N_{13} = c_1 a_1 b_1 + r\alpha_1 \alpha_2 - rb_1 \alpha_2$,
$N_{14} = c_1 a_1 \alpha_1 - r\alpha_1 \alpha_2 + \frac{rb_1 \alpha_2}{k}$, $N_{15} = a_2(1-m_1)^2 r(c_2 a_2 - d_2 \alpha_3 - q_1 E\alpha_3 + \rho A\alpha_3)$,
$N_{16} = b_2 \rho A - b_2 d_2 - b_2 q_1 E$, $N_{17} = c_1 a_1 - r\alpha_2$, $N_{18} = \alpha_4 b_2^2 - \alpha_4 b_2 d_2$.

5.4.1 Local Stability Analysis

In this section, we have examined the local stability of the system around each of its equilibrium points.

Theorem 5.2 The trivial equilibrium $E_0(0,0,0)$ is always unstable.

Proof. The characteristic equation of the variational matrix of the system (5.1) at $E_0(0,0,0)$ is given by

$$(\lambda - r)(\lambda - [(1-\rho)A - d_1])(\lambda - \rho A + d_2 + q_1 E) = 0.$$

The eigenvalues of this characteristic equation are $r, [(1-\rho)A - d_1]$ and $\rho A - d_2 - q_1 E$. Since one of the eigenvalue r is positive. So, we can say that the trivial equilibrium is always unstable.

Theorem 5.3 The axial equilibrium $E_1(k, 0, 0)$ will be locally asymptotically stable if $A < min\{(d_2 + q_1 E)/\rho, (d_1 b_1 + k(d_1\alpha_1 - a_1))/((1-\rho)(b_1 + \alpha_1 k))\}$.

Proof. The characteristic equation of the variational matrix of the system (5.1) at $E_1(k, 0, 0)$ is given by

$$(\lambda + r)(\lambda - \frac{a_1 k}{b_1 + \alpha_1 k} - (1-\rho)A + d_1)(\lambda - \rho A + d_2 + q_1 E) = 0.$$

The eigenvalues of the above characteristic equation are $-r, \frac{a_1 k}{b_1 + \alpha_1 k} + (1-\rho)A - d_1$ and $\rho A - d_2 - q_1 E$. Hence the axial equilibrium point will be locally asymptotically stable if $\frac{a_1 k}{b_1 + \alpha_1 k} + (1-\rho)A < d_1$ and $\rho A < d_2 + q_1 E$.

Observation 1. The stability of predator free equilibrium depends on the quantity of additional food provided to the predator population. Lower amount than a threshold value of the parameter on additional food will make the unstable predator free equilibrium to a stable one.

Theorem 5.4 The super predator free equilibrium $E_2(\hat{x}, \hat{y}, 0)$ will be locally asymptotically stable if $E > \frac{1}{q_1}\left[\frac{c_2 a_2(1-m_1)\hat{y}}{b_2 + \alpha_3(1-m_1)\hat{y}} + \rho A - d_2\right]$ and $r - \frac{2r\hat{x}}{k} + (1-\rho)A + \frac{1}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2}\{a_1\hat{x}(b_1 + \alpha_1\hat{x}) - c_1 a_1\hat{y}(b_1 + \alpha_2\hat{y})\} < d_1 < (1-\rho)A - \frac{(r - \frac{2r\hat{x}}{k})a_1\hat{x}(b_1 + \alpha_1\hat{x})}{(r - \frac{2r\hat{x}}{k})(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2 - c_1 a_1\hat{y}(b_1 + \alpha_2\hat{y})}$

Proof. The characteristic equation of the variational matrix of the system (5.1) at $E_2(\hat{x}, \hat{y}, 0)$ is given by

$$(\lambda - A_{33})\left(\lambda^2 - \lambda(A_{11} + A_{22}) + A_{11}A_{22} - A_{12}A_{21}\right) = 0.$$

where $A_{11} = r - \frac{2r\hat{x}}{k} - \frac{c_1 a_1\hat{y}(b_1 + \alpha_2\hat{y})}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2}, A_{12} = -\frac{c_1 a_1\hat{x}(b_1 + \alpha_1\hat{x})}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2}$, $A_{21} = \frac{a_1\hat{y}(b_1 + \alpha_2\hat{y})}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2}$, $A_{22} = \frac{a_1\hat{x}(b_1 + \alpha_1\hat{x})}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2} + (1-\rho)A - d_1, A_{23} = -\frac{a_2(1-m_1)\hat{y}}{(b_2 + \alpha_3(1-m_1)\hat{y})}$ and $A_{33} = \frac{c_2 a_2(1-m_1)\hat{y}}{(b_2 + \alpha_3(1-m_1)\hat{y})} + \rho A - d_2 - q_1 E$. Then, all the eigenvalues of the characteristic equation will be negative if $A_{33} < 0, (A_{11} + A_{22}) < 0$ and $A_{11}A_{22} - A_{12}A_{21} > 0$. From this conditions it can be said that the super predator free equilibrium point will be locally asymptotically stable if $E > \frac{1}{q_1}\left[\frac{c_2 a_2(1-m_1)\hat{y}}{b_2 + \alpha_3(1-m_1)\hat{y}} + \rho A - d_2\right]$ and $r - \frac{2r\hat{x}}{k} + (1-\rho)A + \frac{1}{(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2}\{a_1\hat{x}(b_1 + \alpha_1\hat{x}) - c_1 a_1\hat{y}(b_1 + \alpha_2\hat{y})\} < d_1 < (1-\rho)A - \frac{(r - \frac{2r\hat{x}}{k})a_1\hat{x}(b_1 + \alpha_1\hat{x})}{(r - \frac{2r\hat{x}}{k})(b_1 + \alpha_1\hat{x} + \alpha_2\hat{y})^2 - c_1 a_1\hat{y}(b_1 + \alpha_2\hat{y})}$.

Observation 2. The stability of super predator free equilibrium depends on harvesting rate and the death rate of the super predator population.

Theorem 5.5 The interior equilibrium $E^*(x^*, y^*, z^*)$ will be locally asymptotically stable if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$ where σ_1, σ_2 and σ_3 are defined in subsequent steps.

Proof. The characteristic equation of the variational matrix of the system (5.1) at $E^*(x^*, y^*, z^*)$ is given by

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0.$$

where $\sigma_1 = -(B_{11} + B_{22} + B_{33})$, $\sigma_2 = B_{11}B_{33} + B_{22}B_{33} + B_{11}B_{22} - B_{23}B_{32} - B_{12}B_{21}$, $\sigma_3 = B_{12}B_{21}B_{33} + B_{11}B_{23}B_{32} - B_{11}B_{22}B_{33}$, $B_{11} = r - \frac{2rx^*}{k} - \frac{c_1a_1y^*(b_1+\alpha_2y^*)}{(b_1+\alpha_1x^*+\alpha_2y^*)^2}$, $B_{12} = -\frac{c_1a_1x^*(b_1+\alpha_1x^*)}{(b_1+\alpha_1x^*+\alpha_2y^*)^2}$, $B_{21} = \frac{a_1y^*(b_1+\alpha_2y^*)}{(b_1+\alpha_1x^*+\alpha_2y^*)^2}$, $B_{22} = \frac{a_1x^*(b_1+\alpha_1x^*)}{(b_1+\alpha_1x^*+\alpha_2y^*)^2} - \frac{a_2(1-m_1)z^*(b_2+\alpha_4z^*)}{(b_2+\alpha_3(1-m_1)y^*+\alpha_4z^*)^2} + (1-\rho)A - d_1$, $B_{23} = -\frac{a_2(1-m_1)y^*(b_2+\alpha_3(1-m_1)y^*)}{(b_2+\alpha_3(1-m_1)y^*+\alpha_4z^*)^2}$, $B_{32} = \frac{c_2a_2(1-m_1)z^*(b_2+\alpha_4z^*)}{(b_2+\alpha_3(1-m_1)y^*+\alpha_4z^*)^2}$, $B_{33} = \frac{c_2a_2(1-m_1)y^*(b_2+\alpha_3(1-m_1)y^*)}{(b_2+\alpha_3(1-m_1)y^*+\alpha_4z^*)^2} + \rho A - d_2 - q_1E$. According to Routh-Hurwith criteria all the eigenvalues of the above characteristic equations will negative or negative real parts if $\sigma_1 > 0$, $\sigma_3 > 0$ and $\sigma_1\sigma_2 - \sigma_3 > 0$.

5.4.2 Global Stability Analysis

In this section, global stability of the system around interior equilibrium points has been investigated using Lyapunov function.

Theorem 5.6 The system will be globally asymptotically stable around interior equilibrium point if $r > c_1a_1\alpha_1ky^*$, $m_1 < 1$ and $\{a_2(1-m_1)^2\alpha_3z^* - \alpha_2a_1x^*\} < 0$.

Proof. First we construct a suitable Lyapunov function as follows

$$
\begin{aligned}
F(x, y, z) &= \left[(x - x^*) - x^*log\left(\frac{x}{x^*}\right)\right] + Q_1\left[(y - y^*) - y^*log\left(\frac{y}{y^*}\right)\right] \\
&\quad + Q_2\left[(z - z^*) - z^*log\left(\frac{z}{z^*}\right)\right].
\end{aligned}
$$

where Q_1 and Q_2 will be defined in the subsequent steps.
Now, taking time derivative of the function we have

$$\frac{dF}{dt} = \frac{x - x^*}{x}\frac{dx}{dt} + Q_1\frac{y - y^*}{y}\frac{dy}{dt} + Q_2\frac{z - z^*}{z}\frac{dz}{dt}.$$

Substituting the values of $\frac{dx}{dt}, \frac{dy}{dt}$ and $\frac{dz}{dt}$ in the above we have

$$
\begin{aligned}
\frac{dF}{dt} &= (x - x^*)\left\{r(1 - \frac{x}{k}) - \frac{c_1a_1y}{b_1 + \alpha_1x + \alpha_2y}\right\} \\
&\quad + Q_1(y - y^*)\left\{\frac{a_1x}{b_1 + \alpha_1x + \alpha_2y} - \frac{a_2(1-m_1)z}{b_2 + \alpha_3(1-m_1)y + \alpha_4z} + (1-\rho)A - d_1\right\} \\
&\quad + Q_2(z - z^*)\left\{\frac{c_2a_2(1-m_1)y}{b_2 + \alpha_3(1-m_1)y + \alpha_4z} + \rho A - d_2 - q_1E\right\}.
\end{aligned}
$$

$$i.e., \frac{dF}{dt} = (x - x^*)\left\{r(1 - \frac{x}{k}) - \frac{c_1 a_1 y}{b_1 + \alpha_1 x + \alpha_2 y} - r(1 - \frac{x^*}{k}) + \frac{c_1 a_1 y^*}{b_1 + \alpha_1 x^* + \alpha_2 y^*}\right\}$$

$$+ Q_1(y - y^*)\left\{\frac{a_1 x}{b_1 + \alpha_1 x + \alpha_2 y} - \frac{a_2(1 - m_1)z}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + (1 - \rho)A - d_1\right.$$

$$\left. - \frac{a_1 x^*}{b_1 + \alpha_1 x^* + \alpha_2 y^*} + \frac{a_2(1 - m_1)z^*}{b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*} - (1 - \rho)A + d_1\right\}$$

$$+ Q_2(z - z^*)\left\{\frac{c_2 a_2(1 - m_1)y}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + \rho A - d_2 - q_1 E\right.$$

$$\left. - \frac{c_2 a_2(1 - m_1)y^*}{b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*} - \rho A + d_2 + q_1 E\right\}.$$

$$i.e., \frac{dF}{dt} = (x - x^*)\left\{-\frac{r}{k}(x - x^*) - \frac{c_1 a_1 y}{b_1 + \alpha_1 x + \alpha_2 y} + \frac{c_1 a_1 y^*}{b_1 + \alpha_1 x^* + \alpha_2 y^*}\right\}$$

$$+ Q_1(y - y^*)\left\{\frac{a_1 x}{b_1 + \alpha_1 x + \alpha_2 y} - \frac{a_1 x^*}{b_1 + \alpha_1 x^* + \alpha_2 y^*}\right.$$

$$+ \frac{a_2(1 - m_1)z^*}{b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*} - \frac{a_2(1 - m_1)z}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z}\right\}$$

$$+ Q_2(z - z^*)\left\{\frac{c_2 a_2(1 - m_1)y}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} - \frac{c_2 a_2(1 - m_1)y^*}{b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*}\right\}.$$

$$i.e., \frac{dF}{dt} = -\frac{r}{k}(x - x^*)^2 + c_1 a_1(x - x^*)\left\{\frac{b_1(y - y^*) + \alpha_1(xy^* - yx^*)}{(b_1 + \alpha_1 x + \alpha_2 y)(b_1 + \alpha_1 x^* + \alpha_2 y^*)}\right\}$$

$$+ Q_1(y - y^*)\left\{\frac{a_1 b_1(x - x^*) + a_1 \alpha_2(xy^* - yx^*)}{(b_1 + \alpha_1 x + \alpha_2 y)(b_1 + \alpha_1 x^* + \alpha_2 y^*)}\right.$$

$$+ \frac{a_2 b_2(1 - m_1)(z^* - z) + a_2(1 - m_1)\alpha_3(yz^* - y^* z)}{(b_2 + \alpha_3(1 - m_1)y + \alpha_4 z)(b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*)}\right\}$$

$$+ Q_2(1 - m_1)\left\{\frac{(c_2 a_2(z - z^*))(b_2(y - y^*) + \alpha_4(yz^* - y^* z))}{(b_2 + \alpha_3(1 - m_1)y + \alpha_4 z)(b_2 + \alpha_3(1 - m_1)y^* + \alpha_4 z^*)}\right\}.$$

$$i.e., \frac{dF}{dt} \leq -\frac{r}{k}(x - x^*)^2 + c_1 a_1(x - x^*)\left\{b_1(y - y^*) + \alpha_1 y^*(x - x^*) - \alpha_1 x^*(y - y^*)\right\}$$

$$+ Q_1(y - y^*)\left\{a_1 b_1(x - x^*) + a_1 \alpha_2 y^*(x - x^*) - a_1 \alpha_2 x^*(y - y^*)\right.$$

$$- a_2 b_2(1 - m_1)(z - z^*) + a_2(1 - m_1)^2 \alpha_3 z^*(y - y^*) - a_2 \alpha_3(1 - m_1)^2 y^*(z - z^*)\right\}$$

$$+ Q_2(1 - m_1)c_2 a_2(z - z^*)\left\{b_2(y - y^*) + \alpha_4 z^*(y - y^*) - \alpha_4 y^*(z - z^*)\right\}$$

$$i.e., \frac{dF}{dt} \leq \left\{c_1 a_1 \alpha_1 y^* - \frac{r}{k}\right\}(x - x^*)^2 + \left\{Q_1 a_2(1 - m_1)^2 \alpha_3 z^* - Q_1 \alpha_2 a_1 x^*\right\}(y - y^*)^2$$

$$- Q_2(1 - m_1)a_2 c_2 \alpha_4 y^*(z - z^*)^2$$

$$+ \left\{ a_1 b_1 Q_1 + Q_1 \alpha_2 a_1 y^* - a_1 b_1 c_1 - a_1 c_1 \alpha_1 x^* \right\} (x - x^*)(y - y^*)$$

$$+ \left\{ Q_2 (1 - m_1) a_2 b_2 c_2 + Q_2 (1 - m_1) a_2 c_2 \alpha_4 z^* - Q_1 a_2 (1 - m_1)^2 \alpha_3 y^* \right.$$

$$- \left. Q_1 a_2 b_2 (1 - m_1) \right\} (y - y^*)(z - z^*).$$

If we set $Q_1 = \frac{c_1(\alpha_1 x^* + b_1)}{b_1 + \alpha_2 y^*}$ and $Q_2 = \frac{c_1(b_1 + \alpha_1 x^*)(\alpha_3(1-m_1)y^* + b_2)}{c_2(b_1 + \alpha_2 y^*)(b_2 + \alpha_4 z^*)}$ then the above equation reduces to the form

$$i.e., \frac{dF}{dt} \leq \left\{ c_1 a_1 \alpha_1 y^* - \frac{r}{k} \right\} (x - x^*)^2 + \frac{c_1(\alpha_1 x^* + b_1)}{b_1 + \alpha_2 y^*} \left\{ a_2 (1 - m_1)^2 \alpha_3 z^* - \alpha_2 a_1 x^* \right\} (y - y^*)^2$$

$$- \frac{c_1(b_1 + \alpha_1 x^*)(\alpha_3(1 - m_1)y^* + b_2)}{c_2(b_1 + \alpha_2 y^*)(b_2 + \alpha_4 z^*)} (1 - m_1) a_2 c_2 \alpha_4 y^* (z - z^*)^2.$$

Now, from the above equation $\frac{dF}{dt} \leq 0$ if $r > c_1 a_1 \alpha_1 k y^*$, $m_1 < 1$ and $\left\{ a_2(1 - m_1)^2 \alpha_3 z^* - \alpha_2 a_1 x^* \right\} < 0$.

5.5 Hopf bifurcation Analysis

In this section, the Hopf bifurcation analysis of our proposed system has been done with respect to some important parameters.

Theorem 5.7 The necessary and sufficient conditions for the occurrence of Hopf bifurcation at $a_2 = a_2^*$ are stated as follows:
(i) $\sigma_i(a_2^*) > 0, i = 1, 2, 3$
(ii) $\sigma_1(a_2^*)\sigma_2(a_2^*) - \sigma_3(a_2^*) = 0$
(iii) $Re \left(\frac{d\lambda_i}{da_2} \right)_{a_2 = a_2^*} \neq 0, i = 1, 2, 3$
where λ_i are the roots of the characteristic equation stated in **Theorem 5.5**.

Proof. The characteristic equation of system (5.1) around interior equilibrium is given by

$$\lambda^3 + \sigma_1 \lambda^2 + \sigma_2 \lambda + \sigma_3 = 0. \tag{5.5}$$

When the bifurcation parameter reaches its critical value i.e., when $a_2 = a_2^*$ then the above characteristic equation becomes

$$(\lambda^2 + \sigma_2)(\lambda + \sigma_1) = 0. \tag{5.6}$$

since at the critical value of the parameter $a_2 = a_2^*$, $\sigma_1(a_2^*)\sigma_2(a_2^*) - \sigma_3(a_2^*) = 0$.
So, the equation (5.6) has three roots such as $\lambda_1 = i\sqrt{\sigma_2}, \lambda_2 = -i\sqrt{\sigma_2}$ and $\lambda_3 = -\sigma_1$.

Let us assume that for $a_2 \in (a_2^* - \epsilon, a_2^* + \epsilon)$, the roots of the characteristic equation are

$$\lambda_1(a_2) = \phi_1(a_2) + i\phi_2(a_2),$$
$$\lambda_2(a_2) = \phi_1(a_2) - i\phi_2(a_2),$$
$$\lambda_3(a_2) = -\sigma_1.$$

Next, we want to verify the transversality condition $Re\left(\frac{d\lambda_i}{da_2}\right)_{a_2=a_2^*} \neq 0, i = 1, 2, 3$.

Substituting $\lambda_1(a_2) = \phi_1(a_2) + i\phi_2(a_2)$ in equation (5.5) and calculating the derivatives we have

$$P(a_2)\phi_1'(a_2) - Q(a_2)\phi_2(a_2) + U(a_2) = 0,$$
$$Q(a_2)\phi_1'(a_2) + P(a_2)\phi_2(a_2) + V(a_2) = 0,$$

where

$$P(a_2) = 3\phi_1^2(a_2) + 2\sigma_1(a_2)\phi_1(a_2) + \sigma_2(a_2) - 3\phi_2^2(a_2),$$
$$Q(a_2) = 6\phi_1(a_2)\phi_2(a_2) + 2\sigma_1(a_2)\phi_2(a_2),$$
$$U(a_2) = \phi_1^2(a_2)\sigma_1'(a_2) + \sigma_2'(a_2)\phi_1(a_2) + \sigma_3'(a_2) - \sigma_1'(a_2)\phi_2^2(a_2),$$
$$V(a_2) = 2\phi_1(a_2)\phi_2(a_2)\sigma_1'(a_2) + \sigma_2'(a_2)\phi_2(a_2).$$

Now, $P(a_2^*) = -2\sigma_2(a_2^*), Q(a_2^*) = 2\sigma_1(a_2^*)\sqrt{\sigma_2(a_2^*)}, U(a_2^*) = \sigma_3'(a_2^*) - \sigma_1'(a_2^*)\sigma_2(a_2^*),$
$V(a_2^*) = \sigma_2'(a_2^*)\sqrt{\sigma_2(a_2^*)}$. Therefore,

$$\left(\frac{d}{da_2}(Re(\lambda_i(a_2)))\right)_{a_2=a_2^*} = -\frac{Q(a_2^*)V(a_2^*) + P(a_2^*)U(a_2^*)}{P^2(a_2^*) + Q^2(a_2^*)} \neq 0, \text{ for } i = 1, 2$$

if $Q(a_2^*)V(a_2^*) + P(a_2^*)U(a_2^*) \neq 0$ and $\lambda_3(a_2^*) = -\sigma_1 \neq 0$.

Therefore, transversality conditions hold. This indicates that Hopf bifurcation occurs at $a_2 = a_2^*$.

Hence the theorem.

5.6 Optimal Harvesting

In commercial exploitation of renewable resources, our main target is to determine the optimal trade off between the present and future harvest. In this section, we study the optimal harvesting policy by considering the profit earned by harvesting. Here, quadratic cost is used and the main reason behind this is to find the analytical expression for the optimal harvesting. It is assumed that the price is a function which is inversely proportional to the available biomass of top predator population. Thus, to maximize the total discounted net revenues from the model, the optimal control problem can be formulated as follows:

$$J(E) = \int_{t_0}^{t_f} e^{-\psi t} \left[(p_1 - w_1 q_1 E z)q_1 E z - hE\right] dt \tag{5.7}$$

where ψ is the instantaneous discount rate, p_1 is cost of selling unit biomass of super predator, w_1 is an economic constant and h is constant harvesting cost. Here, the control E is bounded in $0 \leq E \leq E_{max}$ and our target is to find out an optimal control E_0 such that $J(E_0) = \max_{E \in U} J(E)$ where U is the control set defined by $U = \{E: E$ is measurable and $0 \leq E \leq E_{max}$, for all $t\}$. The problem (5.7) is solved by Pontryagin's maximum principle [183] subject to the conditions system (5.1). To optimize the objective function $J(E)$, we construct the hamiltonian as follows:

$$
\begin{aligned}
H &= (p_1 - w_1 q_1 Ez)q_1 Ez - hE + \lambda_1 \left\{ rx(1 - \frac{x}{k}) - \frac{c_1 a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} \right\} \\
&+ \lambda_2 \left\{ \frac{a_1 xy}{b_1 + \alpha_1 x + \alpha_2 y} - \frac{a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + (1 - \rho)Ay - d_1 y \right\} \\
&+ \lambda_3 \left\{ \frac{c_2 a_2(1 - m_1)yz}{b_2 + \alpha_3(1 - m_1)y + \alpha_4 z} + \rho Az - d_2 z - q_1 Ez \right\}.
\end{aligned}
$$

To find the optimal harvesting we equate $\frac{\partial H}{\partial E}$ to zero, then we have

$$
E = \frac{p_1 q_1 z - h - \lambda_3 q_1 z}{2 w_1 q_1^2 z^2}
$$

Corresponding adjoint equations are

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= \psi \lambda_1 - \frac{\partial H}{\partial x}. \\
&= \lambda_1 \left\{ \psi - r + \frac{2rx}{k} \right\} + \frac{a_1 y(b_1 + \alpha_2 y)}{(b_1 + \alpha_1 x + \alpha_2 y)^2}(\lambda_1 c_1 - \lambda_2). \quad\quad (5.8)
\end{aligned}
$$

$$
\begin{aligned}
\frac{d\lambda_2}{dt} &= \psi \lambda_2 - \frac{\partial H}{\partial y}. \\
&= \lambda_2 (\psi - (1 - \rho)A + d_1) + \frac{a_1 x(b_1 + \alpha_1 x)}{(b_1 + \alpha_1 x + \alpha_2 y)^2}(\lambda_1 c_1 - \lambda_2) \\
&+ \frac{a_2(1 - m_1)z(b_2 + \alpha_4 z)}{(b_2 + \alpha_3(1 - m_1)y + \alpha_4 z)^2}(\lambda_2 - \lambda_3 c_2). \quad\quad (5.9)
\end{aligned}
$$

$$
\begin{aligned}
\frac{d\lambda_3}{dt} &= \psi \lambda_3 - \frac{\partial H}{\partial z}. \\
&= \lambda_3 (\psi - \rho A + d_2 + q_1 E) + \frac{a_2(1 - m_1)y((b_2 + \alpha_3(1 - m_1)y))}{(b_2 + \alpha_3(1 - m_1)y + \alpha_4 z)^2}(\lambda_2 - \lambda_3 c_2) \\
&- p_1 q_1 E + 2 w_1 (q_1)^2 E^2 z. \quad\quad (5.10)
\end{aligned}
$$

The above results can be described briefly by the following theorem.

Theorem 5.8 There exists an optimal control E_ψ and the corresponding solutions of the system (5.1) (x_ψ, y_ψ, z_ψ) which optimize the objective functional J over the region U. Then, there exists adjoint variables λ_1, λ_2 and λ_3 which satisfies the equations $(5.8-5.10)$ with transversality conditions $\lambda_i(t_f) = 0$ for $i = 1, 2, 3$, where at the optimal harvesting level the values of the state variables x, y and z are x_ψ, y_ψ and z_ψ and the optimal control is given by $E_\psi = \frac{p_1 q_1 z_\psi - h - \lambda_3 q_1 z_\psi}{2 w_1 q_1^2 z_\psi^2}$.

5.7 Numerical Simulation

First we consider the following set of parametric values $r = 2, k = 120, c_1 = 0.1, a_1 = 1.2, b_1 = 8.0, \alpha_1 = 1.0, \alpha_2 = 0.1, a_2 = 0.1, m_1 = 0.4, b_2 = 12.0, \alpha_3 = 1.0, \alpha_4 = 0.1, \rho = 0.8, A = 0.1, d_1 = 0.5, c_2 = 0.2, d_2 = 0.001, q1 = 0.4, E = 0.2$. For the above set of parametric values Figure 5.1 has been drawn. From this figure, it is observed that our proposed system (5.1) is locally asymptotically stable. Also, we numerically calculate the the value of $\sigma_1 = 1.8889 > 0, \sigma_3 = 14683 > 0$ and $\sigma_1 \sigma_2 - \sigma_3 = 0.3007 > 0$. So **Theorem 5.5** is numerically verified. For the same parametric values if we increase the harvesting effort $E = 0.3$ then the super predator population will be extinct which has been shown in Figure 5.2.
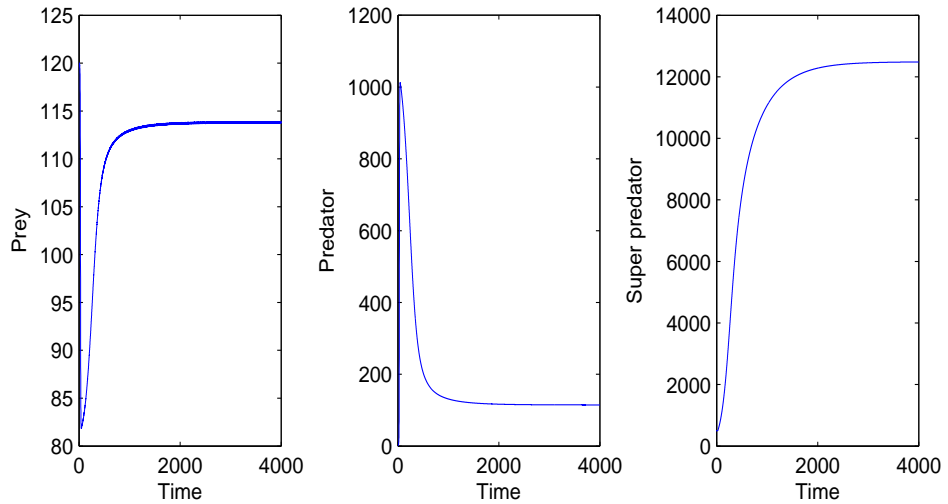

Figure 5.1: Stable interior equilibrium.

For the same set of parametric values used in Figure 5.1 except $r = 1.0, k = 100, m_1 = 0.5, c_1 = 0.01, \alpha_1 = 0.1, a_2 = 0.01, \alpha_2 = 0.1, \alpha_3 = 1.0$, Figure 5.3 has been drawn. This figure shows that the interior equilibrium point will be globally asymptotically stable when the values of a_1 varies from 0 to 0.125.
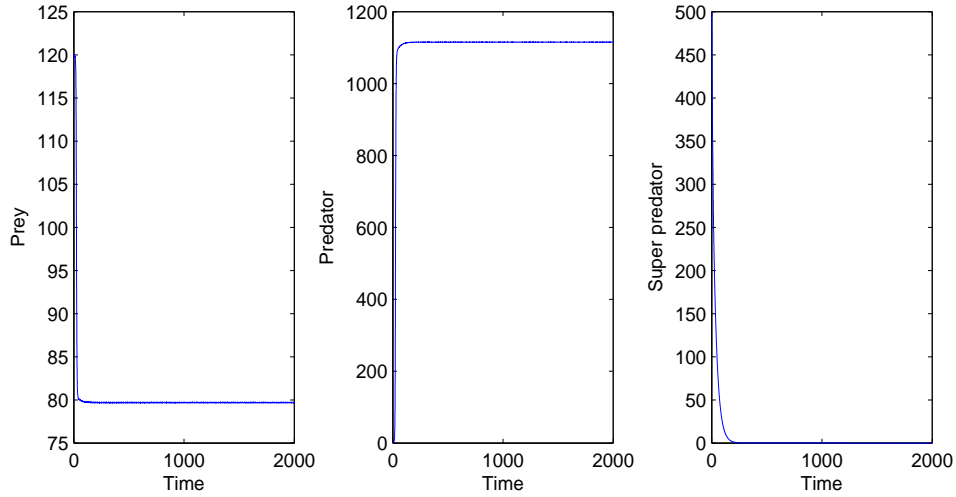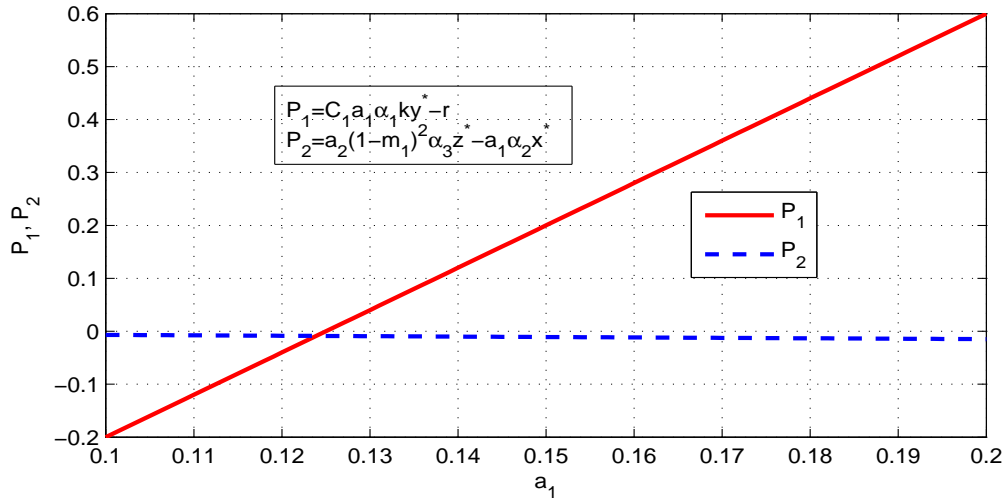
Figure 5.2: Extinction of the super predator.

Figure 5.3: Global stability condition of interior equilibrium point.

Again, using the same set of parametric values used in Figure 5.1 except $c_2 = 0.05$ the Figure 5.4 has been drawn. From this figure, it is seen that as the refuge increases then the prey population decreases, predator population increases and super predator population decreases. Because as the refuge increases then the predation rate of predator to the super predator decreases and so the predator population gradually increases and super predator population gradually decreases.
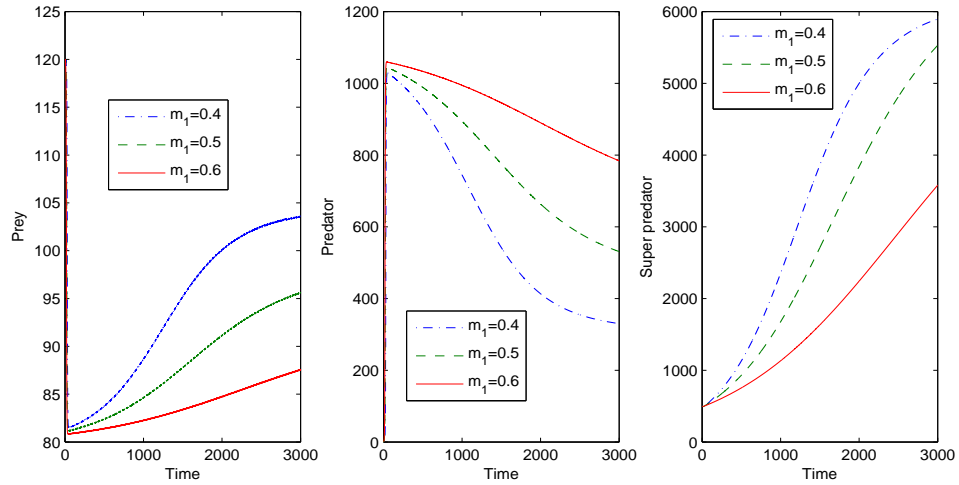
Figure 5.4: Sensitivity analysis of the system (5.1) with respect to m_1.

Then, we consider same set of parametric values used as Figure 5.1 except $r = 1.0, k = 100, a_1 = 2.0, m_1 = 0.5, A = 0.0, c_2 = 0.1, d_1 = 0.08, d_2 = 0.001$. For this set of parametric values Figure 5.5 has been drawn and from this figure, it is seen that when the value of a_2 crosses a critical value 0.1765 then the value of $\sigma_1\sigma_2 - \sigma_3$ becomes negative. So, if we change the value of a_2 from 0.1 to 0.2 then Hopf bifurcation must occurs and due to bifurcation the unstable solution curve of the system has been shown through Figure 5.6.
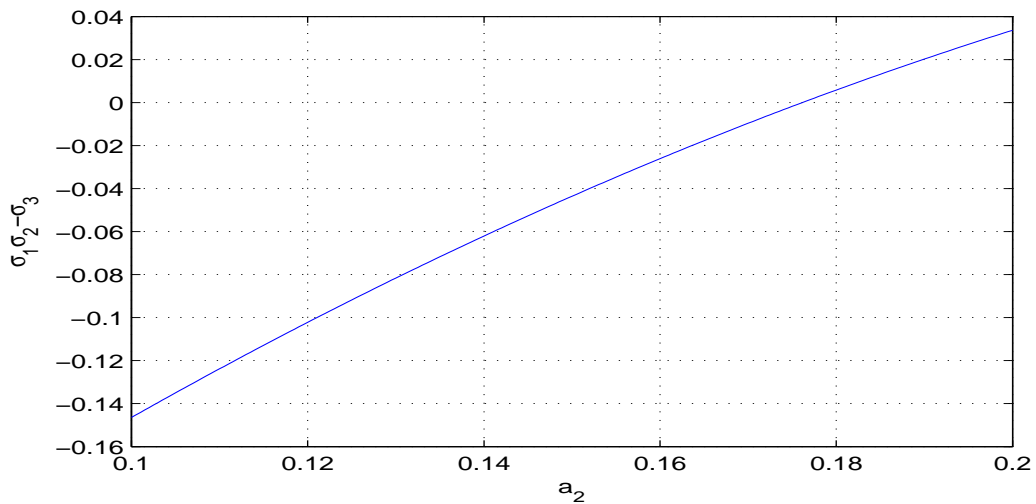

Figure 5.5: Represents the graph of $(\sigma_1\sigma_2 - \sigma_3)$ with respect to a_2.
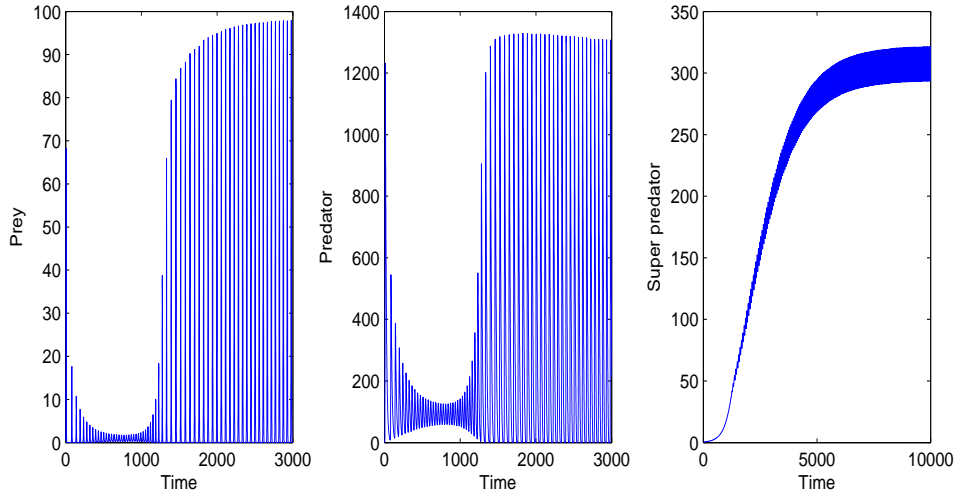
Figure 5.6: Unstable solution of system (5.1) for a_2.
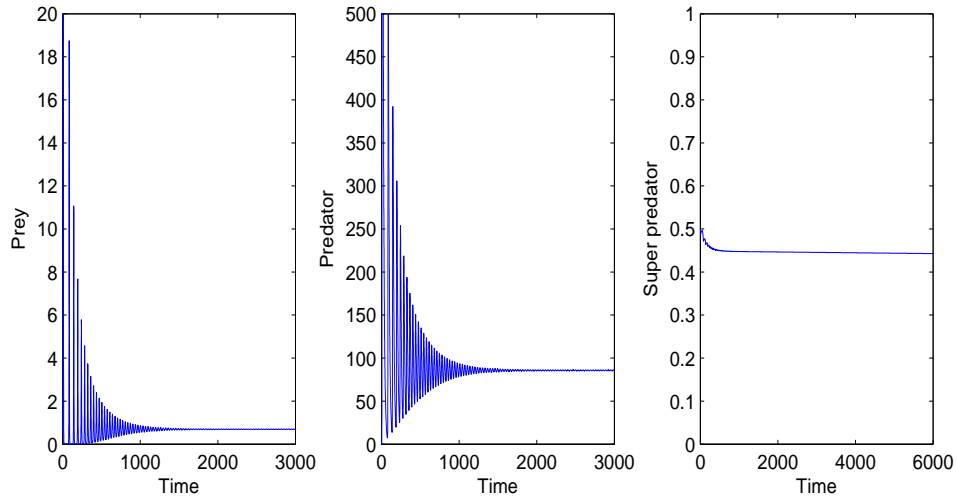
Figure 5.7: Stable solution of system (5.1) for $m_1 = 0.7$.

But, if we change the refuge rate $m_1 = 0.7$, it makes the system stable which is also shown in Figure 5.7. From this figure, it is also concluded that the refuge has a great role to make the system stable.

Again, for the parametric values $r = 1, k = 100, c_1 = 0.1, a_1 = 2.5, b_1 = 8.0, \alpha_1 = 1.0, \alpha_2 = 0.1, a_2 = 0.4, m_1 = 0.8, b_2 = 12.0, \alpha_3 = 1.0, \alpha_4 = 0.1, \rho = 0.6, d_1 = 0.08, c_2 = 0.1, d_2 = 0.001, q_1 = 0.4, E = 0.01$ in the absence of additional food the system shows unstable solution which is also shown in Figure 5.8. But, in the presence of additional food $A = 0.03$ the system becomes stable which is also shown in Figure 5.9. So, from

this figure, it can be concluded that if the refuge is high then the unstable system may be stabilized by supplying additional food to the system.
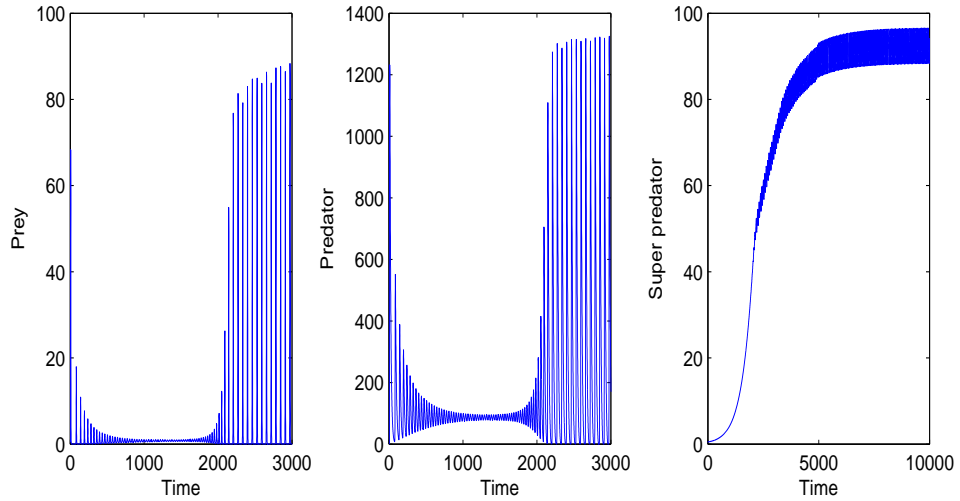
Figure 5.8: Unstable solution of the system (5.1) in the absence of additional food.
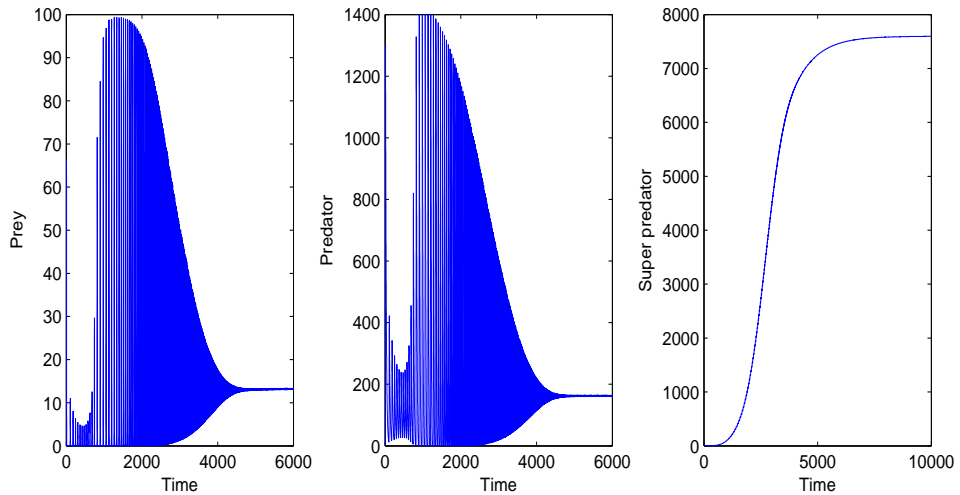
Figure 5.9: Stable solution of the system (5.1) in the presence of additional food.

To solve the optimal control problem, first we solve the state variables by forward Runge-kutta method and then using the solutions of state variables the adjoint variables are solved by backward Runge kutta method. For solving optimal control problem the parametric values has been considered as $r = 2, k = 120, c_1 = 0.1, a_1 = 1.2, b_1 = 8.0, \alpha_1 = 1.0, \alpha_2 = 0.1, a_2 = 0.1, m_1 = 0.4, b_2 = 12.0, \alpha_3 = 1.0, \alpha_4 = 0.1, \rho = 0.8, A = 0.1, d_1 =$

$0.5, c_2 = 0.2, d_2 = 0.001, q_1 = 0.4, \psi = 0.05, p_1 = 1.5, h = 0.05, w_1 = 1.2$. We have shown the state variables of the system (5.1) in Figure 5.10 when optimal harvesting is done. Variation of optimal harvesting have been shown in Figure 5.11 with different values of the additional food. From this figure, it is concluded that as the supply of additional food increases then the optimal harvesting rate increases. The change of harvesting effort with respect to different costs have been shown in Figure 5.12. From this figure, it is observed that if we increase the selling price (p_1) of unit biomass of super predator then the harvesting effort gradually increases. But if the harvesting cost (h) and economic constant (w_1) increased then the harvesting rate decreases. The graph of adjoint variables λ_1, λ_2 and λ_3 have been shown in Figure 5.13 when the optimal harvesting is done. From this figure, it is observed that the adjoint variables are monotone decreasing and according to the theory the final values are zero.
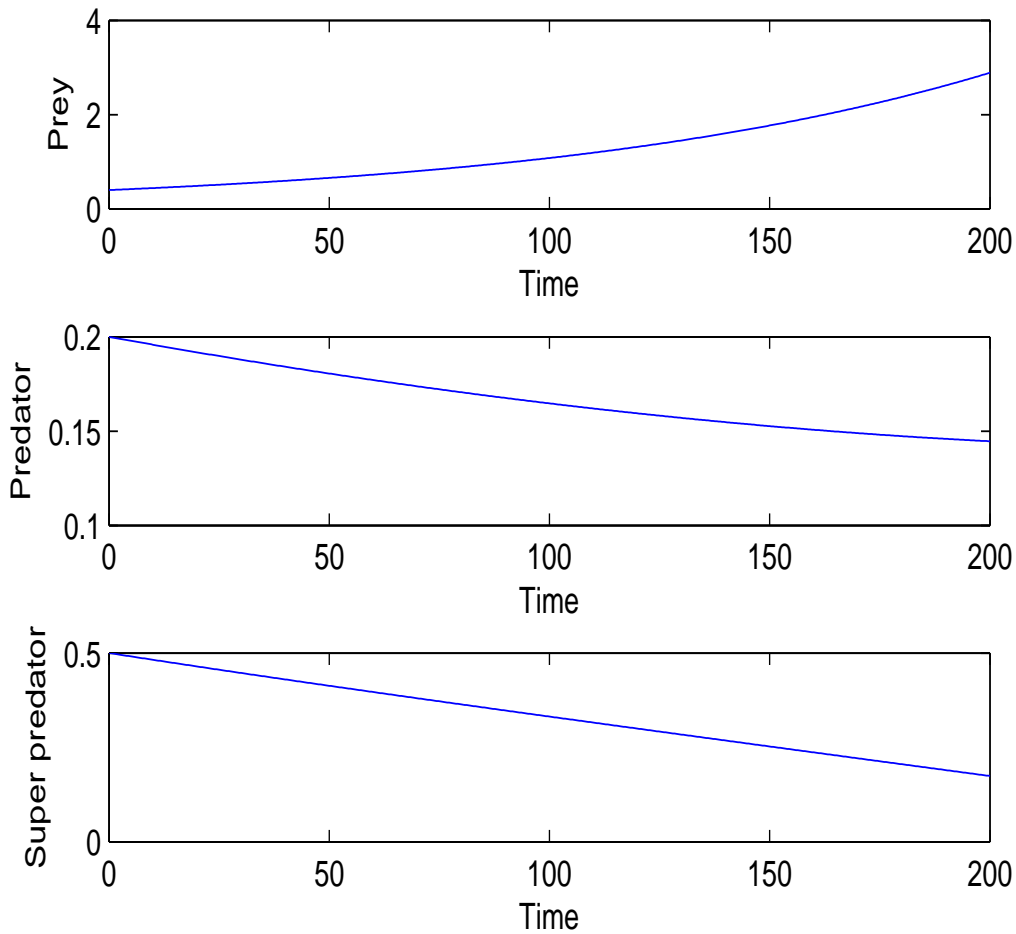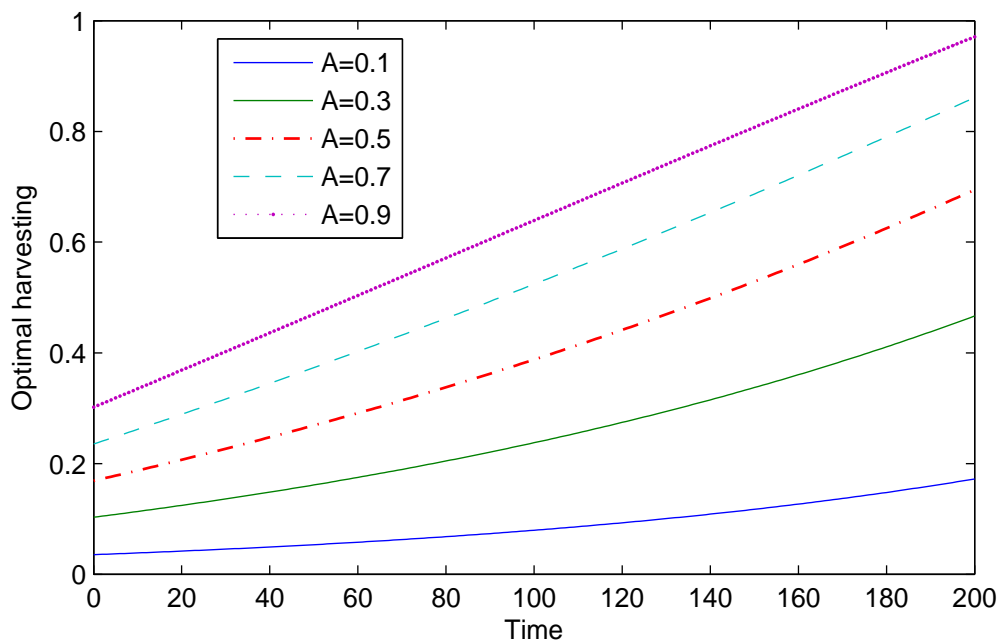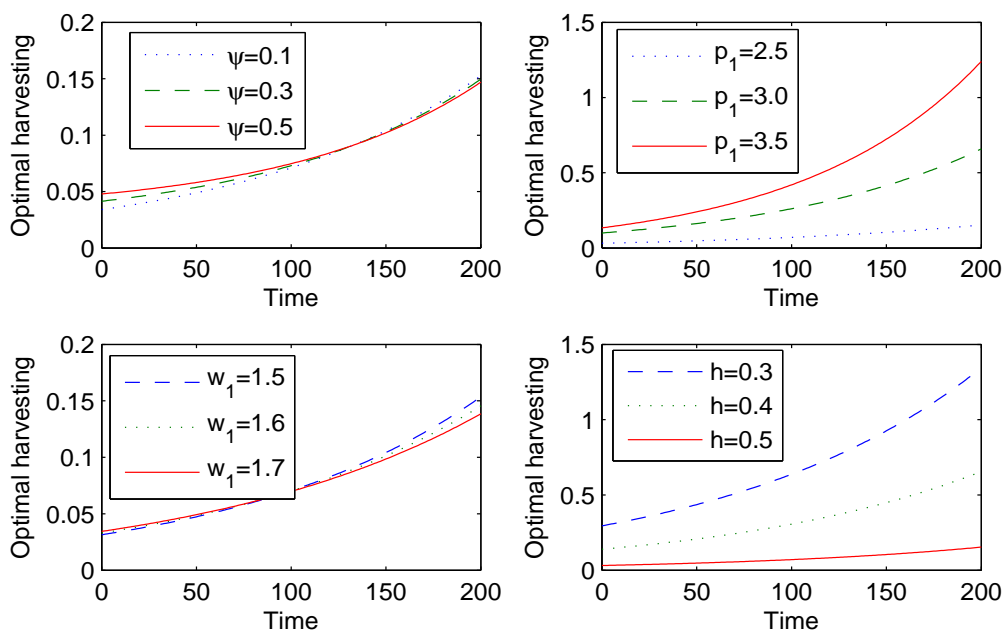

Figure 5.10: Represents the state variables.

Figure 5.11: Variation of optimal harvesting effort with the change of A.

Figure 5.12: Variation of optimal harvesting effort with respect to different cost.
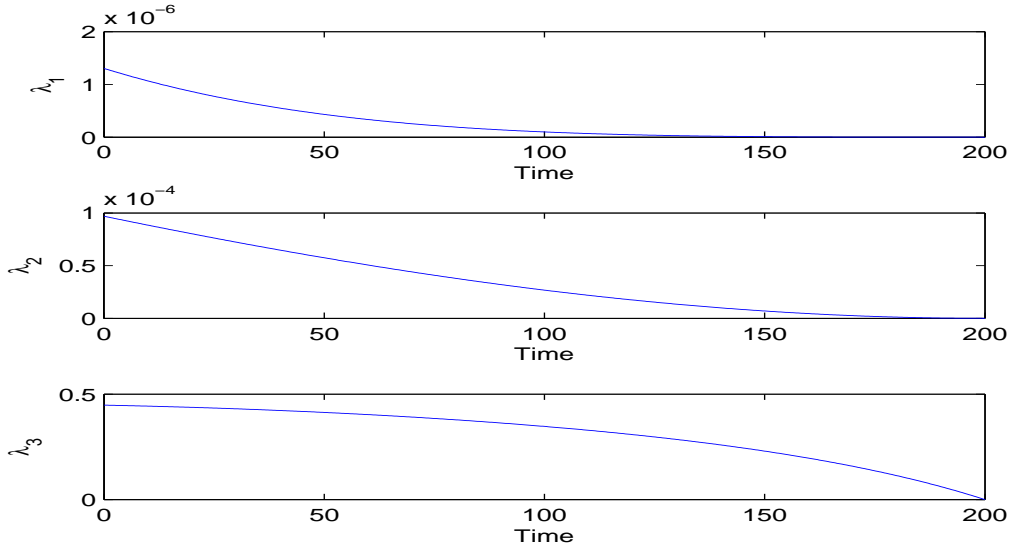
Figure 5.13: Graphs for the adjoint variables.

5.8   Conclusion

In this chapter, a three species such as prey, predator and super predator populations have been considered to analyze the interaction among them. It is assumed that the predator population consumes only prey population and super predator population consumes only predator population. Also, it is assumed that predator population shows refuge behavior when they are attacked by super predator. Thus higher refuge rate can make the crisis of food for super predator. So, in this model, it is considered that the additional food is to be supplied to the super predator and predator population also benefited from some portion of this additional food. It can reduce the risk of extinction of the super predator population and also increases the harvesting effort. Probably this is the first article where it is considered both refuge on the predator and additional food to the predator populations with harvesting and Beddington-DeAngelis type functional response. The boundedness of all solutions of the system has been done. It is observed that the boundedness of all solutions depends upon the supply of additional food. The extinction condition of the super predator population has been investigated theoretically. From the numerical simulations, it is observed that the change of consumption rate of predator to the super predator population i.e., a_2 makes the system unstable through Hopf bifurcation. But the constant rate of refuge by predator to the super predator (m_1) makes the system stable. So, the refuge by predator to the super predator acts a control of instability of the system. Again, for the higher value of the refuge makes the system unstable. But, if we supply some additional food to the super predator makes the system

stable. So, from this results we can conclude that the refuge (m_1) and constant supply of additional food (A) have a significant effects on predator prey dynamics. From the analysis, it is observed that neither prey nor super predator survive with the help of additional food only. Additional food is used here only as a supplementary food and to increase the growth of the predators. From the numerical simulations of optimal harvesting problem it can be concluded that as the amount of additional food (A) and selling price (p_1) of unit biomass of harvested super predator increases then the harvesting rate increases. But if the cost of harvesting (h) increases then the harvesting rate gradually decreases.

Gateway from Chapter 5 to Chapter 6

In Chapter 5, we have analyzed the effects of refuge and supply of additional food on the dynamics of predator-prey system and also on the Super predator harvesting. With the development of civilization, it is observed that different kinds of infectious diseases such as Cholera, Malaria, Japanese Encephalitis etc. are attacking human population at different times worldwide. So, in the next chapter we are interested to investigate the dynamics of Cholera disease in a periodic environment in the presence of bacteriophage.

Part III

Studies on Dynamics in Some Epidemiological Problems

Chapter 6

Dynamics of Cholera Outbreak with Bacteriophage and Periodic Rate of Contact

6.1   Introduction

The Cholera, a waterborne gastroenteric infection, remains a significant threat to public health in the developing countries. It is caused by the bacterium Vibrio Cholerae. It is typically transmitted through water and food that have been contaminated with fecal matter from a person who is infected with the disease. Now-a-days, several number of mathematical models have been developed for understanding the dynamics of Cholera disease. In 1973, Capasso [32] developed a mathematical model for Cholera epidemic occurred in the European Mediterranean region. In this model, Capasso used two differential equations to describe the dynamics of infected people in the infected region and the dynamics of the bacterium Vibrio Cholerae. Codeco [51] extended the model of Capasso by introducing one additional equation of susceptible human population and described the dynamics of the persistence of the disease Cholera. After that Codeco's model was extended by Hartley [90] including hyper-infectious vibrio bacterium. Again, Hartley's mathematical model was analyzed by Liao and Wang [132].

This disease is typically seasonal due to climatic, physical and many biological factors. It is observed that in Bangladesh, the seasonality of Cholera exhibits two peaks per year and differs from that of other diarrheal diseases [105]. This disease typically increases from November to January and April to May. In the same time, the seasonal Cholera disease is occurred in the peak form in April, May and June [65] in every year. There are

also many research papers [6, 97, 134, 174, 175, 259, 264] on the Cholera disease dynamics
to study its unknown behavior.

It is known that the bacteriophage might control the natural population of pathogens.
Moreover, the recent studies in marine microbiology also have revealed the elegant
balance between bacteriophage and their mycobacterial prey. In 2009, J.E. Nelson
et al. [165] explored that in a closed experimental system, the transmission of Vibrio
Cholerae may be minimized when these two factors such as bacteriophage and bac-
terium are combined in the aquatic environments. Therefore, the dynamical interaction
between bacteriophage and bacterium in pond water suggests that a model of Cholera
transmission should incorporate a measure for the rapid decay of bacterial culturability
and predation by bacteriophage.

There are two hundred species that infect V.Cholerae known as vibriophage. In 2006
a mathematical model was described by Jensen et al. [110] in the role of bacteriophage
in the control of Cholera outbreak. They suggested that either bacteria in the environ-
mental reservoir are hyper-infectious or most victims ingest bacteria amplified in food
or drinking water contaminated by environmental water carrying few viable V.Cholerae.
The consequent reduction of bacteria numbers in the effluent might fully account for
the decline in disease incidence and density of phage preying on these bacteria. In this
interpretation, the outbreak drives the changes in phage populations, rather than the
reverse. They showed that the large numbers of phage to the reservoir at the time of
the bacterial bloom decreases the size of the epidemic. If there are few number of phage
such as 10^5 virion per liter or less then there is virtually no effect on the epidemic. There
are some research papers [3, 33, 41] on bacteriophage dynamics. A Cholera epidemic
model of an optimal cost effectiveness study on Zimbabwe Cholera seasonal data from
$2008 - 2011$ was developed by Sardar et al. [206] in 2013. We have modified this model
by introducing the bacteriophage and discussed the dynamical behavior of the proposed
Cholera epidemic model.

Though there are many research articles available on the study of Cholera disease trans-
mission and its control strategies but till now there exists some space in the literature
which are as follows:

It is known that contaminated food and water by Vibrio Cholerae are the main rea-
son of spread of the infectious disease Cholera. From the experimental study, it is seen
that for a certain amount of time Vibrio Cholerae remains hyper-infectious and after
that time it becomes low-infectious. Also, experimentally proved that bacteriophage can
reduce the density of Vibrio Cholerae and prevent the burden of spread of Cholera dis-
ease in human population. Again, in the real world every parameters has been changed
with respect to time. But in mathematical model these concepts are not included till
now.

Considering the above phenomena a Cholera disease epidemic model has been developed in this chapter. Here, the dynamical behavior of the interaction between Cholera pathogen Vibrio Cholerae and bacteriophage has been discussed. Also, two types of transmission rates during hyper-infectious and low infectious period of bacterium Vibrio Cholerae have been considered as a periodic function of time. Then, the extinction and uniform persistence of the disease have been explored here. Also, the existence and stability of positive ω-periodic solution have been discussed precisely. Finally, a numerical simulations have been presented to support the analytic results of the proposed model.

6.2 Model Formulation [1]

In this chapter, it is considered that a human population in an area is effected with Vibrio Cholerae. Obviously, at time t, the population $N(t)$ consists of three kinds of populations such as (i) susceptible human $S(t)$, (ii) infected human $I(t)$ and (iii) recovered human $R(t)$ i.e., $N(t) = S(t) + I(t) + R(t)$. Here, the bacterial population at time t is also considered of two types such as: (i) hyper-infectious bacteria $B_H(t)$ and (ii) low-infectious bacteria $B_L(t)$. Also here, the population of bacteriophage $P(t)$ has been introduced.

Now, the susceptible population is increased (i) by a constant recruitment of newborn at a rate Λ_H and (ii) by those recovered persons who lose their temporary immunity to Cholera at a rate w. It is reduced by getting infected on contact with hyper-infectious and low-infectious bacterium at the rates $\beta_H(t)\frac{B_H}{K_H+B_H}$ and $\beta_L(t)\frac{B_L}{K_L+B_L}$ respectively and also decreased by natural death at a rate μ_d [206], where $\beta_H(t)$ and $\beta_L(t)$ have been defined in this chapter as follows:

$$
\begin{aligned}
\beta_H(t) &= \beta_{H0}(1 + \delta cos(\frac{2\pi t}{365})) \\
\beta_L(t) &= \beta_{L0}(1 + \delta cos(\frac{2\pi t}{365}))
\end{aligned}
$$

where δ is the amplitude of seasonality.

Here, infected human is increased by those susceptible humans who get infected in contact with hyper-infectious and low-infectious vibrios and decreased by (i) those who are recovered from Cholera at a rate γ, (ii) those who die due to Cholera infection at a rate μ_c and (iii) those who die naturally at a rate μ_d [206].

Again, recovered human population is increased by those infected people who get recovery from the disease at a rate γ. It is observed that the recovered population is reduced due to the loss of natural immunity to Cholera at a rate w and due to the natural dies at a rate μ_d [206].

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[1]Published in **International Journal of Dynamics and Control**, Springer, vol-4, 2016, 284-292, with title *Dynamics of cholera outbreak with bacteriophage and periodic rate of contact.*

Hyper-infectious bacterium is enriched from the amount of hyper-infectious V.Cholerae bacterium in the contaminated aquatic environment due to infected human feces, vomiting etc. at a rate ξ [51].

It is assumed that hyper-infectivity bacterium loses their hyper-infectivity at a rate χ. Here, it is also assumed that the both bacterium populations decrease by the consumption of phage (bacteriophage). The number of phage produced per infected bacterium (burst size) is denoted by β_1. The death rate of phage in the reservoir is w_1 per day [110]. Therefore, under the above considerations a mathematical model of Vibrio Cholerae has been suggested as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda_H + wR - \beta_H(t)\frac{B_H S}{K_H + B_H} - \beta_L(t)\frac{B_L S}{K_L + B_L} - \mu_d S \\[2mm]
\frac{dI}{dt} &= \beta_H(t)\frac{B_H S}{K_H + B_H} + \beta_L(t)\frac{B_L S}{K_L + B_L} - (\mu_d + \mu_c + \gamma)I \\[2mm]
\frac{dR}{dt} &= \gamma I - (w + \mu_d)R \\[2mm]
\frac{dB_H}{dt} &= \xi I - \chi B_H - \gamma_1 B_H P \\[2mm]
\frac{dB_L}{dt} &= \chi B_H - \delta_L B_L - \gamma_1 B_L P \\[2mm]
\frac{dP}{dt} &= \beta_1 \gamma_1 (B_H + B_L)P - w_1 P
\end{aligned}\right\} \tag{6.1}$$

The initial conditions are taken as $S(0) \geq 0, I(0) \geq 0, R(0) \geq 0, B_H(0) \geq 0,$ $B_L(0) \geq 0, P(0) \geq 0$.

6.3 Boundedness of Solutions

In this section, boundedness of all solutions of system (6.1) has been discussed.

Theorem 6.1 The solution of the proposed model (6.1), $(S(t), I(t), R(t), B_H(t), B_L(t))$ is uniformly and ultimately bounded i.e., there exists a positive real number M such that $(S(t), I(t), R(t), B_H(t), B_L(t)) \leq (M, M, M, M, M)$ for $t \geq T$ where T is a fixed time.

Proof. From the first three equations of proposed model (6.1), it is obtained that

$$\begin{aligned}
\frac{d(S + I + R)}{dt} &= \Lambda_H - \mu_d(S + I + R) - \mu_c I \\
&\leq \Lambda_H - \mu_d(S + I + R)
\end{aligned}$$

Hence, by standard comparison theorem [125], there exists $t_1 > 0$ such that $S + I + R \leq \frac{\Lambda_H}{\mu_d}$ for all $t \geq t_1$. Then, we have $S \leq \frac{\Lambda_H}{\mu_d}$, $I \leq \frac{\Lambda_H}{\mu_d}$ and $R \leq \frac{\Lambda_H}{\mu_d}$ for all $t \geq t_1$.

Again, from the fourth equation of the proposed model (6.1), it is obtained that

$$\begin{aligned}
\frac{dB_H}{dt} &\leq \xi \frac{\Lambda_H}{\mu_d} - \chi B_H - \gamma_1 B_H P \\
&\leq \xi \frac{\Lambda_H}{\mu_d} - \chi B_H
\end{aligned}$$

Hence, again by standard comparison theorem, there exists $t_2 \geq t_1$ such that $B_H(t) \leq \frac{\xi \Lambda_H}{\chi \mu_d}$ for all $t \geq t_2$.

From, the fifth equation of the proposed model (6.1), it is obtained that

$$\begin{aligned}
\frac{dB_L}{dt} &\leq \frac{\xi \Lambda_H}{\chi \mu_d} - \delta_L B_L - \gamma_1 B_L P \\
&\leq \frac{\xi \Lambda_H}{\chi \mu_d} - \delta_L B_L
\end{aligned}$$

Again, also by standard comparison theorem, there exists $t_3 \geq t_2 \geq t_1$ such that $B_L(t) \leq \frac{\xi \Lambda_H}{\mu_d \delta_L}$ for all $t \geq t_3$.

Let $M = max\{\frac{\Lambda_H}{\mu_d}, \frac{\xi \Lambda_H}{\chi \mu_d}, \frac{\xi \Lambda_H}{\delta_L \mu_d}\}$.

Thus, it follows that $S(t) \leq M$, $I(t) \leq M$, $R(t) \leq M$, $B_H(t) \leq M$, and $B_L(t) \leq M$ for all $t \geq T$. Therefore, all human populations, hyper-infectious and low-infectious Vibrio Cholerae are uniformly and ultimately bounded.

Again, from the sixth equation of the proposed model (6.1), it is obtained that

$$\frac{dP}{dt} \leq \beta_1 \gamma_1 (\frac{\xi \Lambda_H}{\chi \mu_d} + \frac{\xi \Lambda_H}{\mu_d \delta_L})P - w_1 P$$

Now, if $\beta_1 \gamma_1 \left(\frac{\xi \Lambda_H}{\chi \mu_d} + \frac{\xi \Lambda_H}{\mu_d \delta_L} \right) > w_1$ then the bacteriophage population becomes unbounded.

6.4 Local Stability of Disease Free Equilibrium Point

Let (R^n, R_+^n) be the standard ordered n- dimensional Euclidean space with a norm $||.||$. For $u, v \in R^n$, we denote $u \geq v$ if $u - v \in R_+^n$; $u > v$, if $u - v \in R^n$; $u \gg v$, if $u - v \in Int(R_+^n)$.

Let $A(t)$ be a continuous, cooperative, irreducible and ω periodic $n \times n$ matrix function. Then $\phi_A(t)$ be a fundamental solution matrix of

$$\frac{dx}{dt} = A(t)x \tag{6.2}$$

Let $\rho(\phi_A(\omega))$ be the spectral radius of $\phi_A(\omega)$ [259]. According to Perron-Frobenius theorem, $\rho(\phi_A(\omega))$ is the simple principal eigenvalue of $\phi_A(\omega)$ and it admits an eigenvector v^* which is grater than equal to zero vector.

Lemma 6.1 (Zhang and Zhao [259]) Let $s = \frac{1}{\omega}ln\rho(\phi_A(\omega))$, then there exists a positive ω periodic function $v(t)$ such that $e^{st}v(t)$ is a solution of (6.2).

In the following, we calculate the basic reproduction number of proposed system (6.1). It is easy to see that the proposed system (6.1) has exactly one disease free equilibrium point $E_0(S_0, 0, 0, 0, 0, 0)$ where $S_0 = \frac{\Lambda_H}{\mu_d}$.

Now, $F(t) = \begin{bmatrix} 0 & \frac{\beta_H(t)\Lambda_H}{K_H\mu_d} & \frac{\beta_L(t)\Lambda_H}{K_L\mu_d} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$ and $V(t) = \begin{bmatrix} (\gamma + \mu_c + \mu_d) & 0 & 0 \\ -\xi & \chi & 0 \\ 0 & -\chi & \delta_L \end{bmatrix}$

Let $Y(t, s)$ is an 3×3 matrix solution of the system

$$\frac{dY(t, s)}{dt} = -V(t)Y(t, s)$$

for any $t \leq s$, $Y(s, s) = I$ where I is an 3×3 identity matrix.
Let C_ω be the ordered banach space of all ω periodic functions from R to R^3 which is equipped with maximum norm $||.||_\infty$ and the positive cone $C_\omega^+ = \{\phi \in C_\omega : \phi(t) \geq 0,\ \text{for all t in } R\}$. Now, we consider the linear operator $L : C_\omega \longrightarrow C_\omega$ by

$$(L\phi)(t) = \int_0^{+\infty} Y(t, t-a)F(t-a)\phi(t-a)da \tag{6.3}$$

for any $t \in R$ and $\phi \in C_\omega$.
Finally, for the system (6.1) we define the basic reproduction number R_0 as the spectral radius of L i.e., $R_0 = \rho(L)$ which has been motivated by the concept of next generation method introduced in the article of [244] and [15, 59].

From the above discussion, the following theorem for the local asymptotically stability of disease free equilibrium $E_0(S_0, 0, 0, 0, 0, 0)$ has been obtained.

Theorem 6.2 (Wang and Zhao [244]) The following statements are valid
(i) $R_0 = 1$ if and only if $\rho(\phi_{F-V}(\omega)) = 1$
(ii) $R_0 > 1$ if and only if $\rho(\phi_{F-V}(\omega)) > 1$
(iii) $R_0 < 1$ if and only if $\rho(\phi_{F-V}(\omega)) < 1$
Thus, we can say that the disease free equilibrium $E_0(S_0, 0, 0, 0, 0, 0)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Observation: It is seen that the parameters γ_1 (the phage absorption rate) and β_1 (the number of phage produced per infected bacterium) are absent in the matrix $F(t)$ and $V(t)$. Hence, it can be concluded that the phage population does not have any

effect on the reduction of R_0. But the higher value of absorption rate (γ_1) may decrease the Vibrio Cholerae bacterium population and it may reduce the spread of the Cholera disease.

6.5 Global Stability of Disease Free Equilibrium Point

In this section, the global stability of disease free equilibrium point has been investigated.

Theorem 6.3 If $R_0 < 1$, then the disease free state is globally asymptotically stable.

Proof. From second, fourth and fifth equations of the system (6.1), we have

$$
\begin{aligned}
\frac{dI}{dt} &= \beta_H(t)\frac{B_H S}{K_H + B_H} + \beta_L(t)\frac{B_L S}{K_L + B_L} - (\gamma + \mu_d + mu_c)I \\
&\leq \beta_H(t)\frac{B_H}{K_H + B_H}\frac{\Lambda_H}{\mu_d} + \beta_L(t)\frac{B_L}{K_L + B_L}\frac{\Lambda_H}{\mu_d} - (\gamma + \mu_d + mu_c)I \\
&\leq \beta_H(t)\frac{\Lambda_H B_H}{K_H \mu_d} + \beta_L(t)\frac{\Lambda_H B_L}{\mu_d K_L}\frac{\Lambda_H}{\mu_d} - (\gamma + \mu_d + mu_c)I \\
\frac{dB_H}{dt} &\leq \xi I - \chi B_H \\
\frac{dB_L}{dt} &\leq \chi B_H - \delta_L B_L
\end{aligned}
$$

Then, for all $t \geq 0$, hence $0 \leq S(t) \leq \frac{\Lambda_H}{\mu_d}$, $B_H(t) \geq 0$, $B_L(t) \geq 0$ and $P(t) \geq 0$. Now, consider the following auxiliary system:

$$
\begin{aligned}
\frac{dI}{dt} &= \beta_H(t)\frac{\Lambda_H B_H}{K_H \mu_d} + \beta_L(t)\frac{\Lambda_H B_L}{\mu_d K_L}\frac{\Lambda_H}{\mu_d} - (\gamma + \mu_d + mu_c)I \\
\frac{dB_H}{dt} &= \xi I - \chi B_H \\
\frac{dB_L}{dt} &= \chi B_H - \delta_L B_L
\end{aligned}
$$

which can be written as

$$
\frac{dX}{dt} = (F(t) - V(t))X
$$

where $X = (I(t), B_H(t), B_L(t))^T$.
Then using above **Lemma 6.1**, there exists a positive ω-periodic function $\bar{X}(t)$ such that $X(t) = e^{st}\bar{X}(t)$ is a solution of the above system where $s = \frac{1}{\omega}ln\rho(\phi_{F-V}(\omega))$. Again, from **Theorem 6.2**, we know that for $R_0 < 1, \rho(\phi_{F-V}(\omega)) < 1$, so s must be a negative

constant. Therefore, when $t \to \infty$, we have $X(t) \to 0$.

$$i.e., \lim_{t \to \infty} I(t) = 0, \lim_{t \to \infty} B_H(t) = 0 \text{ and } \lim_{t \to \infty} B_L(t) = 0.$$

$$i.e., \lim_{t \to \infty} R(t) = 0, \lim_{t \to \infty} P(t) = 0 \text{ and } \lim_{t \to \infty} S(t) = \frac{\Lambda_H}{\mu_d}$$

Hence, the above implies that the disease free equilibrium point E_0 is globally asymptotically stable.

6.6 Uniform Persistence of the Disease

In this section, the uniform persistence of the disease has been investigated.

Theorem 6.4 If $R_0 > 1$, there exists a positive constant ϵ such that for all initial value $(S(0), I(0), R(0), B_H(0), B_L(0), P(0)) \in \{(S, I, R, B_H, B_L, P) \in R_+^6 : I > 0, B_H > 0, B_L > 0\}$, the solution of system (6.1) satisfies the following

$$\lim_{t \to \infty} \inf I(t) \geq \epsilon, \lim_{t \to \infty} \inf B_H(t) \geq \epsilon \text{ and } \lim_{t \to \infty} \inf B_L(t) \geq \epsilon.$$

i.e., for $R_0 > 1$, the disease in system (6.1) is uniformly persistent.

Proof. Let us consider the sets $X = R_+^6$, $X_0 = \{(S, I, R, B_H, B_L, P) \in R_+^6 : I > 0, B_H > 0, B_L > 0\}$ and $\partial X_0 = X \setminus X_0$.
Next, we define a Poincare map $P : R_+^6 \longrightarrow R_+^6$ satisfying $P(x^0) = u(\omega, x^0), \forall x^0 \in R_+^6$ where $u(t, x^0)$ be the unique solution of system (6.1) satisfying $u(0, x^0) = x^0$.
At first, we show that P is uniformly persistent with respect to $(X_0, \partial X_0)$. It is easy to see from the system (6.1) that X and X_0 are positively invariant. Moreover, ∂X_0 is relatively closed set in X. Now, from **Theorem 6.1**, it follows that the solutions of the system (6.1) are uniformly and ultimately bounded. Thus, the semi-flow P is point dissipative on R_+^6 and $P : R_+^6 \to R_+^6$ is compact by Theorem 3.4.8 in [262]. Then, it follows that P admits a global attractor which attracts every bounded set in R_+^6.

Now, we define another set M_∂ as

$$M\partial = \{(S(0), I(0), R(0), B_H(0), B_L(0), P(0)) \in \partial X_0 : P^m(S_0, I_0, R_0, B_{H0}, B_{L0}, P_0) \in \partial X_0,$$
$$\forall m \in N \cup 0\}.$$

Next, it is claimed that

$$M\partial = \{(S, 0, R, 0, 0, P) : S \geq 0, R \geq 0, P \geq 0\}.$$

In fact, it is obvious that

$$\{(S, 0, R, 0, 0, P) : S \geq 0, R \geq 0, P \geq 0\} \subseteq M\partial$$

For any $(S(0), I(0), R(0), B_H(0), B_L(0), P(0)) \in \partial X_0 / \{(S, 0, R, 0, 0, P) : S \geq 0,$
$R \geq 0, P \geq 0\}$, if $I(0) = 0, B_H(0) = 0, B_L(0) > 0$ it is clear that $S > 0$ and $B_L > 0$ for all
$t > 0$. Now, from the second equation of (6.1) we have $\dot{I}(0) = \beta_L(t) \frac{S(0)B_L(0)}{K_L + B_L(0)} > 0 \Rightarrow I(0) >$
0. Thus, from the fourth equation of (6.1) we have $\dot{B}_H(0) = \xi I(0) > 0$ else if $I(0) = 0$,
$B_L(0) = 0$ and $B_H > 0$. Then, similarly we can show that $\dot{I}(0) > 0$ and $B_H(0) > 0$
and similarly for other cases also. Therefore, if $(S(0), I(0), R(0), B_H(0), B_L(0), P(0)) \notin$
$\{(S, 0, R, 0, 0, P) : S \geq 0, R \geq 0, P \geq 0\}$ then $(S(t), I(t), R(t), B_H(t), B_L(t), P(t)) \notin \partial X_0$
for simultaneously small $t > 0$. This implies that $M_\partial \subseteq \{(S, 0, R, 0, 0, P) : S \geq 0, R \geq$
$0, P \geq 0\}$. Therefore, we have $M_\partial = \{(S, 0, R, 0, 0, P) : S \geq 0, R \geq 0, P \geq 0\}$. Clearly,
E_0 is one fixed point of P in M_∂. If $(S(t), I(t), R(t), B_H(t), B_L(t), P(t))$ is a solution of
system (6.1) initiating from M_∂, it then follows from (6.1) that $S(t) \to S_0$,
$I(t) \to 0, R(t) \to 0, B_H(t) \to 0, B_L(t) \to 0, P(t) \to 0$ as $t \to \infty$. So, any solution of (6.1)
initiating in M_∂ will remain into M_∂.

We will now show that $\{E_0\}$ is an acyclic covering of E_0. It is enough to show that
$\{E_0\}$ is a isolated invariant subset of M_∂ i.e., $W^s(E_0) \cap X_0 = \emptyset$, where $W^s(E_0)$ is the
stable set of E_0. Let $x^0 = (S(0), I(0), R(0), B_H(0), B_L(0), P(0)) \in X_0$, then by the conti-
nuity of solution with respect to initial values $\forall x \in (0, \frac{\Lambda_H}{\mu_d})$, then there exists $\xi > 0$ such
that $\forall x^0 \in X_0$ with $||x^0 - E_0|| \leq \xi$, it follows that $||u(t, x^0) - u(t, E_0)|| \leq \epsilon \forall t \in [0, w]$.
To show $x^0 \in X_0 \Rightarrow x^0 \notin W^s(E_0)$, it is enough to show that $\lim_{m \to \infty} d(P^m(x^0), E_0) \geq \xi$ for
some $m > 0$. If not let $\exists x^0 \in X_0$ such that $\lim_{m \to \infty} d(P^m(x^0), E_0) < \eta$ for all $m > 0$. This
implies that $||u(t, P^m(x^0)) - u(t, E_0)|| \leq \epsilon, \forall t \in [0, w]$.
To show, $x^0 \in X_0 \Rightarrow x^0 \notin W^s(E_0)$, it is enough to show that $\lim_{m \to \infty} \sup d(P^m(x^0), E_0) \geq$
ξ for some $m > 0$. If not let $\exists x^0 \in X_0$ such that $\lim_{m \to \infty} \sup d(P^m(x^0), E_0) \geq \eta$ for all
$m > 0$. This implies that $||u(t, P^m(x^0)) - u(t, E_0)|| < \epsilon, \forall t \in [0, w]$. For any $t \geq 0$, let
$t = mw + t_1$ where $t_1 \in [0, w]$ and $m = [\frac{t}{w}]$ which is the greatest integer less than or
equal to $\frac{t}{w}$. Then, we have $||u(t, P^m(x^0)) - u(t, E_0)|| = ||u(t_1, P^m(x^0)) - u(t, E_0)|| \leq \epsilon$,
$\forall t \in [0, w]$.

By selecting $(S(t), I(t), R(t), B_H(t), B_L(t), P(t)) = u(t, x^0)$, it follows that $\frac{\Lambda_H}{\mu_d} - \epsilon \leq$
$S(t) \leq \frac{\Lambda_H}{\mu_d} + \epsilon, 0 \leq I \leq \epsilon, 0 \leq R \leq \epsilon, 0 \leq B_H \leq \epsilon, 0 \leq B_L \leq \epsilon, 0 \leq P \leq \epsilon$ for all $t \geq 0$.
Then, we have $\frac{S(t)}{K_H + B_H} \geq (\frac{\Lambda_H}{\mu_d K_H} - \frac{\epsilon}{K_H + \epsilon})$ and $\frac{S(t)}{K_L + B_L} \geq (\frac{\Lambda_H}{\mu_d K_L} - \frac{\epsilon}{K_L + \epsilon})$. Therefore, from
system (6.1) we have

$$\frac{dI}{dt} \geq \beta_H(t)(\frac{\Lambda_H}{\mu_d K_H} - \frac{\epsilon}{K_H + \epsilon})B_H + \beta_L(t)(\frac{\Lambda_H}{\mu_d K_L} - \frac{\epsilon}{K_L + \epsilon})B_L$$
$$\frac{dB_H}{dt} = \xi I - \chi B_H$$
$$\frac{dB_L}{dt} = \chi B_H - \delta_L B_L$$

Then, a matrix $M_\epsilon(t)$ can be obtained as follows:

$$M_\epsilon(t) = \begin{bmatrix} 0 & \beta_H(t)\frac{\epsilon}{K_H+\epsilon} & \beta_L(t)\frac{\epsilon}{K_L+\epsilon} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Already, from **Theorem 6.2**, it is known that as $R_0 > 1$ so $\rho(\phi_{F-V}(\omega)) > 1$, choosing ϵ is very very small such that $\rho(\phi_{F-V-M_\epsilon}(\omega)) > 1$. Again, by **lemma 6.1** and the standard comparison principle [125], there exists a positive ω periodic function $f_2(t)$ such that $\jmath(t) \geq f_2(t)e^{s_2(t)}$ where $\jmath(t) = (I(t), B_H(t), B_L(t))^T$ and $s_2 = \frac{1}{\omega}ln\rho(\phi_{F-V}(\omega)) > 0$. This implies that

$$\lim_{t\to\infty} I(t) = \infty, \lim_{t\to\infty} B_H(t) = \infty \text{ and } \lim_{t\to\infty} B_L(t) = \infty$$

which is a contradiction in M_∂. Hence, $W^s(E_0) \cap X_0 = \emptyset$. Then, by Theorem 1.3.1 [262] we obtain that P is uniformly persistent with respect to $(X_0, \partial X_0)$. Thus, by Theorem 1.3.1 [262] it follows that the solution of (6.1) is uniformly persistent.

6.7 Periodic Solution

In this section, the existence and stability of a positive periodic solution of the system (6.1) have been investigated in **Theorem 6.5** as follows:

Theorem 6.5 If $R_0 > 1$, then the system (6.1) admits a positive ω-periodic solution which is globally asymptotically stable.

Proof. We have already proved in Theorem 6.3 that the Poincare map, $P : R_+^6 \longrightarrow R_+^6$ of the system (6.1) is point dissipative and compact as well as P is uniformly persistent with respect to $(X_0, \partial X_0)$. Then, it follows from Theorem 1.3.6 [262] that the Poincare map P has a fixed point $(\tilde{S}, \tilde{I}, \tilde{R}, \tilde{B}_H, \tilde{B}_L, \tilde{P}) \in Int(R_+^6)$. Hence, $u(t, (\tilde{S}, \tilde{I}, \tilde{R}, \tilde{B}_H, \tilde{B}_L, \tilde{P})) \in Int(R_+^6)$ for all $t > 0$. Thus, $(\tilde{S}, \tilde{I}, \tilde{R}, \tilde{B}_H, \tilde{B}_L, \tilde{P})$ is a positive $\omega-$ periodic solution of system (6.1) due to the definition of the semi-flow P.

Let $\tilde{X} = (\tilde{S}, \tilde{I}, \tilde{R}, \tilde{B}_H, \tilde{B}_L, \tilde{P})$ be positive ω-periodic solution of system (6.1) and $X(t) = (S(t), I(t), R(t), B_H(t), B_L(t), P(t))$ be any solution of system (6.1) initiating from nonnegative initial values. Then, a Lyapunov function is defined as follows:

$$L(S, I, R, B_H, B_L, P) = |S(t) - \tilde{S}(t)| + |I(t) - \tilde{I}(t)| + |R(t) - \tilde{R}(t)|$$
$$+ \frac{\mu_d}{\xi}|B_H(t) - \tilde{B}_H(t)| + \frac{\mu_d}{\xi}|B_L(t) - \tilde{B}_L(t)| + \frac{1}{\beta_1}|P(t) - \tilde{P}(t)|$$

Using the following

$$|x|' = \begin{cases} x' & if\ x \geq 0 \\ -x' & if\ x < 0 \end{cases}$$

the right upper derivative $D^+L(t)$ of $L(t)$ of the system (6.1) is obtained as follows:

$$
\begin{aligned}
D^+L(S, I, R, B_H, B_L, P) = sign(S(t) - \tilde{S}(t)) &\left\{ \beta_H(t) \frac{\tilde{B}_H(t)\tilde{S}(t)}{K_H + \tilde{B}_H(t)} + \beta_L(t) \frac{\tilde{B}_L(t)\tilde{S}(t)}{K_L + \tilde{B}_L(t)} \right. \\
-\beta_H(t) \frac{B_H(t)S(t)}{K_H + B_H(t)} - \beta_L(t) \frac{B_L(t)S(t)}{K_L + B_L(t)} &\left. + w(R(t) - \tilde{R}(t)) - \mu_d(S(t) - \tilde{S}(t)) \right\} \\
+ sign(I(t) - \tilde{I}(t)) &\left\{ \beta_H(t) \frac{B_H(t)S(t)}{K_H + B_H(t)} + \beta_L(t) \frac{B_L(t)S(t)}{K_L + B_L(t)} - \beta_H(t) \frac{\tilde{B}_H(t)\tilde{S}(t)}{K_H + \tilde{B}_H(t)} \right. \\
-\beta_L(t) \frac{\tilde{B}_L(t)\tilde{S}(t)}{K_L + \tilde{B}_L(t)} &\left. - (\gamma + \mu_c + \mu_d)(I(t) - \tilde{I}(t)) \right\} + sign(R(t) - \tilde{R}(t)) \left\{ \gamma(I(t) - \tilde{I}(t)) \right. \\
-(w + \mu_d)(R(t) - \tilde{R}(t)) &\left. \right\} + \frac{\mu_d}{\xi} sign(B_H(t) - \tilde{B}_H(t)) \left\{ \xi(I(t) - \tilde{I}(t)) \right. \\
-\chi(B_H(t) - \tilde{B}_H(t)) &\left. - \gamma_1(B_H(t)P(t) - \tilde{B}_H(t)\tilde{P}(t)) \right\} \\
+\frac{\mu_d}{\xi} sign(B_L(t) - \tilde{B}_L(t)) &\left\{ \chi(B_H(t) - \tilde{B}_H(t)) - \delta_L(B_L(t) - \tilde{B}_L(t)) \right. \\
-\gamma_1(B_L(t)P(t) - \tilde{B}_L(t)\tilde{P}(t)) &\left. \right\} + \frac{1}{\beta_1} sign(P(t) - \tilde{P}(t)) \left\{ \beta_1\gamma_1(B_H(t)P(t) - \tilde{B}_H(t)\tilde{P}(t)) \right. \\
+\beta_1\gamma_1(B_L(t)P(t) - \tilde{B}_L(t)\tilde{P}(t)) &\left. - w_1(P(t) - \tilde{P}(t)) \right\} \\
\leq -\mu_d|S(t) - \tilde{S}(t)| - \mu_c|I(t) - \tilde{I}(t)| &- \mu_d|R - \tilde{R}| - \frac{\mu_d\delta_L}{\xi}|B_L(t) - \tilde{B}_L(t)| \\
-\frac{w_1}{\beta_1}|P(t) - \tilde{P}(t)| & \\
\leq -K \bigg(|S(t) - \tilde{S}(t)| + |I(t) - \tilde{I}(t)| &+ |R - \tilde{R}| + |B_L(t) - \tilde{B}_L(t)| + |P(t) - \tilde{P}(t)| \bigg)
\end{aligned}
$$

where $K = min\{\mu_d, \mu_c, \frac{\mu_d\delta_L}{\xi}, \frac{w_1}{\beta_1}\}$.

Now, integrating the above inequality from $\bar{t}$ to ∞, it is obtained that

$$
\begin{aligned}
L(t) + K \int_{\bar{t}}^{\infty} \bigg(|S(t) - \tilde{S}(t)| + |I(t) - \tilde{I}(t)| &+ |R - \tilde{R}| + |B_L(t) - \tilde{B}_L(t)| \\
+ |P(t) - \tilde{P}(t)| \bigg) ds \leq L(\bar{t}) &
\end{aligned}
$$

provided that $t > \bar{t}$. Then, it follows that

$$\sup_{t \to \infty} K \int_{\bar{t}}^{\infty} \bigg(|S(t) - \tilde{S}(t)| + |I(t) - \tilde{I}(t)| + |R - \tilde{R}| + |B_L(t) - \tilde{B}_L(t)|$$

$$+ |P(t) - \tilde{P}(t)| \bigg) ds \leq \frac{L(\bar{t})}{K} < +\infty$$

i.e., $\quad \lim_{t \to \infty} |S(t) - \tilde{S}(t)| = 0, \lim_{t \to \infty} |I(t) - \tilde{I}(t)| = 0, \lim_{t \to \infty} |R(t) - \tilde{R}(t)| = 0,$

$$\lim_{t \to \infty} |B_H(t) - \tilde{B}_H(t)| = 0, \lim_{t \to \infty} |B_L(t) - \tilde{B}_L(t)| = 0 \text{ and } \lim_{t \to \infty} |P(t) - \tilde{P}(t)| = 0.$$

Therefore, the solution $(\tilde{S}(t), \tilde{I}(t), \tilde{R}(t), \tilde{B}_H(t), \tilde{B}_L(t), \tilde{P}(t))$ of system (6.1) is globally asymptotically stable.

6.8 Numerical Simulation

For numerical simulation to illustrate the proposed mathematical model, the standard software MATLAB 2010a has been used. After finding the value of R_0 numerically using the parametric values in Table 6.1, it has been shown that when disease persists and disappears from the human population.

Table 6.1: (Estimation of parameters)

Parameters	Values	Unit	References
Λ_H	15	day^{-1}	Assumed
μ_d	5.48×10^{-5}	day^{-1}	[90]
μ_c	0.015	day^{-1}	[90]
γ	0.004	day^{-1}	[90]
ξ	100	$cells/Lday^{-1}$	[51]
χ	33.6	$cells/Lday^{-1}$	[206]
γ_1	1.4×10^{-9}	liter per virion/day	[41]
δ_L	0.2333	day^{-1}	[51]
β_1	100	virion per cell	[33]
w	0.025	day^{-1}	Assumed
w_1	$0.5 - 7.9$	virion per day	[110]

Now, using the following initial values of the state variables such as $S(0) = 800000, I(0) = 800, R(0) = 100, B_H(0) = 3000000, B_L(0) = 3000000, P(0) = 300000$ and also taking $K_H = 10^9, K_L = 10^9, \delta = 0.75, B_{H0} = 0.2143, B_{L0} = 0.2$, Figure 6.1 has been drawn from which, it is observed that when $R_0 = 0.6043 < 1$, then it is confirmed that the disease free equilibrium point of the system (6.1) is globally asymptotically stable. Therefore, it is concluded that the Cholera disease will be disappeared from the human population when the basic reproduction number must be less than one in the presence of bacteriophage.

Again, using the following initial values of the state variables such as $S(0) = 8000, I(0) = 800, R(0) = 100, B_H(0) = 30000, B_L(0) = 30000, P(0) = 3000$ and also $K_H = 10^9, K_L = \frac{10^9}{7}, \delta = 0.75, B_{H0} = 0.2143, B_{L0} = 0.2$, Figure 6.2 has been drawn from which it is observed that when $R_0 = 4.2030 > 1$, then it is confirmed that the disease persists in the system (6.1) and it is globally asymptotically stable. Therefore, it is concluded that the Cholera disease will be positively persisted in the human population when the basic reproduction number is greater than one in the presence of bacteriophage.
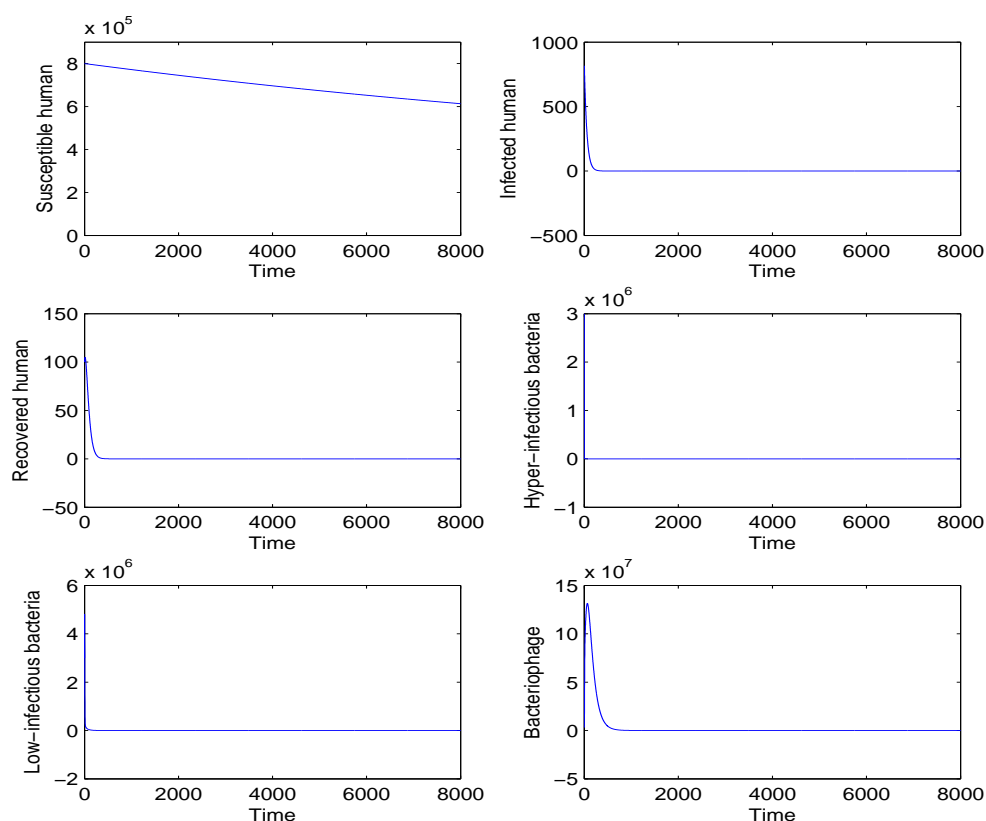

Figure 6.1: Represents the solution of the system (6.1) when $R_0 < 1$.
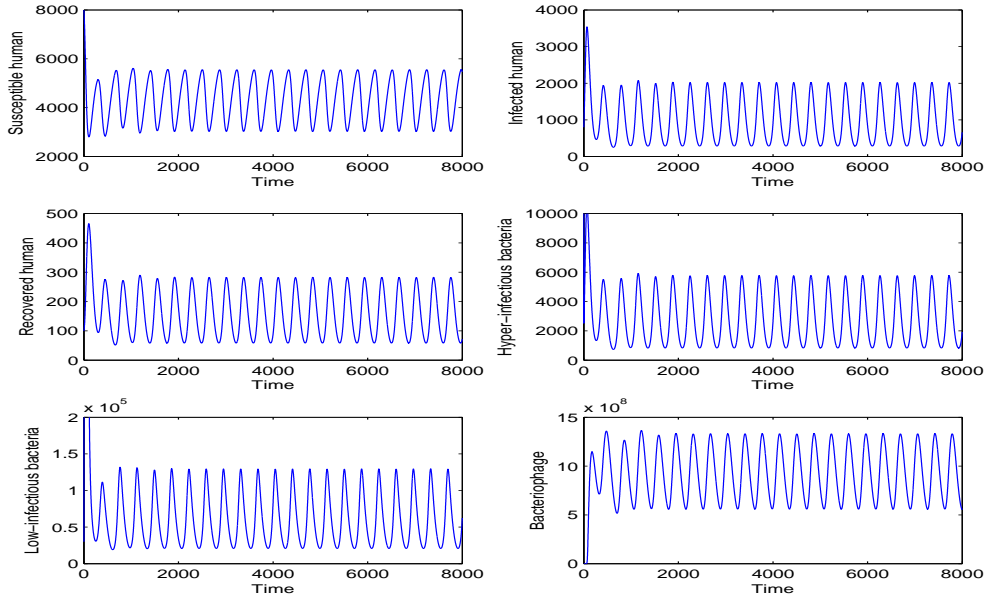
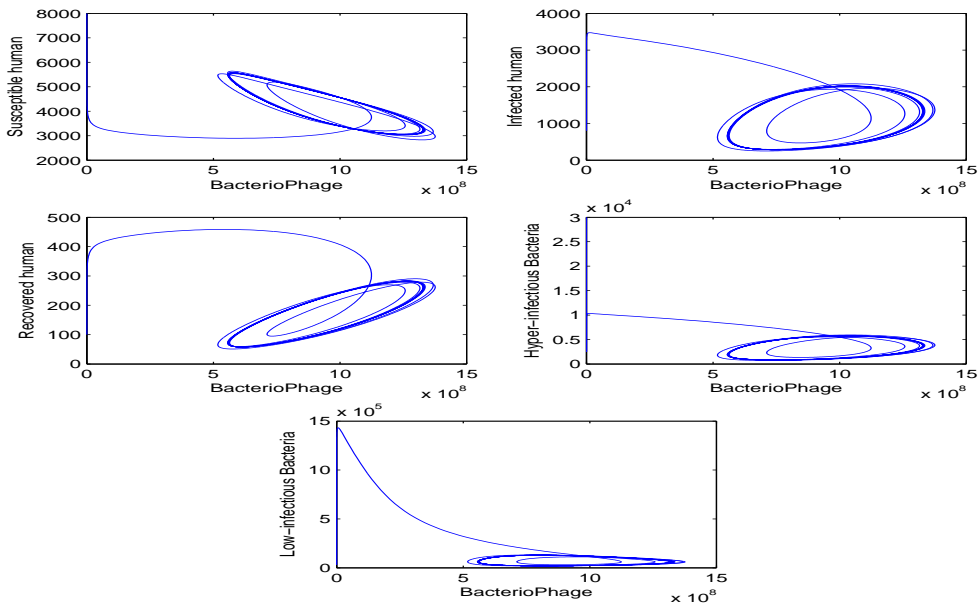Figure 6.2: Represents the solution of the system (6.1) when $R_0 > 1$.

Figure 6.3: Limit cycle.

Using the same parametric values and initial condition that is used in Figure 6.2, Figure 6.3 has been drawn and from this figure, it is seen that the limit cycle of each population such as susceptible human, infected human, recovered human, hyper-infectious Vibrio Cholerae bacteria and low-infectious Vibrio Cholerae bacteria with respect to bacteriophage is stable. Again, it is known that the stable limit cycles are example of attractors. So, they imply self-sustained oscillations i.e., the closed trajectory describes perfect periodic behavior of the system and any small perturbation from this closed trajectory causes the system to return to it, making the system stick to the limit cycle.

6.9 Conclusion

In this chapter, a Cholera epidemic model with periodic transmission rate has been discussed. Here, the total human population is divided into three sub populations such as (*i*) susceptible human (*ii*) infected human (*iii*) recovered human as well as total bacterial population to be divided into three sub populations such as (*i*) hyper-infectious Vibrio Cholerae bacterium (*ii*) low-infectious bacterium and (*iii*) bacteriophage. The host immunity, pathogen hyper-infectivity and phages are all factors that can be leveraged for outbreak control. Here, it has been shown that the disease free equilibrium point is globally asymptotically stable and the Cholera disease is disappeared if the basic reproduction number is less than one. It is observed that when the basic reproduction number is grater than one, then the endemic equilibrium is globally asymptotically stable and the disease persists in the human population. It is also observed that the system has a stable limit cycle with respect to bacteriophage and this closed trajectory describes perfect periodic behavior of the proposed system. Here, numerical simulations of the mathematical model support our all analytical results.

Gateway from Chapter 6 to Chapter 7
In Chapter 6, we have studied the dynamics of Cholera disease in a time periodic environment. Most of the parameters involved in this chapter are crisp. Also, most of the published papers on mathematical models of Cholera disease are crisp in nature. But in real world, every parameter changes with respect to time due to different human activities or natural disasters. So, the parameters related to the Cholera disease transmission may be uncertain in nature. Taking this issue in mind, we study the dynamics of Cholera disease model in a fuzzy environment in the next chapter.

Chapter 7

Dynamical Study in Fuzzy Threshold Dynamics of a Cholera Epidemic Model

7.1 Introduction

Cholera is an acute diarrhoeal infection caused by ingestion of food or water contaminated with the bacterium Vibrio Cholerae. It causes mortality, disability, social and economic damage for millions of people in the whole world specially in developing countries. So, it is a major threat to human being. Two of the toxigenic Vibrio Cholerae $O1$ and $O139$ are free-living bacterial organisms found in fresh and briny water. Frequently, they are found in association with zooplankton, shellfish and aquatic plants. It spreads through the contaminated food, drinking water and also from the feces of infected human. The main symptoms of Cholera are watery diarrhea, vomiting, rapid dehydration, rapid heart rate, loss of skin elasticity, dry mucous membranes etc. Severe outbreaks usually occur in underdeveloped areas with inadequate sanitation, poor hygiene and limited access to safe water supplies. Although there are many recent progresses in medical sciences, Cholera remains now as a global threat in some parts of the world.

Mathematical models have become more important tools for analyzing the spread of Cholera disease. Basically, ordinary differential equation is used for formulation of this type of problem and it provides some mathematical answer and explanation. At first a crisp mathematical model on Cholera disease was described by Capasso [32] in 1979. It was consisted with two equations to follow the dynamics of infected individuals and the number of free-living infective stages. More recently, Codeco [51] developed a more

general model of Cholera with an additional equation included in the Capasso's model. Modeling and analysis of the spread of carrier dependent infectious diseases with environmental effects was explored by Singh et al. [218]. There exists many mathematical models [7, 9, 90, 99, 153, 154, 174–176] in crisp environment on Cholera disease which explore the spread and control strategies of the disease.

The parameters involved in the mathematical models on Cholera disease discussed above are crisp in nature. But it is found that the biological parameters involved in the differential equations are not always fixed. In the real world, it is seen that every community is changing continuously with the varying environments. In the present time, the global warming is the main problem in the whole globe. It is the increase of earth's average surface temperature due to effect of greenhouse gases such as carbon dioxide emissions from burning fossil fuels or from deforestation which trap heat that would otherwise escape from earth. The change of temperature strongly effects on the reproduction rate of the bacterial population. Many parameters may oscillate with the change of environments in real world ecosystem. These parameters are also varying due to both natural and human activities such as earthquake, climate warming, financial crisis etc. Therefore, due to continuous interactions between the human and bacteria, the dynamics of Cholera disease are strongly influenced by the environmental variations. Therefore, the parameters in the systems are uncertain in nature. In the literature there are different methods to deal the uncertainty. One of them fuzzy set theoretic approach is more significant to analyze the uncertain parameters. At first the fuzzy concept was introduced by Zadeh [258] in 1965. After that many developments [60–62, 265] is going on the fuzzy numbers. So, in this chapter, we have used fuzzy set theory to formulate this Cholera model. There exist very few number of papers in infectious disease model in a fuzzy environment [57]. Mizukoshi et al. [155] explored the stability analysis of dynamical system with variables and parameters in uncertain environment. Then, Peixto et al. [179] and Pal et al. [172] developed fuzzy parameter based predator-prey mathematical model.

From the above literature reviews, it is observed that many investigation have been done on the Cholera disease transmission and its control in crisp environment. But till now no one has investigated the Cholera model in fuzzy environment.

To explore this unveiled direction in the Cholera disease here it has been developed by considering all parameters to be fuzzy numbers. Here, total human population is classified into three subpopulations such as susceptible human, infected human and recovered human. Also, one bacterial population (Vibrio Cholerae) is considered in this mathematical model. Existence condition and boundedness of solution of our proposed mathematical model have been discussed. Also, the different equilibrium points and the stability condition of the system around these equilibrium points have been analyzed. The global stability conditions of the proposed system around the positive equilibrium point have been also discussed. Ultimately, some numerical simulations have been given to verify our analytical findings.

7.2 Preliminaries

Fuzzy Set
Fuzzy sets deals with objects that are 'matter of degree' with all possible grades of truth between yes or no. So a fuzzy set is a class of objects in which there is no sharp boundary between those objects that belong to the class and those do not. Let X be a collection of objects and x be an element of X then a fuzzy set $\tilde{A}$ in X is a set of ordered pairs $\tilde{A} = \{(x, \mu_{\tilde{A}}(x)) : x \in X\}$ where $\mu_{\tilde{A}}(x)$ is called the membership function or grade of membership of x in $\tilde{A}$ which maps X to the membership space M which is considered as the closed interval $[0, u]$ where $0 \leq u \leq 1$.

Triangular Fuzzy Number
A Triangular fuzzy number $\tilde{A}$ is specified by the triplet (a, b, c) and is defined by its continuous membership (Figure 7.1) function $\mu_{\tilde{A}}(x) : X \to [0, 1]$ as follows

$$\mu_{\tilde{A}}(x) = \begin{cases} \dfrac{x - a}{b - a} & if\ a \leq x \leq b \\ \dfrac{c - x}{c - b} & if\ b \leq x \leq c \\ 0 & \text{otherwise} \end{cases}$$
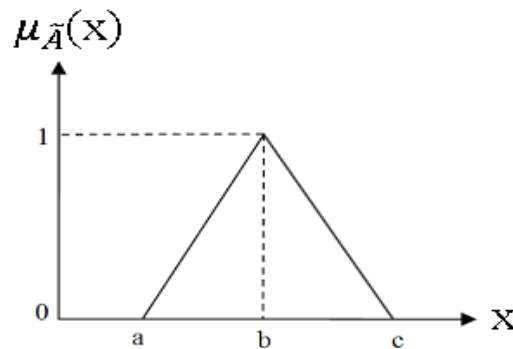
Figure 7.1: Triangular fuzzy number.

α - Cut of a Fuzzy Number
A α - cut of a fuzzy number $\tilde{A}$ in X is denoted by A_α and is defined as the following crisp set

$$A_\alpha = \{x : \mu_{\tilde{A}}(x) \geq \alpha, x \in X\} \ \text{ where } \alpha \in [0, 1]$$

A_α is a non-empty bounded closed interval contained in X and it can be denoted by $A_\alpha = [A_L(\alpha), A_R(\alpha)]$ where $A_L(\alpha)$ and $A_R(\alpha)$ are the lower and upper bounds (Figure 7.2) of the closed interval respectively.

It is clear that α - cut of triangular fuzzy number $\tilde{A} = (a_1, a_2, a_3)$ is a closed and bounded interval $[A_L(\alpha), A_R(\alpha)]$ where $A_L(\alpha) = a_1 + \alpha(a_2 - a_1)$ and $A_R(\alpha) = a_3 - \alpha(a_3 - a_2)$.

Figure 7.2: α - cut of a Triangular fuzzy number.

Interval Arithmetic of a Fuzzy Number

Let $[P_L, P_R]$ and $[Q_L, Q_R]$ be two interval numbers. The addition and subtraction of two interval numbers are given by
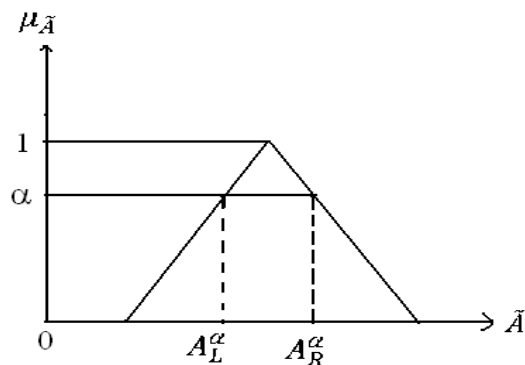
$$[P_L, P_R] + [Q_L, Q_R] = [P_L + Q_L, P_R + Q_R]$$
$$[P_L, P_R] - [Q_L, Q_R] = [P_L - Q_R, P_R - Q_L]$$

Utility Function Method (UFM)

In UFM, a utility function is defined for each of the objectives g_i according to their relative importance. A simple utility function may be defined as $w_i g_i$ for $i - th$ objective where w_i is a scalar and represent the weight assigned to the corresponding objective. Then the total utility defined as the weighted sum of the objectives as follows

$$U = \sum_{i=1}^{n} w_i g_i, w_i \geq 0$$

subject to the condition $\sum_{i=1}^{n} w_i = 1$.

7.3 Model Formulation

We have considered $S(t)$, $I(t)$, $R(t)$ and $V_E(t)$ as the population densities of the susceptible human, infected human, recovered human and Vibrio Cholerae in the environment at time t respectively. Let $\tilde{A}$, $\tilde{\mu}_d$, $\tilde{\beta}$ and $\tilde{\delta}$ be the fuzzy intrinsic growth rate of susceptible human, fuzzy natural death rate of susceptible human, fuzzy transmission rate of susceptible human to infected human and fuzzy rate of loose of natural immunity respectively. Let $\tilde{\mu}_d$, $\tilde{m}$, $\tilde{\alpha_1}$ and $\tilde{\gamma}$ be the fuzzy natural death rate of infected human, fuzzy

disease related death rate of infected human, fuzzy recovery rate of infected human and fuzzy rate of excretion of Vibrio Cholerae in the environment by vomiting, feces etc. by infected human. Let $\widetilde{\mu}_d$ be the fuzzy natural death rate of recovered human and fuzzy natural death rate of Vibrio Cholerae in the environment. By using the concept on the fuzzy initial value problem [210] and differentials of fuzzy functions [186] and considering the above assumptions, a set of fuzzy differential equations regarding the Cholera disease has been developed as follows:

$$
\left.\begin{aligned}
\frac{\widetilde{dS}}{dt} &= \widetilde{A} - \widetilde{\mu}_d S - \widetilde{\beta} S V_E + \widetilde{\delta} R \\[2mm]
\frac{\widetilde{dI}}{dt} &= \widetilde{\beta} S V_E - \widetilde{\mu}_d I - \widetilde{m} I - \widetilde{\alpha_1} I - \widetilde{\gamma} I \\[2mm]
\frac{\widetilde{dR}}{dt} &= \widetilde{\alpha_1} I - \widetilde{\mu}_d R - \widetilde{\delta} R \\[2mm]
\frac{\widetilde{dV_E}}{dt} &= \widetilde{\gamma} I - \widetilde{\mu_{V_E}} V_E
\end{aligned}\right\} \tag{7.1}
$$

To find the solution of (7.1) let

$$
\left[\frac{\widetilde{dx}}{dt}\right]_\alpha = \left[\left(\frac{dx}{dt}\right)_L^\alpha, \left(\frac{dx}{dt}\right)_R^\alpha\right]
$$

The deterministic system of the model (7.1) is given by

$$
\left(\frac{dS}{dt}\right)_L^\alpha = (A_L)^\alpha - (\mu_{d_R})^\alpha S - (\beta_R)^\alpha S V_E + (\delta_L)^\alpha R
$$
$$
\left(\frac{dS}{dt}\right)_R^\alpha = (A_R)^\alpha - (\mu_{d_L})^\alpha S - (\beta_L)^\alpha S V_E + (\delta_R)^\alpha R
$$
$$
\left(\frac{dI}{dt}\right)_L^\alpha = (\beta_L)^\alpha S V_E - [(\mu_{d_R})^\alpha + (m_R)^\alpha + (\alpha_{1_R})^\alpha + (\gamma_R)^\alpha] I
$$
$$
\left(\frac{dI}{dt}\right)_R^\alpha = (\beta_R)^\alpha S V_E - [(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha] I
$$
$$
\left(\frac{dR}{dt}\right)_L^\alpha = (\alpha_{1_L})^\alpha I - [(\mu_{d_R})^\alpha + (\delta_R)^\alpha] R
$$
$$
\left(\frac{dR}{dt}\right)_R^\alpha = (\alpha_{1_R})^\alpha I - [(\mu_{d_L})^\alpha + (\delta_L)^\alpha] R
$$
$$
\left(\frac{dV_E}{dt}\right)_L^\alpha = (\gamma_L)^\alpha I - \left(\mu_{V_{E_R}}\right)^\alpha V_E
$$
$$
\left(\frac{dV_E}{dt}\right)_R^\alpha = (\gamma_R)^\alpha I - \left(\mu_{V_{E_L}}\right)^\alpha V_E
$$

Using the concept of UFM, we can write the above system of differential equations as follows:

$$
\left.
\begin{aligned}
\frac{dS}{dt} &= w_1 \left(\tfrac{dS}{dt}\right)_L^\alpha + w_2 \left(\tfrac{dS}{dt}\right)_R^\alpha \\[2mm]
\frac{dI}{dt} &= w_1 \left(\tfrac{dI}{dt}\right)_L^\alpha + w_2 \left(\tfrac{dI}{dt}\right)_R^\alpha \\[2mm]
\frac{dR}{dt} &= w_1 \left(\tfrac{dR}{dt}\right)_L^\alpha + w_2 \left(\tfrac{dR}{dt}\right)_R^\alpha \\[2mm]
\frac{dV_E}{dt} &= w_1 \left(\tfrac{dV_E}{dt}\right)_L^\alpha + w_2 \left(\tfrac{dV_E}{dt}\right)_R^\alpha
\end{aligned}
\right\}
\tag{7.2}
$$

where w_1 and w_2 are two weight functions such that $w_1 + w_2 = 1$ and $w_1, w_2 \geq 0$. Then the equation (7.2) can be written as

$$
\left.
\begin{aligned}
\frac{dS}{dt} &= a_{11} - a_{12}S - a_{13}SV_E + a_{14}R \\[2mm]
\frac{dI}{dt} &= a_{21}SV_E - a_{22}I \\[2mm]
\frac{dR}{dt} &= a_{31}I - a_{32}R \\[2mm]
\frac{dV_E}{dt} &= a_{41}I - a_{42}V_E
\end{aligned}
\right\}
\tag{7.3}
$$

where

$a_{11} = w_1 \left(A_L\right)^\alpha + w_2 \left(A_R\right)^\alpha, a_{12} = w_1 \left(\mu_{d_R}\right)^\alpha + w_2 \left(\mu_{d_L}\right)^\alpha, a_{13} = w_1 \left(\beta_R\right)^\alpha + w_2 \left(\beta_L\right)^\alpha,$
$a_{14} = w_1 \left(\delta_L\right)^\alpha + w_2 \left(\delta_R\right)^\alpha, a_{21} = w_1 \left(\beta_L\right)^\alpha + w_2 \left(\beta_R\right)^\alpha$
$a_{22} = w_1 \left[\left(\mu_{d_R}\right)^\alpha + \left(m_R\right)^\alpha + \left(\alpha_{1_R}\right)^\alpha + \left(\gamma_R\right)^\alpha\right] + w_2 \left[\left(\mu_{d_L}\right)^\alpha + \left(m_L\right)^\alpha + \left(\alpha_{1_L}\right)^\alpha + \left(\gamma_L\right)^\alpha\right]$
$a_{31} = w_1 \left(\alpha_{1_L}\right)^\alpha + w_2 \left(\alpha_{1_R}\right)^\alpha, a_{32} = w_1 \left[\left(\mu_{d_R}\right)^\alpha + \left(\delta_R\right)^\alpha\right] + w_2 \left[\left(\mu_{d_L}\right)^\alpha + \left(\delta_L\right)^\alpha\right]$
$a_{41} = w_1 \left(\gamma_L\right)^\alpha + w_2 \left(\gamma_R\right)^\alpha, a_{42} = w_1 \left(\mu_{V_{E_R}}\right)^\alpha + w_2 \left(\mu_{V_{E_L}}\right)^\alpha$

7.4   Boundedness of Solutions

In this section, the boundedness of all solutions of the proposed system (7.3) has been shown. Before proving the boundedness at first **Lemma 7.1** has been proved.

Lemma 7.1 $(a_{22} - a_{31}) > 0, (a_{32} - a_{14}) > 0$ and $(a_{21} - a_{13}) > 0$ provided that $\left[\left(\mu_{d_L}\right)^\alpha + \left(m_L\right)^\alpha + \left(\gamma_L\right)^\alpha\right] + \left(\alpha_{1_L}\right)^\alpha > \left(\alpha_{1_R}\right)^\alpha$, $\left(\mu_{d_L}\right)^\alpha + \left(\delta_L\right)^\alpha > \left(\delta_R\right)^\alpha$ and $0 \leq w_1 \leq 0.5$ respectively.

Proof. We have

$$a_{22} - a_{31} = w_1 \left[(\mu_{d_R})^\alpha + (m_R)^\alpha + (\alpha_{1_R})^\alpha + (\gamma_R)^\alpha \right]$$

$$+ w_2 \left[(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha \right]$$

$$= w_1 \left\{ \left[(\mu_{d_R})^\alpha + (m_R)^\alpha + (\alpha_{1_R})^\alpha + (\gamma_R)^\alpha \right] - \left[(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha \right] \right.$$

$$\left. + (\alpha_{1_R})^\alpha - (\alpha_{1_L})^\alpha \right\} + \left[(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha \right] - (\alpha_{1_R})^\alpha \quad (7.4)$$

since $w_1 + w_2 = 1$.
Now, when $w_1 = 0$ we have

$$a_{22} - a_{31} = [(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha] - (\alpha_{1_R})^\alpha$$

Then $a_{22} - a_{31}$ will be positive for $w_1 = 0$ if

$$[(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha] > (\alpha_{1_R})^\alpha \quad (7.5)$$

Therefore, from equation (7.4) it is obtained that

$$\frac{d(a_{22} - a_{31})}{dw_1} = [(\mu_{d_R})^\alpha + (m_R)^\alpha + (\alpha_{1_R})^\alpha + (\gamma_R)^\alpha] - [(\mu_{d_L})^\alpha + (m_L)^\alpha + (\alpha_{1_L})^\alpha + (\gamma_L)^\alpha]$$
$$+ (\alpha_{1_R})^\alpha - (\alpha_{1_L})^\alpha > 0, \forall w_1 \in [0, 1]$$

So, $(a_{22} - a_{31})$ is an increasing function with respect to w_1 and it will be positive if condition (7.5) holds.
In the similar way, it can be proved that $(a_{32} - a_{14})$ is an increasing function with respect to w_1 and it will be positive if condition

$$(\mu_{d_L})^\alpha + (\delta_L)^\alpha > (\delta_R)^\alpha$$

holds and also $(a_{21} - a_{13})$ is an increasing function with respect to w_1 and it will be positive if condition $0 \leq w_1 \leq 0.5$ holds.

Theorem 7.1 All solutions of the system (7.3) are bounded in the region R_+^4 provided that $a_{21} \geq a_{13}$ and $\sigma = min\{a_{12}, (a_{22} - a_{31}), (a_{32} - a_{14})\}$.

Proof. Let us define a function

$$W = S + I + R \quad (7.6)$$

Now, differentiating (7.6) with respect to time t and simplifying we have

$$\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$i.e., \frac{dW}{dt} = a_{11} - a_{12}S + (a_{31} - a_{22})I + (a_{14} - a_{32})R + (a_{21} - a_{13})SV_E \quad (7.7)$$

For a positive real number σ, multiplying σ on the both sides of (7.6) and then adding with equation (7.7) we have

$$
\begin{aligned}
\frac{dW}{dt} + \sigma W \;=\;& a_{11} + (\sigma - a_{12})S + (\sigma - (a_{22} - a_{31}))I + (\sigma - (a_{32} - a_{14}))R \\
&+ (a_{21} - a_{13})SV_E
\end{aligned}
$$

Now according to **Lemma 7.1**, if $\sigma = min\{a_{12}, (a_{22} - a_{31}), (a_{32} - a_{14})\}$ then the above equation reduces to in the following form

$$
\frac{dW}{dt} + \sigma W \leq a_{11}
$$

Solving the above we get

$$
W \leq \frac{a_{11}}{\sigma} + c_1 e^{-\sigma t}
$$

Taking t tends to infinity, we have $W \leq \frac{a_{11}}{\sigma}$. So, it can be written as $S(t) \leq \frac{a_{11}}{\sigma}, I(t) \leq \frac{a_{11}}{\sigma}$ and $R(t) \leq \frac{a_{11}}{\sigma}$.

Now, from the fourth equation of system (7.3) we have

$$
\frac{dV_E}{dt} = a_{41}I - a_{42}V_E
$$
$$
\frac{dV_E}{dt} \leq \frac{a_{11}a_{41}}{\sigma} - a_{42}V_E
$$
$$
\frac{dV_E}{dt} + a_{42}V_E \leq \frac{a_{11}a_{41}}{\sigma}
$$

Solving the above equation, we have

$$
V_E \leq \frac{a_{11}a_{41}}{\sigma a_{42}} + c_2 e^{-a_{42}t}
$$

Taking t tends to infinity, it is obtained that

$$
V_E \leq \frac{a_{11}a_{41}}{\sigma a_{42}}
$$

This proves that the solutions of the system are bounded.

7.5   Equilibrium Points

The system (7.3) has two equilibrium points such as
(i) disease free equilibrium point $E_0=(S_0, 0, 0, 0)$ where $S_0 = \frac{a_{11}}{a_{12}}$.

(ii) endemic equilibrium point $(E^*) = (S^*, I^*, R^*, V_E^*)$ where

$$S^* = \frac{a_{22}a_{42}}{a_{21}a_{41}}$$

$$I^* = \frac{a_{11}a_{21}a_{41}a_{32} - a_{22}a_{32}a_{12}a_{42}}{a_{22}a_{32}a_{13}a_{41} - a_{21}a_{41}a_{14}a_{31}}$$

$$R^* = \frac{a_{31}I^*}{a_{32}}, V_E^* = \frac{a_{41}I^*}{a_{42}}$$

The basic reproduction number is $R_0 = \frac{a_{11}a_{21}a_{41}}{a_{22}a_{12}a_{42}}$.

7.6 Stability Analysis

The jacobian matrix of the system (7.3) is given by

$$J(S, I, R, V_E) = \begin{pmatrix} -a_{12} - a_{13}V_E & 0 & a_{14} & -a_{13}S \\ a_{21}V_E & -a_{22} & 0 & a_{21}S \\ 0 & a_{31} & -a_{32} & 0 \\ 0 & a_{41} & 0 & -a_{42} \end{pmatrix}$$

Theorem 7.2 The system (7.3) is locally asymptotically stable at E_0 if $\frac{a_{11}a_{21}a_{41}}{a_{22}a_{12}a_{42}} < 1$.

Proof. The characteristic equation at E_0 of the system (7.3) is given by

$$(a_{32} + x)(x + a_{12})\left[x^2 + b_1 x + b_2\right] = 0$$

where $b_1 = (a_{22} + a_{42})$, $b_2 = a_{22}a_{42} - \frac{a_{11}a_{21}a_{41}}{a_{12}}$.
The roots of the characteristic equation are $x = -a_{32} < 0$, $x = -a_{12} < 0$ since a_{12}, a_{32} are positive and also by Routh-Hurwitz criteria the roots of the quadratic equation will be negative real number or complex with negative real parts if $b_1 > 0, b_2 > 0$ and $b_1^2 - 4b_2 < 0$. Since $a_{22} > 0, a_{42} > 0$ then obviously $b_1 > 0$ and b_2 will be positive if

$$a_{22}a_{12}a_{42} - a_{11}a_{21}a_{41} > 0$$
$$\text{i.e., } \frac{a_{11}a_{21}a_{41}}{a_{22}a_{12}a_{42}} < 1$$

Theorem 7.3 The system (7.3) is locally asymptotically stable at E^* if $c_i > 0$ for $i = 1, 2, 3, 4$ and $c_1c_2 > c_3, c_1c_2c_3 > c_3^2 + c_1^2c_4$.

Proof. The characteristic equation of the system (7.3) at the equilibrium point E^* is

$$x^4 + c_1 x^3 + c_2 x^2 + c_3 x + c_4 = 0$$

where $c_1 = a_{32} + a_{42} + a_{12} + a_{13}V_E^* + a_{22}$, $c_2 = a_{32}a_{42} + a_{22}a_{12} + a_{22}a_{13}V_E^* - a_{21}a_{41}S^*$,
$c_3 = a_{32}a_{42}a_{12} + a_{32}a_{42}a_{13}V_E^* + a_{22}a_{32}a_{42} + a_{22}a_{32}a_{12} + a_{22}a_{32}a_{13}V_E^* + a_{22}a_{42}a_{12} + a_{22}a_{42}a_{13}V_E^*$

$-a_{14}a_{21}a_{31}V_E^* - a_{12}a_{21}a_{41}S^* - a_{21}a_{41}a_{32}S^*$, $c_4 = a_{22}a_{32}a_{42}a_{12} + a_{22}a_{32}a_{42}a_{13}V_E^*$ $-a_{14}a_{21}a_{31}a_{42}V_E^* - a_{12}a_{21}a_{41}a_{32}S^*$. Now, according to Routh-Hurwitch criteria the roots of a biquadratic equation will be negative or have a negative real parts i.e., the system (7.3) will be locally asymptotically stable at E^* if each $c_i > 0$ for $i = 1, 2, 3, 4$ and $c_1c_2 > c_3, c_1c_2c_3 > c_3^2 + c_1^2c_4$.

Theorem 7.4 The system (7.3) is globally asymptotically stable around E^* if $\mu_3 > 0$ where $\mu_3 = a_{21}\mu_2 - a_{22} + min[(a_{12} + a_{22} + a_{13}\mu_2) - max(a_{21}\mu_2, a_{13}\mu_2), a_{42} - a_{41} + min\{a_{21} + \mu_2(a_{13} - a_{21}), a_{22}\}]$.

Proof. Let us consider the subsystem of the system (7.3) as

$$
\left.
\begin{array}{rcl}
\dfrac{dS}{dt} & = & a_{11} - a_{12}S - a_{13}SV_E \\[2mm]
\frac{dI}{dt} & = & a_{21}SV_E - a_{22}I \\[2mm]
\frac{dV_E}{dt} & = & a_{41}I - a_{42}V_E
\end{array}
\right\}
\tag{7.8}
$$

The jacobian matrix of the system (7.8) is given by

$$
J = \begin{pmatrix} -a_{12} - a_{13}V_E & 0 & -a_{13}S \\ a_{21}V_E & -a_{22} & a_{21}S \\ 0 & a_{41} & -a_{42} \end{pmatrix}
$$

Now, the second additive matrix is given by

$$
J^{[2]} = \begin{pmatrix} -a_{12} - a_{22} - a_{13}V_E & a_{21}S & a_{13}S \\ a_{41} & -a_{12} - a_{13}V_E - a_{42} & 0 \\ 0 & a_{21}V_E & -a_{22} - a_{42} \end{pmatrix}
$$

We have $P(x) = P(S, I, V_E) = diag\left(\frac{S}{I}, \frac{S}{I}, \frac{S}{I}\right)$, $P_f = \frac{\partial P}{\partial x} = diag\left(\frac{\dot{S}}{I} - \frac{S\dot{I}}{I^2}, \frac{\dot{S}}{I} - \frac{S\dot{I}}{I^2}, \frac{\dot{S}}{I} - \frac{S\dot{I}}{I^2}\right)$. Now, it follows that $P_fP^{-1} = diag\left(\frac{\dot{S}}{S} - \frac{\dot{I}}{I}, \frac{\dot{S}}{S} - \frac{\dot{I}}{I}, \frac{\dot{S}}{S} - \frac{\dot{I}}{I}\right)$ and $PJ^{[2]}P^{-1} = J^{[2]}$ so that

$B = P_fP^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$

$B_{11} = \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - (a_{12} + a_{22} + a_{13}V_E)$, $B_{12} = \begin{pmatrix} a_{21}S & a_{13}S \end{pmatrix}$, $B_{21} = \begin{pmatrix} a_{41} & 0 \end{pmatrix}^t$

$B_{22} = \begin{pmatrix} \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - (a_{12} + a_{13}V_E + a_{42}) & 0 \\ a_{21}V_E & \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - (a_{22} + a_{42}) \end{pmatrix}$

Now, $\mu_1(B_{11}) = \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - (a_{12} + a_{13}V_E + a_{42})$, $|B_{12}| = max\{a_{21}S, a_{13}S\}$, $B_{21} = a_{41}$, $\mu_1(B_{22}) = \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - a_{42} - min\{a_{21} + V_E(a_{13} - a_{21}), a_{22}\}$.

Therefore,

$$
\mu(B) \leq sup\{p_1, p_2\}
\tag{7.9}
$$

where

$$p_1 = \mu_1(B_{11}) + |B_{12}| = \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - (a_{12} + a_{22} + a_{13}V_E) + max\{a_{21}S, a_{13}S\},$$

$$p_2 = \mu_1(B_{22}) + |B_{21}| = \frac{\dot{S}}{S} - \frac{\dot{I}}{I} - a_{42} + a_{41} - min\{a_{21} + V_E(a_{13} - a_{21}), a_{22}\}$$

We have

$$\dot{I} = a_{21}SV_E - a_{22}I$$

$$\frac{\dot{I}}{I} = \frac{a_{21}SV_E}{I} - a_{22}$$

If there exists $t_1 > 0$ such that $\inf\{S(t), I(t), R(t), V_E(t)\} = \mu_2$ i.e., if $R_0 > 1$ then from the above we have

$$p_1 = \frac{\dot{S}}{S} - a_{21}\mu_2 + a_{22} - (a_{12} + a_{22} + a_{13}\mu_2) + max\{a_{21}\mu_2, a_{13}\mu_2\},$$

$$p_2 = \frac{\dot{S}}{S} - a_{21}\mu_2 + a_{22} - a_{42} + a_{41} - min\{a_{21} + \mu_2(a_{13} - a_{21}), a_{22}\}$$

Then from the equation (7.9) it is obtained that

$$\mu(B) \leq \frac{\dot{S}}{S} - a_{21}\mu_2 + a_{22} - min[(a_{12} + a_{22} + a_{13}\mu_2) - max\,(a_{21}\mu_2, a_{13}\mu_2),$$

$$a_{42} - a_{41} + min\,\{a_{21} + \mu_2(a_{13} - a_{21}), a_{22}\}]$$

$$i.e., \quad \mu(B) \leq \frac{\dot{S}}{S} - \mu_3 \tag{7.10}$$

where

$$\mu_3 \quad = \quad a_{21}\mu_2 - a_{22} + min[(a_{12} + a_{22} + a_{13}\mu_2) - max\,(a_{21}\mu_2, a_{13}\mu_2), a_{42} - a_{41}$$
$$+ \quad min\,\{a_{21} + \mu_2(a_{13} - a_{21}), a_{22}\}].$$

Integrating the above equation (7.10) from 0 to t we have

$$\int_0^t \mu(B)ds \leq log\frac{S(t)}{S(0)} - \mu_3 t$$

$$\frac{1}{t}\int_0^t \mu(B)ds \leq \frac{1}{t}log\frac{S(t)}{S(0)} - \mu_3$$

$$\lim_{t\to\infty} supsup\frac{1}{t}\int_0^t \mu(B)ds < -\mu_3 < 0, \text{ if } \mu_3 > 0.$$

This proves that the positive equilibrium (S^*, I^*, V_E^*) is globally asymptotically stable if $\mu_3 > 0$.

Next, we consider the third equation of system (7.3) as

$$\frac{dR}{dt} = a_{31}I - a_{32}R$$

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and its limiting form is

$$\frac{dR}{dt} = a_{31}I^* - a_{32}R$$

Then, we have $R(t) \to \frac{a_{31}I^*}{a_{32}} = R^*$ as $t \to \infty$. Now, if $\mu_3 > 0$ then, the interior equilibrium point $E^*(S^*, I^*, R^*, V_E^*)$ will be globally asymptotically stable.

7.7 Numerical Simulation

To study the feasibility of the fuzzy model about the Cholera disease, all biological parameters are hypothesized in imprecise nature which are considered here triangular fuzzy number. To discuss the dynamical behavior of Cholera numerically following problems have been considered.

Problem 1: In this problem the following hypothetical data of all parameter involved in the model are considered as: $\tilde{A} = (50, 60, 70), \tilde{\beta} = (0.001, 0.0011, 0.0012), \tilde{\mu_d} = (0.2, 0.3, 0.4), \tilde{\delta} = (0.001, 0.002, 0.003), \tilde{m} = (0.005, 0.006, 0.007), \tilde{\alpha_1} = (0.2, 0.3, 0.4), \tilde{\gamma} = (0.02, 0.021, 0.022), \tilde{\mu_{V_E}} = (1/50, 1/40, 1/30)$.
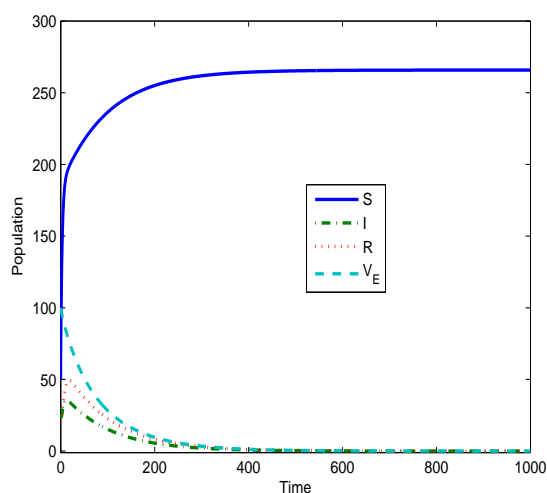

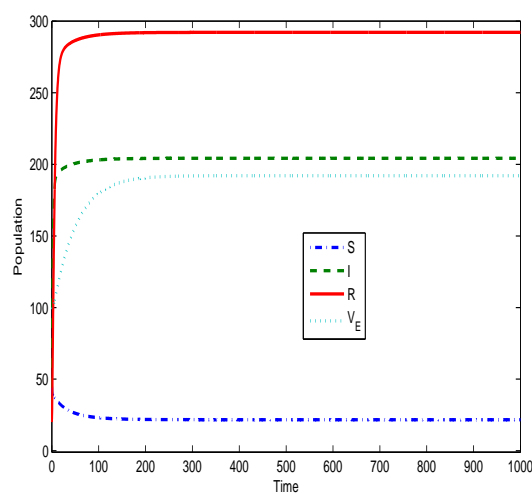Figure 7.3: Local stability of disease free equilibrium point.

Figure 7.4: Local stability of endemic equilibrium point.

For the above set of parametric values with $\alpha = 0.1, w_1 = 0.2, w_2 = 0.8$ the Figure 7.3 has been drawn. From this figure, it is observed that the system is free of disease. Here, the disease free equilibrium point is $E_0(265.8537, 0, 0, 0)$. From **Theorem 7.2** it is also seen that $\frac{a_{11}a_{21}a_{41}}{a_{22}a_{12}a_{42}} = 0.5572 < 1$ so the system (7.3) is locally asymptotically stable around the equilibrium point $E_0(265.8537, 0, 0, 0)$.

Problem 2: In this problem, we consider the same set of parametric values in **Problem 1** except $\beta = (0.01, 0.02, 0.03)$.

Using this data Figure 7.4 has been drawn, from which this figure it is observed that the system is endemic and the endemic equilibrium point is given by

$E^* = (21.68, 204.30, 292.20, 192.10)$. From **Theorem 7.3**, it is also seen that $c_1 = 3.8404 > 0$, $c_2 = 1.5746 > 0$, $c_3 = 0.4374 > 0$, $c_4 = 0.0081 > 0$, $c_1 c_2 - c_3 = 5.6096 > 0$ and $c_1 c_2 c_3 - (c_3)^2 - (c_1)^2 c_4 = 2.3334 > 0$, so the system (7.3) is locally asymptotically stable around the equilibrium point $E^*(21.68, 204.30, 292.20, 192.10)$.
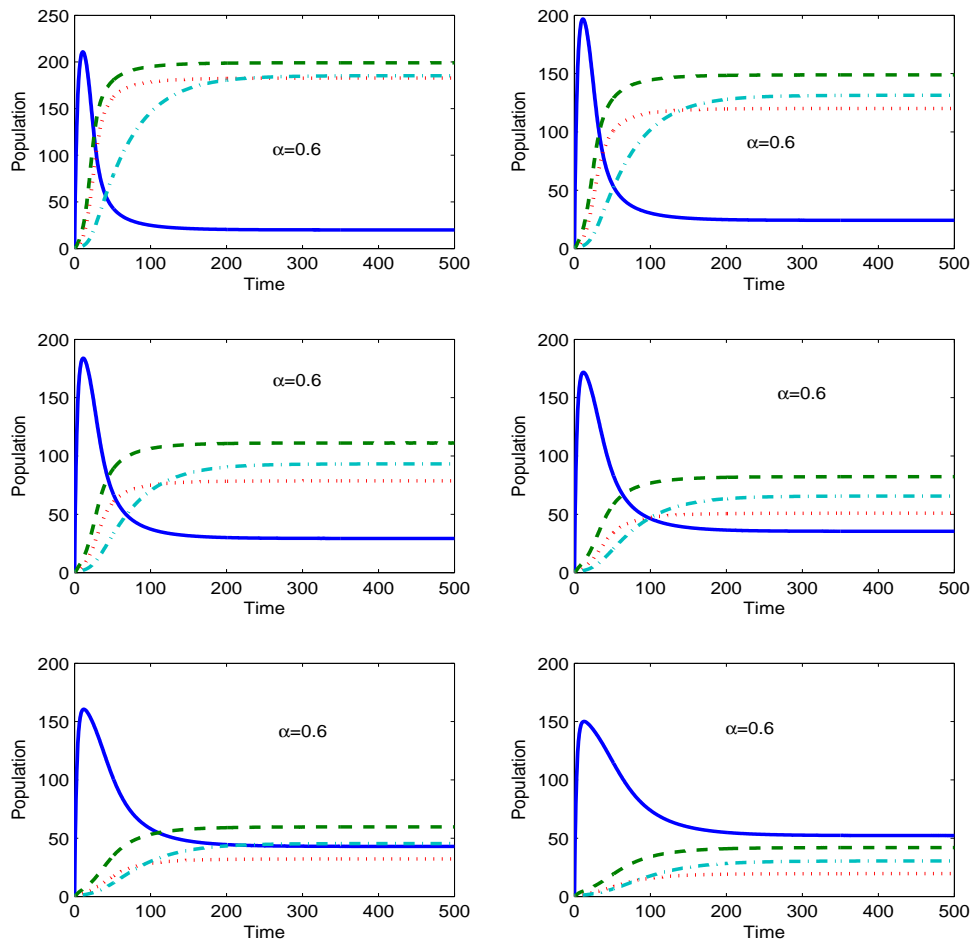

Figure 7.5: Solid line represents susceptible human, dash line represents infected human, dotted line represents recovered human and dash-dot line represents Vibrio Cholerae in the environment.

Problem 3: In this case following data set has been considered as: $\tilde{A} = (50, 60, 70), \tilde{\beta} = (0.01, 0.02, 0.03), \tilde{\mu_d} = (0.2, 0.3, 0.4), \tilde{\delta} = (0.001, 0.002, 0.003), \tilde{m} = (0.005, 0.006, 0.007), \tilde{\alpha_1} = (0.1, 0.2, 0.3), \tilde{\gamma} = (0.02, 0.021, 0.022), \mu_{\tilde{V_E}} = (1/50, 1/40, 1/30)$. For this data set considering different weight w_1, w_2 different equilibrium points have been computed for $\alpha = 0.0, 0.6, 1.0$ in **Table 7.1**.

Table 7.1: Equilibrium points for different α and w_1, w_2.

w_1	w_2	$\alpha = 0.0$	$\alpha = 0.6$	$\alpha = 1.0$
0.0	1.0	(9.85, 655.00, 977.70, 719.90)	(20.00, 199.20, 185.30, 185.8)	(31.39, 96.24, 63.73, 80.83)
0.2	0.8	(16.41, 287.70, 309.90, 273.40)	(24.22, 148.90, 120.10, 131.40)	(31.39, 96.24, 63.73, 80.83)
0.4	0.6	(26.47, 137.70, 107.50, 115.20)	(29.28, 111.20, 78.65, 93.21)	(31.39, 96.24, 63.73, 80.83)
0.6	0.4	(42.47, 64.15, 35.83, 47.60)	(35.42, 82.25, 50.91, 65.64)	(31.39, 96.24, 63.73, 80.83)
0.8	0.2	(69.70, 24.03, 9.28, 15.96)	(45.45, 59.73, 32.23, 45.46)	(31.39, 96.24, 63.73, 80.83)
1.0	0.0	(119.50, 0.99, 0.2483, 0.608)	(52.28, 41.96, 19.61, 30.51)	(31.39, 96.24, 63.73, 80.83)

Again, from Table 7.1, it is seen that for fixed α as the weight w_1 increases and w_2 decreases then the equilibrium levels of susceptible human gradually increases and the equilibrium level of infected human, recovered human and Vibrio Cholerae in the environment gradually decrease. It is also seen that when $\alpha = 1$ and for different combinations of w_1 and w_2 the equilibrium levels of all the populations remain same. These happen because for $\alpha = 1$ then the left and right intervals of triangular fuzzy number coincide with each other.

For this data set Figure 7.5 has been drawn for $\alpha = 0.6$. From this figure, as the weight w_1 increases and w_2 decreases then the equilibrium level of susceptible human gradually increases and the other three populations such as infected human, recovered human and Vibrio Cholerae in the environment gradually decrease.

Also, for the same set of parametric values used in **Problem 3** considering fixed weight values such as $w_1 = 0.4, w_2 = 0.6$ and for different values of α the Figure 7.6 has been drawn. From this figure, it is seen that as the value of α increases then all the human and bacterial populations gradually decrease. Now, from the above discussion, it is concluded that the interaction between human and bacterial population depends on the imprecise nature of the biological parameters.

Now, for the same set of parametric values used in **Problem 3** taking different values of α, w_1, w_2 Figure 7.7 has been drawn. In this figure, infected human and recovered human be plotted with the change of Vibrio Cholerae in the environment. From this figure, it is seen that the intersecting points of infected human and recovered human is influenced by the imprecise value of the parameters. This supports that the imprecise-

ness of parameters included in our proposed model.

Using the above parametric values used in **Problem 3**, Figure 7.8 has been drawn from which it is seen that the endemic equilibrium value will be changed with the change of α. So, it is concluded that the endemic equilibrium point is also influenced by the imprecise values of the parameters.
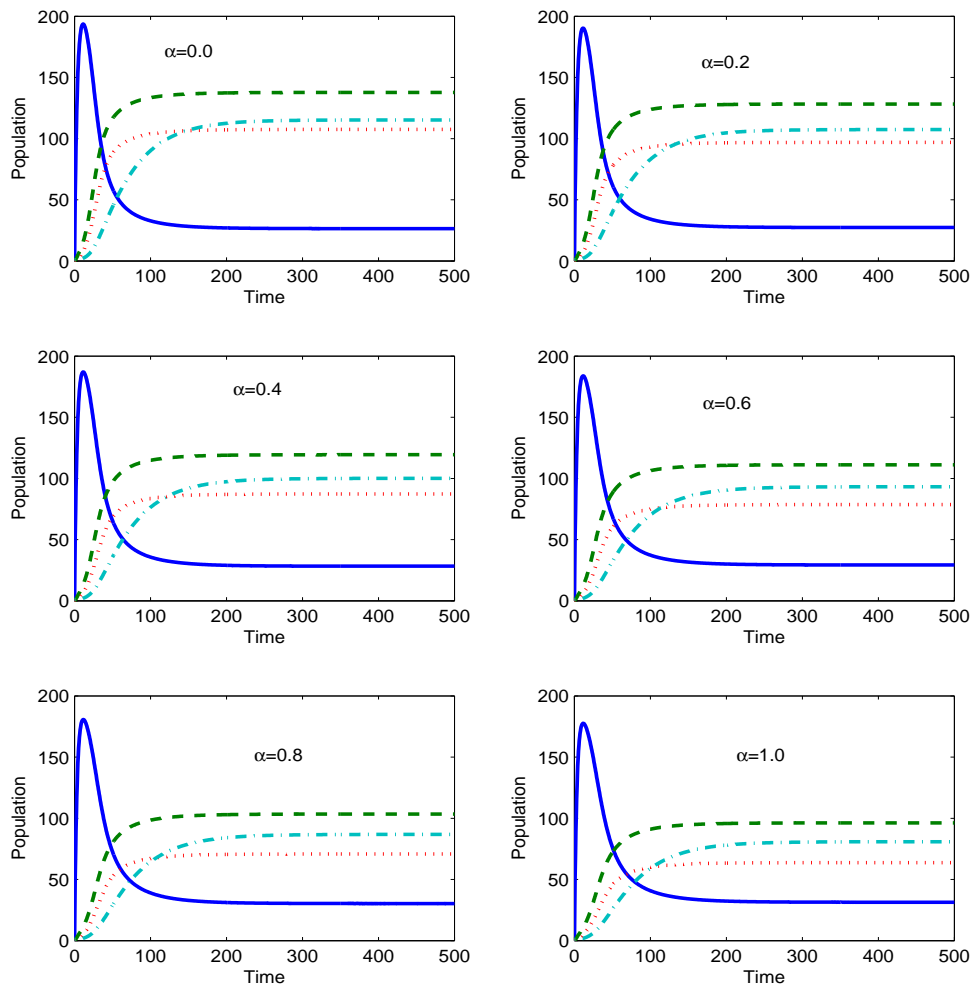

Figure 7.6: Solid line represents susceptible human, dash line represents infected human, dotted line represents recovered human and dash-dot line represents Vibrio Cholerae in the environment.
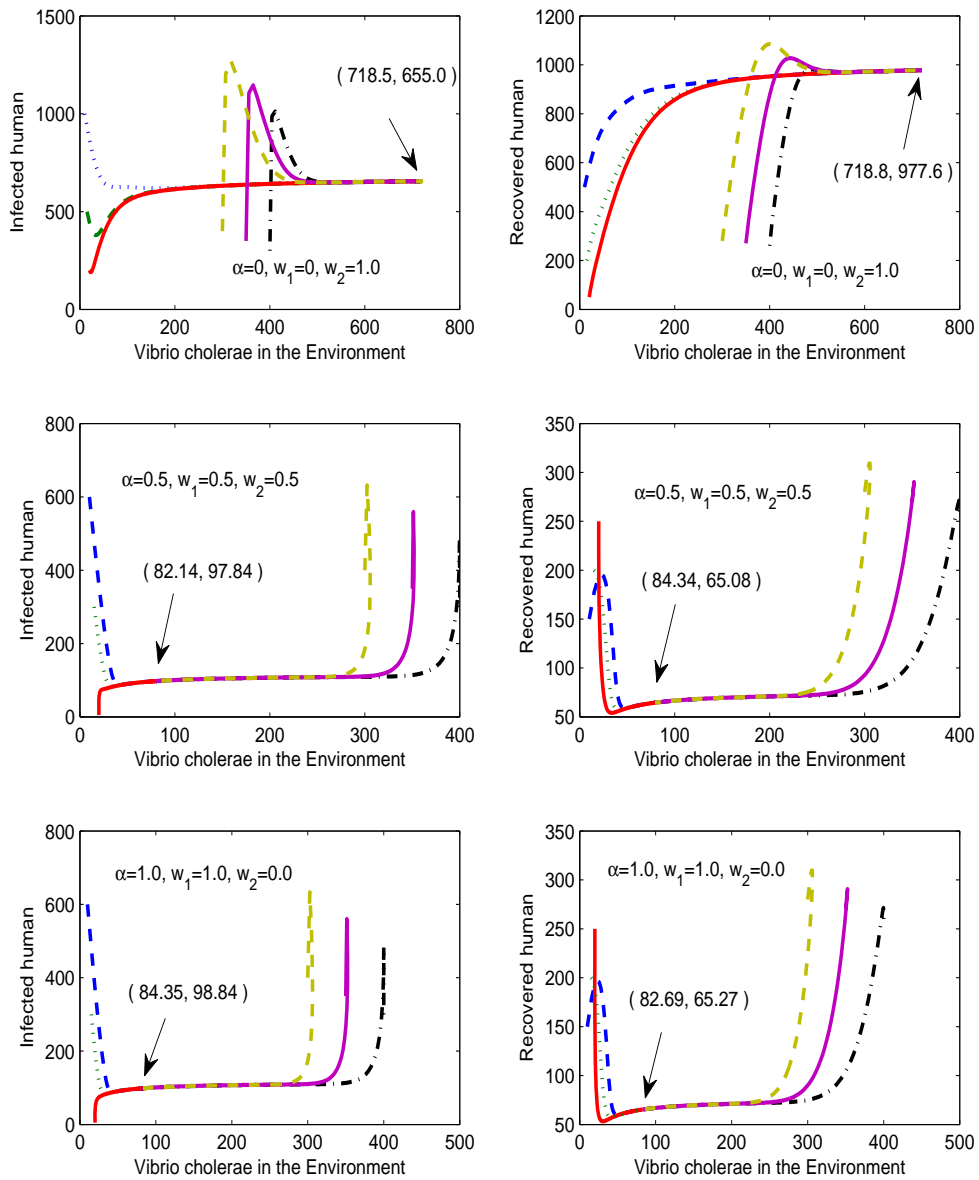
Figure 7.7: Phase space trajectories of infected human and recovered human with respect to Vibrio Cholerae in the environment.
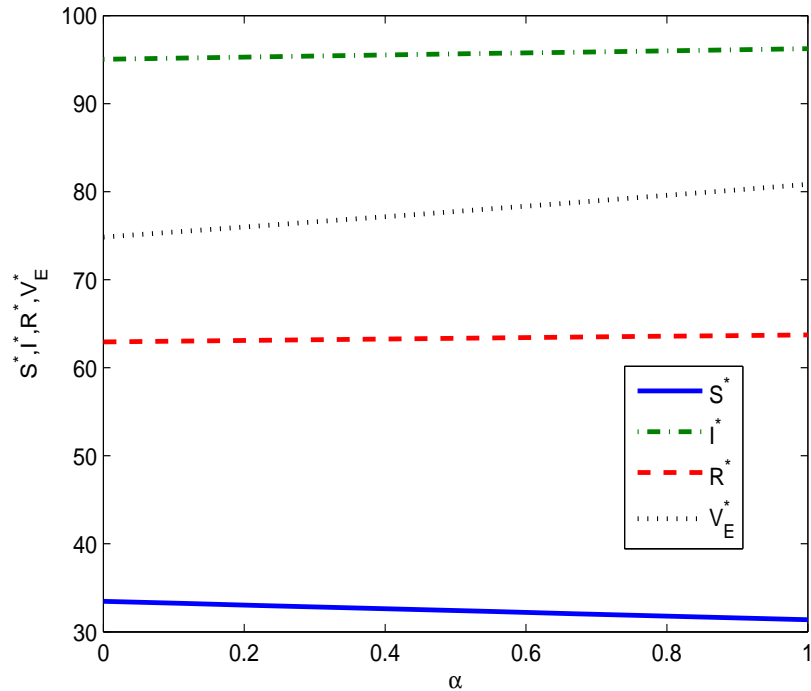
Figure 7.8: Change of equilibrium values with respect to α.

7.8 Conclusion

In this chapter, a Cholera model has been considered incorporating the fuzzyness in all biological parameters due to its natural variability. Here, total human population is divided into three subpopulations such as susceptible human, infected human, recovered human and a bacterial population consists of Vibrio Cholerae in the environment. It is shown that all solutions of our proposed system are bounded under some restriction. Then the possible equilibrium are determined. The local stability analysis of this fuzzy Cholera disease model has been done and it is shown that the system will be disease free and endemic under some conditions discussed earlier. The global stability analysis of this model has been shown in this chapter around the endemic equilibrium point. In numerical simulations, the disease free and endemic equilibrium points have been computed. For fixed value of α and different combinations of weight values w_1 and w_2 we draw different figures from which, it can be concluded that human and bacterial populations have been greatly influenced by imprecise value of the parameters. So, we can say that the fuzzy models are more realistic than the corresponding crisp model since crisp models are the particular case of fuzzy models.

Gateway from Chapter 7 to Chapter 8

In Chapter 7, we have investigated the dynamics of Cholera disease in a uncertain or fuzzy environment. The other important infectious disease which makes several thousands of death over the world is Malaria. It is a vector borne fatal disease caused by a parasite. It spreads in human population through the bites of infected mosquitoes. In the next chapter, we intend to study the dynamics of Malaria disease in a time periodic environment with proper control strategies.

Chapter 8

Threshold Dynamical Behaviors of a Malaria Disease in Control Parameters Based Periodic Environment

8.1   Introduction

Malaria is a disease that can be transmitted to people through the bites of infected mosquitoes. It is a protozoan infection of red blood cells in human by four species of genus Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale and Plasmodium malariae. At first, Ross [192] developed a mathematical model of Malaria disease to study the transmission and control of Malaria in 1911. The Ross model consists of two nonlinear differential equations in two state variables that correspond to the properties of infected human beings and the infected mosquitos. Then, in 1949, Swaroop [229] studied on forecasting of Malaria in Punjab, in India. After that, different mathematical studies had been done by many researchers to investigate the dynamics of Malaria disease and about its control. In 1980, Singer and Cohen [217] reported the impact of recovery rates on the Malaria disease transmission. Koella [120] investigated the importance of the use of mathematical models to understand Malaria transmission in 1991. In 1995, Martens et al. [147] worked on the impact of global climate change on the risk of Malaria. Ngwa and Shu [166] studied a simple mathematical model on Malaria disease with variable human and mosquito populations in 2000. Singh et al. [219] investigated the effects of environmental and ecological fluctuation on the transmission dynamics of Malaria in

2005. Chitnis et al. [45] reported the bifurcation analysis of a mathematical model for Malaria disease transmission in 2006. Then, Wei et al. [249] studied the effects of time delay on the transmission dynamics of Malaria in 2008. In 2010, Saker [202] reported stability and Hopf bifurcation of a Malaria disease transmission model. In the same year, Cai and Li [29] investigated the effects of direct transmission on the transmission of Malaria.

From the life-cycle of mosquitoes, it is known that the climates have an important effects on vector multiplication as well as the development of parasites in the mosquito. It is seen that in moist climates the mosquito breeding is increased and in the arid climates it is restricted. That is, mosquito population fluctuates over time and often exhibits the seasonal behaviors. Already, there exist very few papers on Malaria models in which the periodic environment has been considered. In 2003, Zhao [262] published a book on "Dynamical systems in population biology". In this book, the theoretical study of a non-autonomous system has been explored. Teng et al. [233] investigated the impact of disease induced mortality, the persistence and extinction condition of a disease in non-autonomous system in 2008. In 2010, a mathematical model with periodic birth rate and age structure for the vector population was reported by Lou and Zhao [140]. Nakata and Kuniya [163] studied the global dynamics of a class of SEIRS models in a periodic environment in 2010. In 2011, Bai et al. [17] investigated the effects of seasonality and existence of multiple periodic solutions for an SIR epidemic model. Chitnis et al. [47] studied a mathematical model for the seasonal dynamics of Malaria in mosquitoes in 2012. In 2013, Wang, Teng and Zhao explored a mathematical model on Malaria [246] in which they considered the birth rate of mosquito population logistic and the transmission rates from human to mosquito and mosquito to human are time dependent.

Now, the optimal control is very important to control a disease by taking suitable intervention strategies. Pontryagin's maximum principle [183] is extensively used in a optimal control problem. In 2002, Guyatt et al. [87] studied the impact of use of insecticide-treated nets and indoor residual spraying in highland Kenya to control Malaria. Singh et al. [220] investigated the control of Malaria through indoor residual spraying and larvivorous fish in 2006. In 2009, Sakulku et al. [203] studied the mosquito repellent activity of citronella oil nano emulsion. Thom et al. [237] reported the control of Aedes aegypti mosquitoes by insecticide spray in 2010. In 2012, Lashari and Zaman [126] investigated the optimal control of vector borne disease by introducing suitable control parameters. Agusto et al. [4] studied the impact of bed-net use on Malaria prevalence in 2013. In the same year, Kar and Jana [116] investigated the impact of application of three controls such as treatment, vaccination and pesticide spray in a vector-borne disease. In 2014, Rehman et al. [190] reported the use of plant based products repellents against mosquitoes.

From the above literature review, it is observed that many investigations on the Malaria disease transmission dynamics have been made. But till now there exist some gaps in

the literature which are as follows:

From the above literature survey, it is motivated that due to the seasonal periodicity, the number of mosquito varies periodically. Henceforth the rate of bites by mosquito to human and others also varies with periodic. Again, it has been studied that the controls have significant importance to prevent the mosquitoes bites to human, that is, these have some effects on different transmission rates and death rate of mosquitoes. But in existing literature, these controls have been used directly in the differential equation of the Malaria model. According to our view, it should be used as a function in the transmission rate and death rate. But till now, there is no such consideration in the literature.

To overcome these difficulties a proposed a Malaria disease transmission model has been considered in this chapter. Here, we have developed a Malaria mathematical model in a time periodic environment. Here, three control parameters such as bed-nets, plantation of mosquito repellent plant and spray of insecticide have been considered to study the effects on eradication of Malaria disease. Also, the transmission rate either from human to mosquito or mosquito to human and death rate of infected mosquito have been considered as a function of time and control parameters. It has been proved that Malaria disease goes extinction if $R_0 < 1$ and it uniformly persist if $R_0 > 1$. In autonomous case of our proposed Malaria mathematical model, Hopf bifurcation analysis with respect to the parameters recruitment rate of susceptible human population (λ) and the disease transmission rate from human to mosquito (α) has been done numerically. Next, an optimal control problem has been constructed and solved using Pontryagin's maximum principle. Different possible control strategies and their effectiveness have been discussed by numerical simulations.

8.2 Model Formulation

Based on the transmission mechanism of Malaria, the following mathematical model has been constructed. Most of research papers have considered the birth rate (r) of mosquito population, transmission rate from human to mosquito (α), mosquito to human (β), death rate (d) of mosquito population and environmental carrying capacity K as constants. But, in reality it is seen that r, α, β, d and K may vary from time to time due to many factors in the atmosphere such as temperature, rainfall, humidity etc. So, these should be a function of time. In respect to this, here $K(t)$ and $r(t)$ have been considered as a function of time in the following forms $K(t) = K_0 + b_3\psi(t)$ and $r(t) = r_0 + b_4\phi(t)$ where K_0 and r_0 be the mean values of $K(t)$ and $r(t)$ respectively. Also, b_3 and b_4 are the seasonality constants with the periodic functions $\psi(t)$ and $\phi(t)$ respectively.

Already there exist some works in which transmission rate from mosquito to human $\beta(t)$ has been considered as a function of time such as $\beta(t) = [\beta_0 + b_1\xi(t)]$ where β_0,

b_1 and $\xi(t)$ be the average value of $\beta(t)$, seasonality parameter and a seasonal periodic function of time respectively. Practically to reduce the Malaria disease from a society, some controls such as bed-nets (u_1) and cultivation of mosquito repellent plants (u_2) are used continuously. So, these controls have some effects on transmission rate from mosquito to human. But till now, there is no work of consideration of control parameters in a transmission rate in dynamical model of Malaria disease. So, in this chapter we have considered the transmission rate $\beta(t)$ as a function of time and control parameters in the following form

$$\beta(t) = [\beta_0 + b_1\xi(t) - \lambda_0 u_1 - \lambda_1 u_2]$$

where λ_0 and λ_1 are positive constants and that are chosen in such a way that $\beta(t)$ remains positive and $0 \le u_i \le 1$ for $i = 1, 2$.

Similarly, the transmission rate from human to mosquito depends on natural seasonal periodicity and also on control parameters u_1 and u_2. So, it has been considered as $\alpha(t) = (\alpha_0 + \delta_1\eta(t) - \lambda_0 u_1 - \lambda_1 u_2)$ where α_0, δ_1 and $\eta(t)$ be the mean value of $\alpha(t)$, seasonality constant and seasonal periodic function of time respectively.

Again, in the literature it is seen that there was no consideration of time and control parameter dependent death rate of mosquito in the Malaria model. Basically, when pesticide are used then the normal death rate of mosquito increases seasonally. In this regard, here the death rate $d(t)$ has been considered in the following form

$$d(t) = [d_0 + b_2\chi(t) + \lambda_2 u_3]$$

where $\chi(t)$ is a seasonal periodic function and u_3 is the application of pesticide as a control parameter. Here λ_2 is a positive constant and $0 \le u_3 \le 1$. It is also assumed that $r(t), K(t), \alpha(t), d(t)$ and $\beta(t)$ are continuous, positive ω periodic function.

Under the above considerations, a Malaria model has been developed as follows:

$$\left.\begin{array}{rcl}
\dfrac{dS_M}{dt} & = & r(t)S_M(1 - \frac{S_M}{K(t)}) - \alpha(t)S_M I_H - \lambda_2 u_3 S_M \\[2ex]
\dfrac{dI_M}{dt} & = & \alpha(t)S_M I_H - d(t)I_M \\[2ex]
\dfrac{dS_H}{dt} & = & \lambda - \beta(t)S_H I_M - \delta S_H + \gamma R_H \\[2ex]
\dfrac{dI_H}{dt} & = & \beta(t)S_H I_M - (\delta + \mu + \sigma)I_H \\[2ex]
\dfrac{dR_H}{dt} & = & \mu I_H - \delta R_H - \gamma R_H
\end{array}\right\} \qquad (8.1)$$

with subject to the initial conditions
$S_M(0) \geq 0, I_M(0) \geq 0, S_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0$ where $S_H(t), I_H(t)$ and $R_H(t)$ denote the susceptible, infected and recovered for human population at time t respectively. $S_M(t)$ and $I_M(t)$ represent the densities of the susceptible and infective mosquito populations at time t. Here λ, δ, γ, μ and σ are known as the recruitment rate for human, the natural death rate for human, the portion of the recovered human who becomes susceptible again, the portion of infected human to be recovered and the disease induced death rate respectively.

8.3 Boundedness of Solutions

In this section, the boundedness of all solutions of the system (8.1) has been discussed in subsequent steps.

Theorem 8.1 The solution of the system (8.1) i.e., $(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t))$ is nonnegative.

Proof. It is assumed that $(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t))$ is defined for all $t \in [0, T)$ where $T > 0$. Then, integrating the first equation of system (8.1) in $[0, t]$, we have

$$\int_0^t \frac{dS_M(t)}{S_M(t)} = \int_0^t \left(r(s) - \frac{r(s)}{K(s)} S_M(s) - \alpha(s) I_H(s) - \lambda_2 u_3 \right) ds$$

$$i.e., S_M(t) = S_M(0) exp \left(\int_0^t \left(r(s) - \frac{r(s)}{K(s)} S_M(s) - \alpha(s) I_H(s) - \lambda_2 u_3 \right) ds \right)$$

Now, from the above equation it is observed that $S_M(t) > 0$ for all $t \in [0, T)$ since $S_M(0) > 0$. Suppose there exist a time $t_1 \in (0, T)$ such that $min\{I_M(t_1), I_H(t_1)\} = 0$. Again, since $I_M(0) > 0$ and $I_H(0) > 0$ then we can also assume that $min\{I_M(t), I_H(t)\} > 0$ for all $t \in [0, t_1)$. If $min\{I_M(t_1), I_H(t_1)\} = I_M(t_1)$ since, $I_H > 0$ because of $min\{I_M(t_1), I_H(t_1)\} = I_M(t_1) = 0$ and $S_M(t) > 0$ for all $t \in [0, T)$ then $\alpha(t) S_M I_H \geq 0$. Now, from the second equation of system (8.1) we have

$$\frac{dI_M(t)}{dt} \geq -d(t) I_M \text{ for all } t \in [0, t_1]$$

$$i.e., 0 = I_M(t_1) \geq I_M(0) exp \left(-\int_0^{t_1} d(s) ds \right) > 0$$

which leads to a contradiction. Again, when $min\{I_M(t_1), I_H(t_1)\} = I_H(t_1)$ then from the fifth equation of system (8.1), we have

$$\frac{dR_H(t)}{dt} > -(\delta + \gamma) R_H, \text{ for all } t \in [0, t_1)$$

$$i.e., R_H(t) > R_H(0) exp \left(-(\delta + \gamma) t \right) \geq 0, \text{ for all } t \in (0, t_1]$$

From the third equation of system (8.1) we have

$$\frac{dS_H(t)}{dt} \geq -\left(\beta(t)I_M(t) + \delta\right)S_H, \text{ for all } t \in [0, t_1)$$

$$i.e., S_H(t) \geq S_H(0)exp\left(-\int_0^t (\beta(s)I_M(s) + \delta)ds\right) > 0 \text{ for all } t \in [0, t_1]$$

In the interval $[0, t_1]$, it is also obtained that

$$\frac{dI_H(t)}{dt} \geq -(\delta + \mu + \sigma)I_H$$

$$i.e., 0 = I_H(t_1) \geq I_H(0)exp\left(-(\delta + \mu + \sigma)t_1\right) > 0$$

which leads to a contradiction. This proves that $I_M(t) > 0$, $I_H(t) > 0$ for all $t \in [0, T)$. Then, from the fifth equation of system (8.1) we have

$$\frac{dR_H(t)}{dt} > -(\delta + \gamma)R_H, \text{ for all } t \in [0, T)$$

$$i.e., R_H(t) > R_H(0)exp\left(-(\delta + \gamma)t\right) \geq 0 \text{ for all } t \in (0, T).$$

Again, from the third equation of system (8.1) we have

$$\frac{dS_H(t)}{dt} \geq -\left(\beta(t)I_M(t) + \delta\right)S_H, \text{ for all } t \in [0, T)$$

$$i.e., S_H(t) \geq S_H(0)exp\left(-\int_0^t (\beta(s)I_M(s) + \delta)ds\right) > 0 \text{ for all } t \in [0, T).$$

This proves that $(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t))$ is nonnegative in $[0, T)$ where $T > 0$.

Theorem 8.2 Let $(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t))$ be the solution of the system (8.1). Then $(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t))$ is bounded for all $t \geq 0$ provided that $r^L > \lambda_2 u_3$.

Proof. From the last three equations of the model (8.1), it is obtained that

$$\frac{d(S_H + I_H + R_H)}{dt} = \lambda - \delta(S_H + I_H + R_H) - \sigma I_H$$

$$i.e., \frac{d(S_H + I_H + R_H)}{dt} \leq \lambda - \delta(S_H + I_H + R_H), \text{ since } I_H \text{ is nonnegative.}$$

$$i.e., \frac{d(S_H + I_H + R_H)}{dt} + \delta(S_H + I_H + R_H) \leq \lambda$$

Then solving above linear equation it is obtained that

$$(S_H + I_H + R_H) \leq \frac{\lambda}{\delta} + c_1 e^{-\delta t}$$

where c_1 is a constant to be depended on initial conditions. Now, if $\delta > 0$ and $t \to \infty$, it is obtained that

$$S_H + I_H + R_H \leq \frac{\lambda}{\delta}$$

So, from the above equation, we can write $S_H \leq \frac{\lambda}{\delta}, I_H \leq \frac{\lambda}{\delta}$ and $R_H \leq \frac{\lambda}{\delta}$

i.e., $(S_H(t), I_H(t), R_H(t))$ is bounded.

For a continuous,positive ω periodic system, let us assume that $g^L = sup_{t \in [0,\omega)} g(t)$ and $g^M = inf_{t \in [0,\omega)} g(t)$.

Again, from the first equation of the system (8.1), we have

$$\frac{dS_M}{dt} = r(t)S_M(1 - \frac{S_M}{K(t)}) - \alpha(t)S_M I_H - \lambda_2 u_3 S_M$$

$$i.e., \frac{dS_M}{dt} \leq r^L S_M - \frac{r^M S_M^2}{K^L} - \lambda_2 u_3 S_M, \text{ since } S_M, I_H \text{ are nonnegative.}$$

$$i.e., \frac{dS_M}{dt} \leq S_M \left((r^L - \lambda_2 u_3) - \frac{r^M S_M}{K^L} \right)$$

$$i.e., \frac{dS_M}{dt} \leq S_M \left(r_1 - \frac{r^M S_M}{K^L} \right), \text{ where } r_1 = (r^L - \lambda_2 u_3)$$

$$i.e., \frac{dS_M}{dt} \leq \frac{r^M}{K^L} S_M \left(\frac{K^L r_1}{r^M} - S_M \right)$$

Solving the above equation, it is obtained that

$$S_M \leq \frac{c_2 r_1 K^L}{r^M(c_2 + e^{-\frac{r_1 r^M}{K^L}t})}$$

Now, if $r_1 > 0$ i.e., $r^L > \lambda_2 u_3$ then as t tends to infinity, it is obtained that

$$S_M \leq \frac{r_1 K^L}{r^M}$$

$$i.e., S_M \leq \frac{(r^L - \lambda_2 u_3)K^L}{r^M}$$

Also, from the second equation of system (8.1) we have

$$\frac{dI_M}{dt} = \alpha(t)S_M I_H - d(t)I_M$$

$$i.e., \frac{dI_M}{dt} \leq \frac{\alpha^L r_1 K^L \lambda}{r^M \delta} - d^M I_M$$

$$i.e., \frac{dI_M}{dt} + d^M I_M \leq \frac{\alpha^L r_1 K^L \lambda}{r^M \delta}$$

Now, solving above equation, it is obtained that

$$I_M \leq \frac{\alpha^L r_1 K^L \lambda}{r^M \delta} + c_3 e^{-d^M t}$$

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Since $d^M > 0$, then as t tends to infinity, the above equation reduces to

$$I_M \leq \frac{\alpha^L r_1 K^L \lambda}{r^M \delta}$$

Hence, all the solutions of the system (8.1) are bounded.

8.4   Basic Reproduction Number

Now, the disease free equilibrium point $E_0(t)$ for the system (8.1) is given by
$E_0(t) = (S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$, where $S_M^*(t)$ satisfies the equation

$$\frac{dS_M}{dt} = r(t)S_M(1 - \frac{S_M}{K(t)}) - \lambda_2 u_3 S_M \tag{8.2}$$

Let (R^k, R_+^k) be the standard ordered k-dimensional Euclidean space with a norm $||.||$.
For $u, v \in R^k$, we write $u \geq v$ provided $u - v \in R_+^k$, $u > v$ provided $u - v \in R_+^k/\{0\}$,
$u >> v$ provided $u - v \in Int(R_+^k)$ respectively.
Let $A(t)$ be a continuous, irreducible, cooperative and ω periodic $k \times k$ matrix function,
$\phi_A(t)$ be the fundamental solution matrix of

$$\frac{dx}{dt} = A(t)x \tag{8.3}$$

Let $\rho(\phi_A(\omega))$ be the spectral radius of $\phi_A(\omega)$. By the Perron-Frobenious theorem,
$\rho(\phi_A(\omega))$ is the principle eigenvalue of $\phi_A(\omega)$ in the sense that it is simple and admits a eigenvector $v^* >> 0$. This is useful for the study of global stability of $E_0(t)$ in
the next section.

Lemma 8.1 ([259]). Let $p = \frac{1}{\omega} ln\rho(\phi_A(\omega))$. Then, there exits a positive ω periodic
function $v(t)$ such that $e^{pt}v(t)$ is a solution of (8.3).

Now, the system (8.1) can be written as

$$\dot{x} = \mathcal{F}(t, x) - \nu(t, x) \tag{8.4}$$

where $x = (I_M, I_H, S_M, S_H, R_H)^T$, $\nu(t, x) = \nu^-(t, x) - \nu^+(t, x)$,

$$\mathcal{F}(t, x) = \begin{pmatrix} \alpha(t)S_M I_H \\ \beta(t)S_H I_M \\ 0 \\ 0 \\ 0 \end{pmatrix}, \nu^-(t, x) = \begin{pmatrix} d(t)I_M \\ (\delta + \mu + \sigma)I_H \\ \alpha(t)S_M I_H + \lambda_2 u_3 + \frac{r(t)S_M^2}{K(t)} \\ \beta(t)S_H I_M + \delta S_H \\ \delta S_H + \gamma R_H \end{pmatrix}$$

and $\nu^+(t,x) = \begin{pmatrix} 0 \\ 0 \\ r(t)S_M \\ \lambda + \gamma R_H \\ \mu I_H \end{pmatrix}$

Now, to check the conditions $(A1)$ to $(A7)$ in [244]. It is easy to see that the conditions $A1$ to $A5$ are satisfied. Now, we define $f(t,x) = \mathcal{F}(t,x) - \nu(t,x)$ and $M(t) = \left(\frac{\partial f_i(t,x^*)}{\partial x_j}\right)_{3 \leq i,j \leq 5}$ where $f_i(t,x)$ and x_i is the i−th component of $f(t,x)$ and x respectively. Then $M(t)$ is obtained as follows

$$M(t) = \begin{pmatrix} r(t) - \frac{2r(t)}{K(t)}S_M^*(t) - \lambda_2 u_3 & 0 & 0 \\ 0 & -\delta & \gamma \\ 0 & 0 & -(\delta + \gamma) \end{pmatrix}$$

Now,

$$exp\left(\int_0^\omega \left(r(t) - \frac{2r(t)}{K(t)}S_M^*(t) - \lambda_2 u_3\right)dt\right)$$
$$= exp\left(\int_0^\omega \left(r(t) - \frac{r(t)}{K(t)}S_M^*(t) - \frac{r(t)}{K(t)}S_M^*(t) - \lambda_2 u_3\right)dt\right).$$
$$= exp\left(\int_0^\omega \left(-\frac{r(t)}{K(t)}S_M^*(t)\right)dt\right) < 1.$$

Since $r(t) - \frac{r(t)}{K(t)}S_M^*(t) - \lambda_2 u_3 = 0$ and $S_M^*(t)$ is a ω periodic solution then $\int_0^\omega \left(r(t) - \frac{r(t)}{K(t)}S_M^*(t) - \lambda_2 u_3\right)dt = 0$. So, $\rho(\phi_M(\omega)) < 1$ and the condition $A6$ is also satisfied.

Next, we calculate $F(t)$ and $V(t)$ which are defined by $F(t) = \left(\frac{\partial \mathcal{F}_i(t,x^*(t))}{\partial x_j}\right)_{1 \leq i,j \leq 2}$ and $V(t) = \left(\frac{\partial \nu_i(t,x^*(t))}{\partial x_j}\right)_{1 \leq i,j \leq 2}$ where $\mathcal{F}_i(t,x)$ and $\nu_i(t,x)$ are the i-th component of $\mathcal{F}(t,x)$ and $\nu(t,x)$ respectively. Then, we have $F(t) = \begin{pmatrix} 0 & \alpha(t)S_M^*(t) \\ \frac{\beta(t)\lambda}{\delta} & 0 \end{pmatrix}$ and $V(t) = \begin{pmatrix} d(t) & 0 \\ 0 & (\delta + \mu + \sigma) \end{pmatrix}$.

Let $Y(t,s)$ is 2×2 matrix solution of the system

$$\frac{d}{dt}Y(t,s) = -V(t)Y(t,s) \text{ for any } t \geq s, Y(s,s) = I.$$

where I is the 2×2 identity matrix. Hence, the condition $A7$ is also satisfied.

Let C_ω be the ordered Banach space of all ω periodic function from R to R^2 and which is equipped with maximum norm $||.||$ and the positive cone
$C_\omega^+ = \{\phi \in C_\omega : \phi(t) \geq 0, \text{for any} t \in R\}$. Consider the following linear operator $L : C_\omega \to C_\omega$ by

$$(L\phi)(t) = \int_0^{+\infty} Y(t, t-a)F(t-a)\phi(t-a)da \text{ for any } t \in R, \phi \in C_\omega$$

Then, we define the basic reproduction number R_0 of system (8.1) as $R_0 = \rho(L)$.

From the above discussion, we obtain the following result for the local stability of the disease free periodic solution $E_0(S_M^*, 0, \frac{\lambda}{\delta}, 0, 0)$ for system (8.1).

Theorem 8.3 ([259]). The following statements are valid:
(i) $R_0 = 1$ if and only if $\rho(\phi_{F-V}(\omega)) = 1$.
(ii) $R_0 > 1$ if and only if $\rho(\phi_{F-V}(\omega)) > 1$.
(iii) $R_0 < 1$ if and only if $\rho(\phi_{F-V}(\omega)) < 1$.
Thus $E_0(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ of (7.1) is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

8.5 Threshold Dynamics

In this section, we show that if $R_0 < 1$ then the disease free equilibrium point $E_0(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ is globally asymptotically stable. Next, we show that if $R_0 > 1$ then the Malaria disease is persistent in the human population.

Theorem 8.4 If $R_0 < 1$ then the disease free periodic solution $(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ is globally asymptotically stable.

Proof. From Theorem 8.3 if $R_0 > 1$ then $(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ is unstable and if $R_0 < 1$ then $(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ is locally asymptotically stable. Hence, it is sufficient to show that the global attractivity of $(S_M^*(t), 0, \frac{\lambda}{\delta}, 0, 0)$ for $R_0 < 1$. From Theorem 8.2 we have $0 \leq S_H \leq \frac{\lambda}{\delta}$ and $0 \leq S_M \leq S_M^*(t)$. Then, from the second and forth equation of system (8.1) we have

$$\frac{dI_M}{dt} \leq \alpha(t)S_M^*(t)I_H - d(t)I_M$$
$$\frac{dI_H}{dt} \leq \beta(t)\frac{\lambda}{\delta}I_M - (\delta + \mu + \sigma)I_H \tag{8.5}$$

Now, we consider the auxiliary system as follows

$$\frac{d\hat{I}_M}{dt} = \alpha(t)S_M^*(t)\hat{I}_H - d(t)\hat{I}_M$$

$$\frac{d\hat{I}_H}{dt} = \beta(t)\frac{\lambda}{\delta}\hat{I}_M - (\delta + \mu + \sigma)\hat{I}_H$$

which can be written as

$$\frac{dX}{dt} = (F(t) - V(t))X \tag{8.6}$$

where $X = (\hat{I}_M, \hat{I}_H)^T$.

Since $(F(t) - V(t))$ is continuous, cooperative and irreducible, then by Lemma 8.1 there exist a ω periodic function $\tilde{X}(t)$ such that $X(t) = e^{pt}\tilde{X}(t)$ is a solution of (8.6) where $p = \frac{1}{\omega}ln\rho(\phi_{F-V}(\omega))$. Again, from Theorem 8.3 if $R_0 < 1$ then p is a negative constant. Therefore, we have the solution of system (8.6), $X(t) \to 0$ as $t \to \infty$.

Thus, the zero solution of system (8.6) will be globally stable. Then, for the nonnegative initial condition of system (8.5) there exist a sufficient large number $N > 0$ such that $(I_M(0), I_H(0))^T \leq N\tilde{X}(t)$. According to the comparison principle [222], we have $(I_M, I_H)^T \leq N\tilde{X}(t)$ for all $t > 0$ where $N\tilde{X}(t)$ is also a solution of (8.6). Then, we have $I_M \to 0$ and $I_H \to 0$ as $t \to \infty$. Then from the asymptotic autonomous system [236], it follows then $S_M \to S_M^*(t)$, $S_H \to \frac{\lambda}{\delta}$ and $R_H \to 0$ as $t \to \infty$.
Hence $E_0(t)$ is globally asymptotically stable if $R_0 < 1$.

Theorem 8.5 If $R_0 > 1$ the system (8.1) is uniformly persistent i.e., there exits a positive constant ϵ, such that for all initial condition $S_M(0) \geq 0, I_M(0) \geq 0, S_H(0) \geq 0,$ $I_H(0) \geq 0, R_H(0) \geq 0$, the solution of (8.1) satisfies $lim_{t\to\infty}(S_M(t), I_M(t), S_H(t), I_H(t), R_H(t)) \geq (\epsilon, \epsilon, \epsilon, \epsilon, \epsilon)$.

Proof. Similar to Wang, Teng and Zhang [246].

8.6 Optimal Control Problem

In this section, our objective is to minimize the total number of infected humans by controlling parameters optimally as well as to minimize the total systemic cost to be required to apply the controls. Also, we want to minimize the side effects of the control parameters in the process of applying optimal control approach. Keeping these view in mind, the objective functional of our proposed optimal control problem has been defined as follows

$$J(u_1, u_2, u_3) = \min_{u_1, u_2, u_3} \int_0^{t_f} (A_1 I_H + A_2 u_1^2 + A_3 u_2^2 + A_4 u_3^2)dt \tag{8.7}$$

subject to the system of differential equation (8.1).

Our target is to obtain a control set (u_1^*, u_2^*, u_3^*) such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{u_1, u_2, u_3 \in \oplus} J(u_1, u_2, u_3)$$

where $\oplus = \{u : \text{ is measurable and } 0 \leq u(t) \leq 1 \text{ for all } t \in [0, t_f]\}$. Here A_1 is the weight constant associated with the infected human. Also, the square of the control variables [111, 116] are taken here due to the side effects and overdose of three controls and A_2, A_3, A_4 are the weights associated with the square of the control variables.

Now, to solve the optimal control problem (8.7) with the help of system (8.1), we construct a lagrangian as follows

$$L = A_1 I_H + A_2 u_1^2 + A_3 u_2^2 + A_4 u_3^2 \tag{8.8}$$

To minimize the lagrangian, we have constructed the hamiltonian of the problem as follows

$$H = A_1 I_H + A_2 u_1^2 + A_3 u_2^2 + A_4 u_3^2 + \lambda_{S_M} \frac{dS_M}{dt} + \lambda_{I_M} \frac{dI_M}{dt} + \lambda_{S_H} \frac{dS_H}{dt} + \lambda_{I_H} \frac{dI_H}{dt} + \lambda_{R_H} \frac{dR_H}{dt}$$

where $\lambda_{S_M}, \lambda_{I_M}, \lambda_{S_H}, \lambda_{I_H}, \lambda_{R_H}$ are the adjoint variables. The adjoint variables can be determined from the equations

$$\frac{\lambda_{S_M}}{dt} = \lambda_{S_M}\left(\lambda_2 u_3 + \frac{2r(t)S_M}{K(t)} - r(t)\right) + \alpha(t)I_H(\lambda_{S_M} - \lambda_{I_M})$$

$$\frac{\lambda_{I_M}}{dt} = \lambda_{I_M}d(t) + \beta(t)S_H(\lambda_{S_H} - \lambda_{I_H})$$

$$\frac{\lambda_{S_H}}{dt} = \delta\lambda_{S_H} + \beta(t)I_M(\lambda_{S_H} - \lambda_{IH})$$

$$\frac{\lambda_{I_H}}{dt} = \alpha(t)S_M(\lambda_{S_M} - \lambda_{I_M}) + (\delta + \mu + \sigma)\lambda_{I_H} - A_1 - \mu\lambda_{R_H}$$

$$\frac{\lambda_{R_H}}{dt} = (\delta + \gamma)\lambda_{R_H} - \gamma\lambda_{S_H}$$

and satisfies the transversality condition $\lambda_{S_M}(t_f) = 0.0$, $\lambda_{I_M}(t_f) = 0.0$, $\lambda_{S_H}(t_f) = 0.0$, $\lambda_{I_H}(t_f) = 0.0$, $\lambda_{R_H}(t_f) = 0.0$.

Theorem 8.6 The optimal control set (u_1^*, u_2^*, u_3^*) which minimizes J over the region $\oplus$ defined earlier, is given by

$$u_1^* = max\{0, min(\hat{u}_1)\}$$
$$u_2^* = max\{0, min(\hat{u}_2)\}$$
$$u_3^* = max\{0, min(\hat{u}_3)\}$$

where $\hat{u}_1 = \frac{\lambda_0}{2A_2}(S_M I_H(\lambda_{I_M} - \lambda_{S_M}) + S_H I_M(\lambda_{I_H} - \lambda_{S_H}))$,
$\hat{u}_2 = \frac{\lambda_1}{2A_3}(S_M I_H(\lambda_{I_M} - \lambda_{S_M}) + S_H I_M(\lambda_{I_H} - \lambda_{S_H}))$,
$\hat{u}_3 = \frac{\lambda_2(S_M\lambda_{S_M} + I_M\lambda_{I_M})}{2A_4}$.

Proof. Similar to [116].

8.7 Numerical Simulation

8.7.1 Without Control

To check the feasibility of our analysis regarding the stability conditions for the system (8.1), we have explored some numerical computations using MATLAB in the following problem.

Problem 1: Here, the parametric values of system (8.1) are $\lambda = 0.05, \delta = 0.006, \mu = 0.05, \sigma = 0.01, \gamma = 0.1$, $r(t) = 3 + 0.1sin(\frac{1}{6}\pi t)$, $K(t) = 2 + 0.3cos(\frac{1}{6}\pi t)$, $\alpha(t) = 0.0022(2 + 0.03sin(\frac{1}{6}\pi t))$, $d(t) = 0.5 + 0.1cos(\frac{1}{6}\pi t)$, $\beta(t) = 0.011(7 + 0.06sin(\frac{1}{6}\pi t))$, $\lambda_0 = 0.0, \lambda_1 = 0.0, \lambda_2 = 0.0, u_1 = u_2 = u_3 = 0.0$.

Using the above set of parametric values, we have $R_0 = 0.5375$ which is less than one. So, according to **Theorem 8.4** the system (8.1) is disease free which is also confirmed by Figure 8.1 where it has been shown that after some times infected mosquito, infected human and recovered human tend to zero.
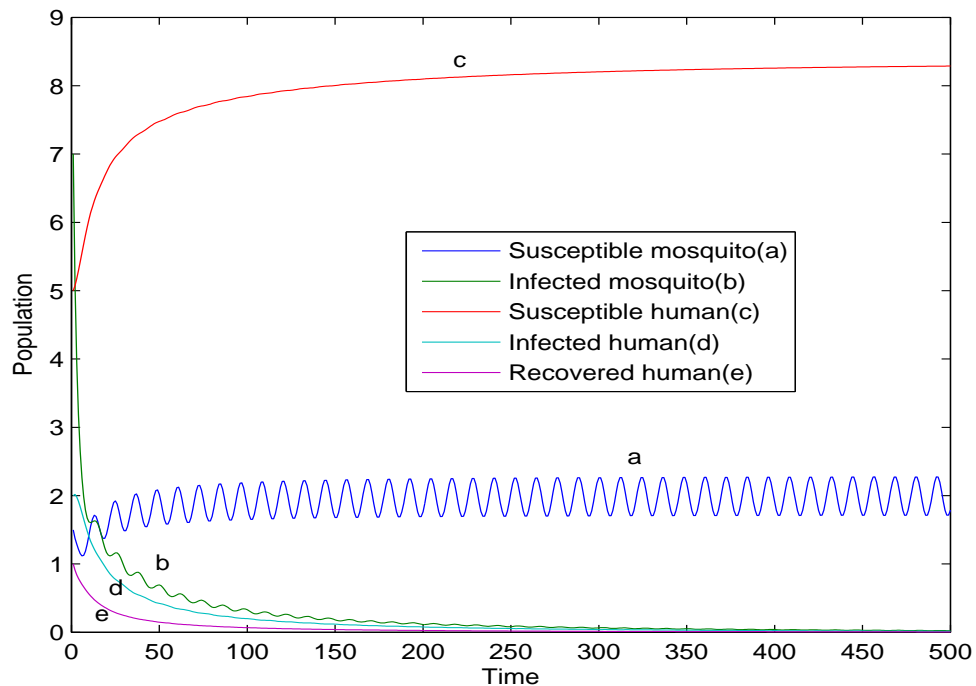

Figure 8.1: Represents the mosquito and human populations for $R_0 < 1$.

Problem 2: Here, $\alpha(t) = 0.02(2 + 0.03sin(\frac{1}{6}\pi t))$ and others to be same as **Problem 1**. For these values, the reproduction number R_0 is 4.8859 which is grater than one. So, according to **Theorem 8.5** the system (8.1) for such parameters is endemic which is

also depicted by Figure 8.2 where it has been shown that mosquito population as well as human population do not vanish as time increases.
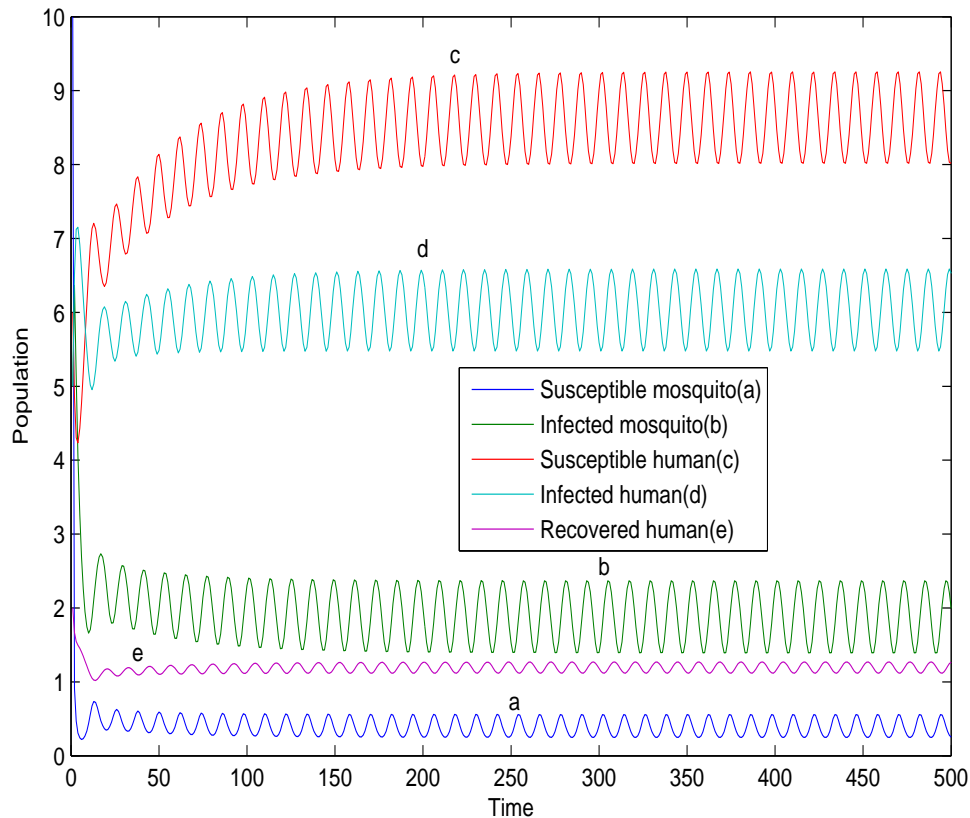

Figure 8.2: Represents the mosquito and human populations for $R_0 > 1$.

8.7.2 With Control

Problem 3: It is same as **Problem 2** including control parameters.
Now, considering $u_1 = 0.5, u_2 = 0.5, u_3 = 0.5$ with $\lambda_0 = \lambda_1 = \lambda_2 = 0.3$, we have the reproduction number R_0 to be 0.6327 which is less than one. Therefore, the endemic system in **Problem 2** becomes disease free using controls. Graphically by Figure 8.3, it is also shown that the infected human, recovered human and infected mosquito vanish ultimately.
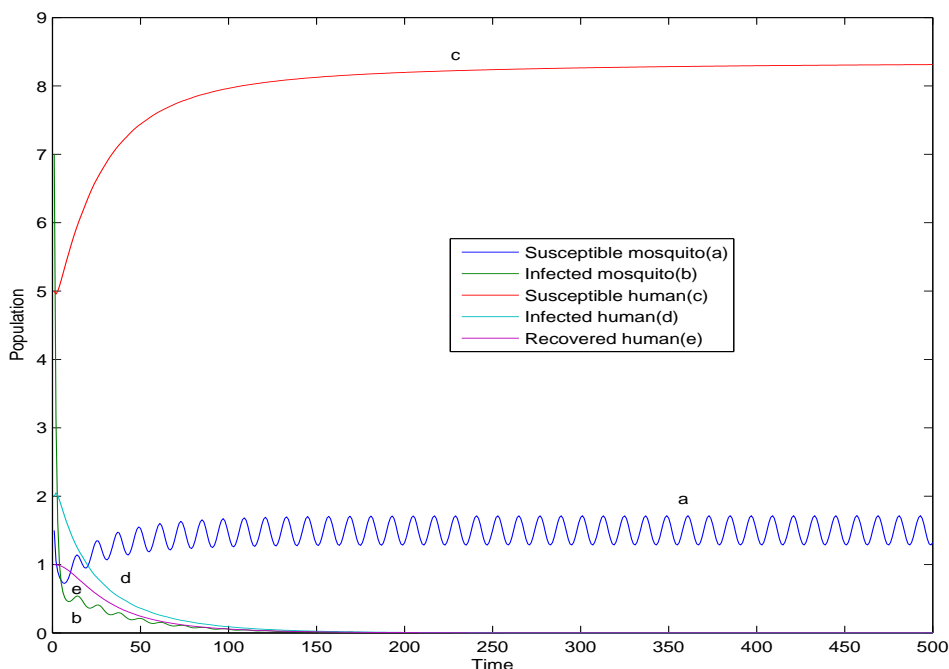
Figure 8.3: Represents the mosquito and human populations in the presence of control parameters for $R_0 = 0.6327 < 1$.

8.7.3 With Hopf bifurcation for Autonomous Model

In this section, the growth rate of susceptible mosquito, environmental carrying capacity of mosquito, the transmission rate from human to mosquito as well as mosquito to human and death rate of infected mosquito have been considered as a constant without any control parameter i.e., $r(t) = r, K(t) = K, \alpha(t) = \alpha, \beta(t) = \beta, d(t) = d, u_1 = 0, u_2 = 0$ and $u_3 = 0$. Then our proposed system (8.1) has been converted to an autonomous system of nonlinear differential equations.

Problem 4: For this autonomous case the following parametric values have been considered as $r = 2.0, K = 10.0, d = 0.1, \delta = 0.06, \beta = 0.012, \mu = 0.05, \sigma = 0.01, \gamma = 0.03$. For this parametric values, it is seen that the autonomous system undergoes Hopf bifurcation with respect to the parameter λ (recruitment rate of human) and α (transmission rate form human to mosquito). If we change the value of λ from 30 to 40 and α from 0.05 to 0.08 then the Hopf bifurcation have been shown in Figure 8.4 and Figure 8.5 respectively. It is clear from the bifurcation diagram that for lower values of λ and α the autonomous system becomes stable, but, above a threshold value of $\lambda = 32.0$ and $\alpha = 0.0638$ the autonomous system losses its stability and periodic solution arises
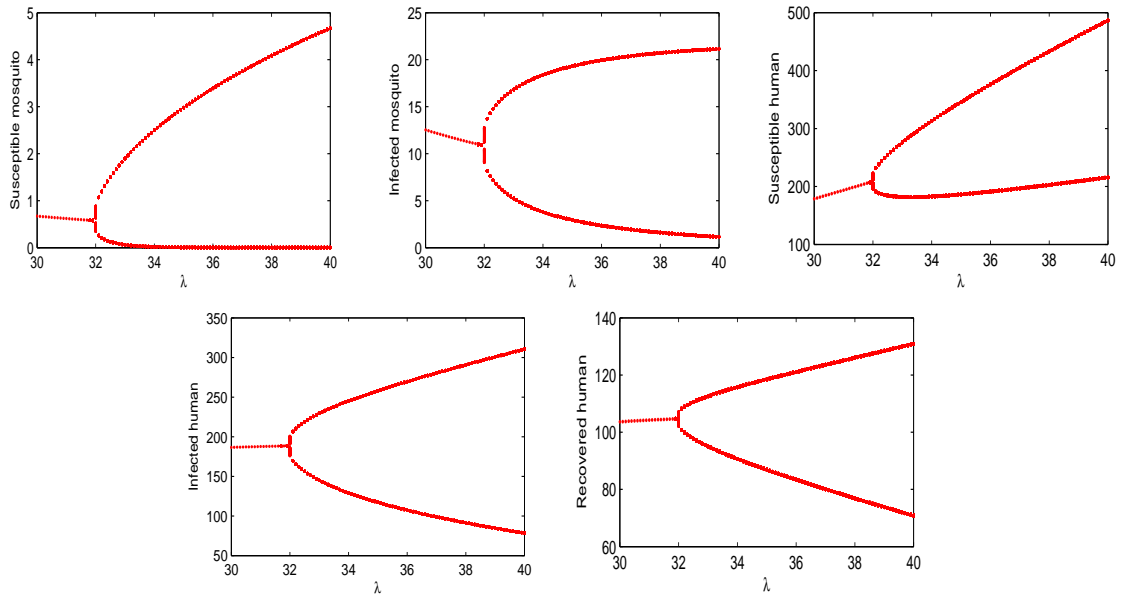
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through Hopf bifurcation.

Figure 8.4: Represents the bifurcation diagram of the autonomous model without control.
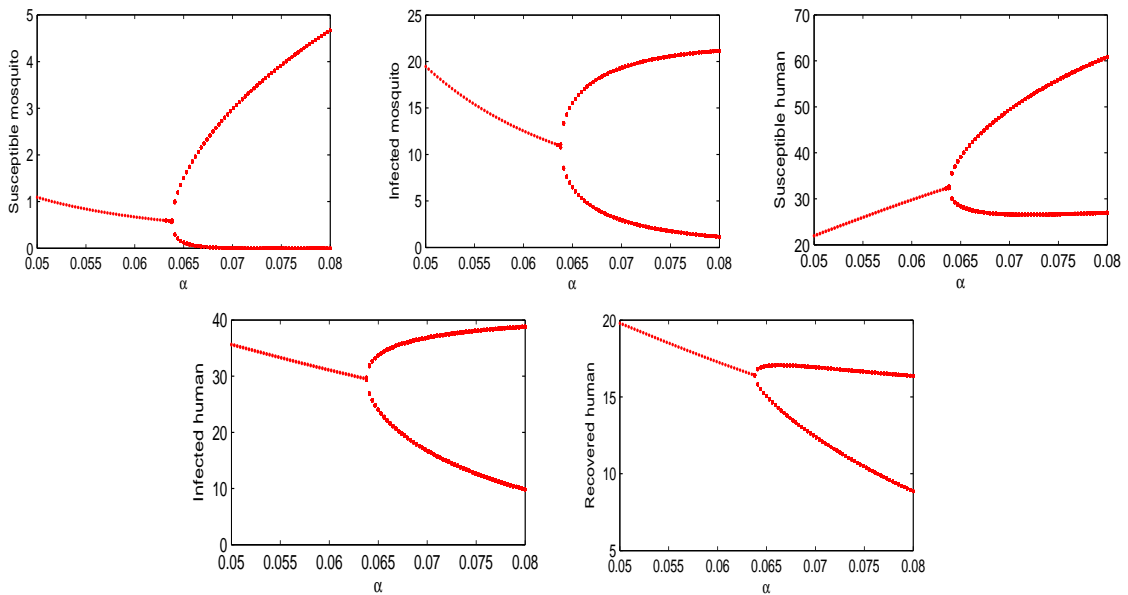

Figure 8.5: Represents the bifurcation diagram of the autonomous model without control.

8.7.4 With Optimal Control Problem

In this section, we have solved the optimal control problem (8.7) with the help of the fourth order Runge-Kutta method in a finite interval of time. First, we solve the state variables by fourth order forward Runge-Kutta method and then using these we solve the adjoint variables by fourth order backward Runge-Kutta method with the help of transversality conditions and the optimality conditions [127]. For this simulations, we consider the following set of parametric values:

$\lambda = 100, \delta = 0.6, \mu = 0.6, \sigma = 0.8, \gamma = 0.7, r_0 = 9.3, b_4 = 0.03333, K_0 = 20.0,$

$b_3 = 0.06, \alpha_0 = 0.3, \delta_1 = 0.015, \lambda_0 = 0.04, \lambda_1 = 0.01, \lambda_2 = 0.2, \beta_0 = 0.2,$

$b_1 = 0.01, d_0 = 1.0, b_2 = 0.1, A_1 = 1.0, A_2 = 0.5, A_3 = 0.1, A_4 = 0.4$

and $\psi(t) = \phi(t) = \xi(t) = \eta(t) = sin(\frac{\pi}{6}t), \chi(t) = cos(\frac{\pi}{6}t)$.

In this model to reduce the infected humans from Malaria disease three control parameters such as bed-nets, cultivation of mosquito repellent plant and pesticide spray have been used. To examine the effect of control parameters numerically possible eight strategies such as use of (*i*) all control variables, (*ii*) without any control, (*iii*) bed-nets control, (*iv*) cultivation of mosquito repellent plant, (*v*) pesticide spray, (*vi*) bed-nets and cultivation of mosquito repellent plant, (*vii*) bed-nets and pesticide spray and (*viii*) cultivation of mosquito repellent plants and pesticide spray have been considered.
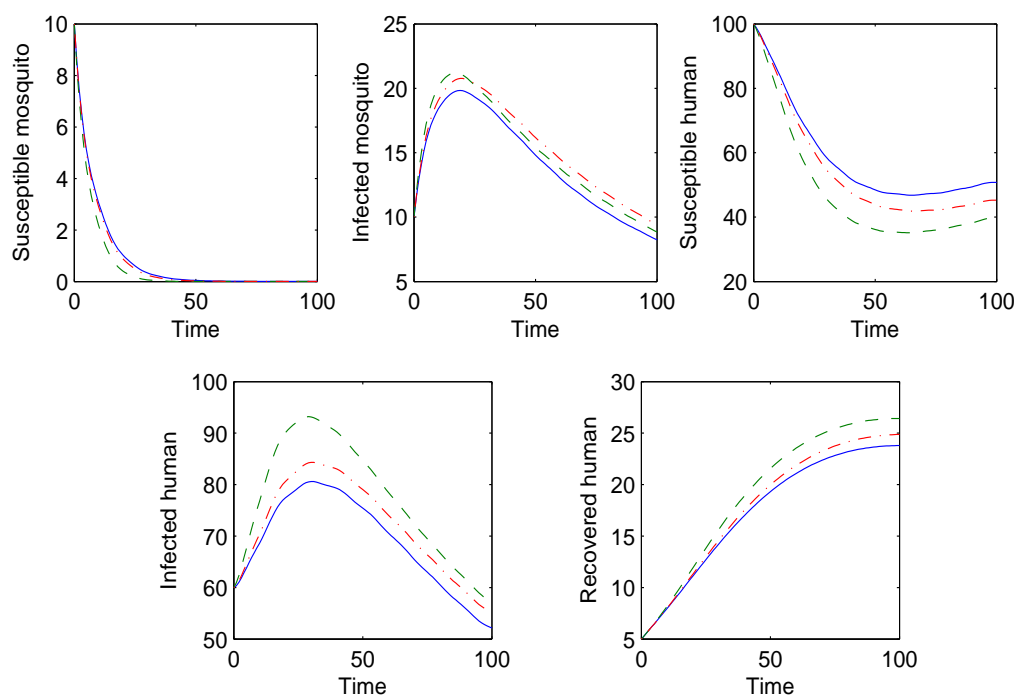

Figure 8.6: Solid line for application of all control, dash line for no control and dash dot line for bed-nets control.

Figure 8.6 shows the solutions of using three controls (i.e., bed-nets, cultivation of mosquito plant and pesticide spray), without any control and only one control such as bed-nets. From this figure, it is observed that when one control such as bed-nets is used then the rate of change of density of infected human is less than that without any control and greater than that all the three controls. The explanation for the susceptible mosquito, infected mosquito, susceptible human, recovered human has been drawn from this figure very easily.

Figure 8.7: Solid line for application of all control, dash line for no control and dash dot line for cultivation of mosquito repellent plant control.

Again, the solutions of using three controls, without any control and only one control such as cultivation of mosquito repellent plant have been shown in Figure 8.7. From this figure, it is seen that when we use only mosquito repellent plant then the rate of change of infected human is less but nearly same as that without using any control. Similarly, the explanation for the other population has been drawn from this figure easily.

Figure 8.8: Solid line for application of all control, dash line for no control and dash dot line for pesticide control.

Also, Figure 8.8 shows the solution of using all controls, without any control and only one control such as pesticide spray. From this figure, it is observed that when we use only pesticide control then the rate of change of infected human is less than that without using any control and greater than that using all three controls. It is also observed that density of infected mosquito using only pesticide control is more reduced than that using all three controls. Changes of other populations have been shown from this figure.

The solutions of using all controls, without any control and only bed-nets and cultivation of mosquito repellent plant control have been shown in Figure 8.9. From this figure, it is seen that if we use only bed-nets and mosquito repellent plant controls together then the rate of change of infected human is less than that without using any control but nearly greater than that for three controls.
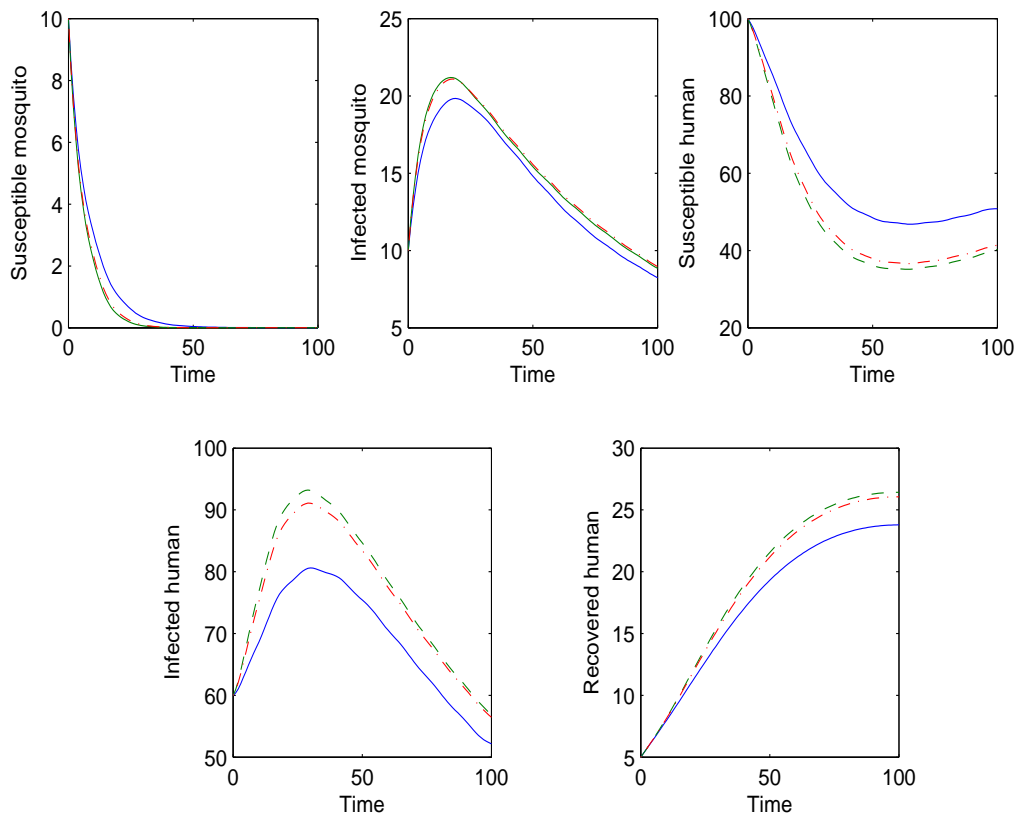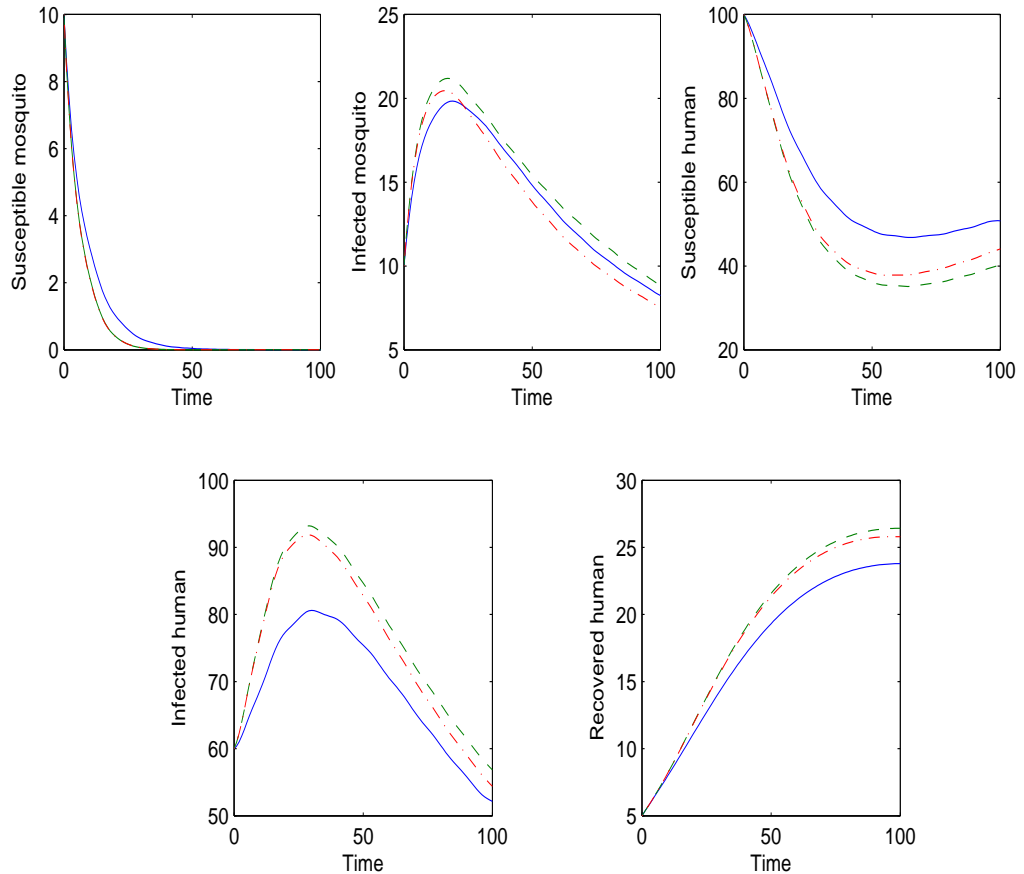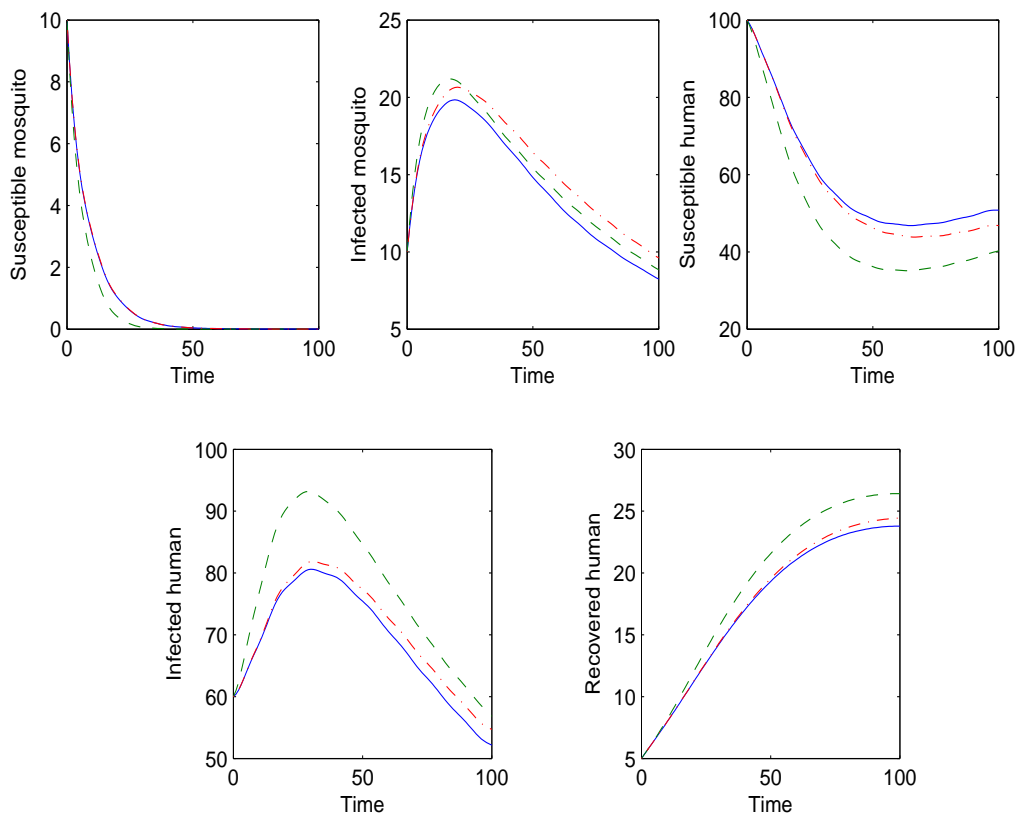
Figure 8.9: Solid line for application of all control, dash line for no control and dash dot line for bed-nets and cultivation of mosquito repellent plant control.

Again, Figure 8.10 represents the solutions of using all controls, without any control and only bed-nets and pesticide control. From this figure, it is observed that if we use only bed-nets and pesticide controls together then the rate of change of infected human is less than that without using any control but nearly greater than that for three controls.

The solutions of using all controls, without any control and only cultivation of mosquito repellent plant have been shown in Figure 8.11. From this figure, it is seen that if we use only mosquito repellent plant and pesticide control then the rate of change of infected human is less than but nearly equal to that without using any control.

Figure 8.12 represents the variation of the three optimal controls. From this figure, it is clear that since we apply all the controls for 100 units of time, all these controls vanishes after this period. Figure 8.13 represents the adjoint variables. According to the theoretical study the adjoint variables becomes zero at the end of the time.
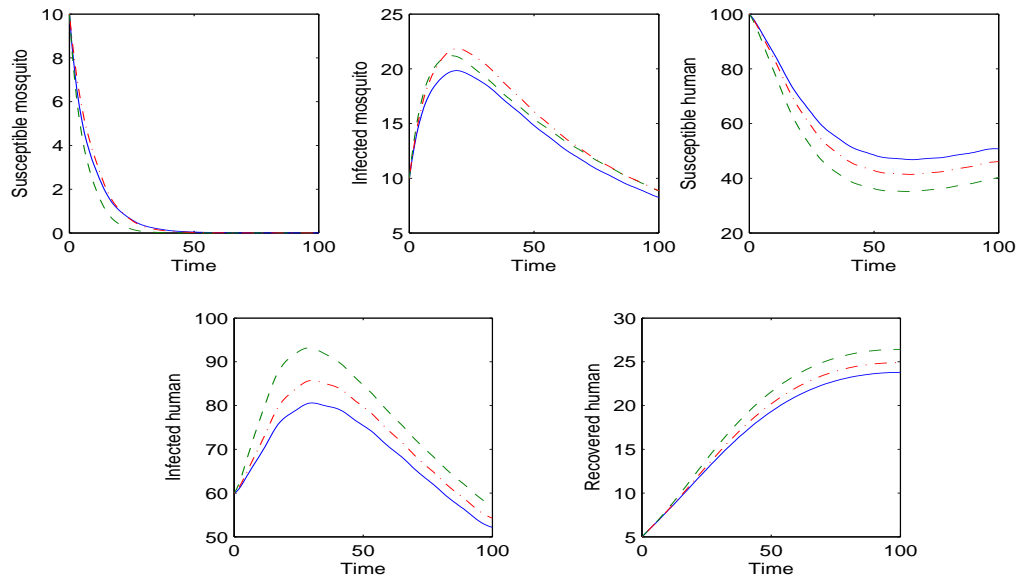
Figure 8.10: Solid line for application of all control, dash line for no control and dash dot line for bed-nets and pesticide control.
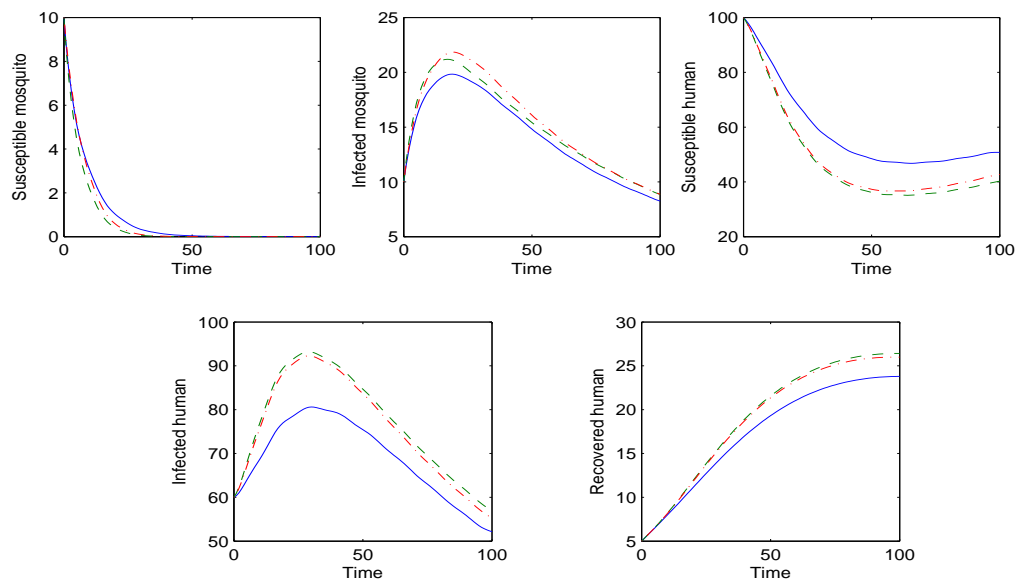

Figure 8.11: Solid line for application of all control, dash line for no control and dash dot line for cultivation of mosquito repellent plant and pesticide control.
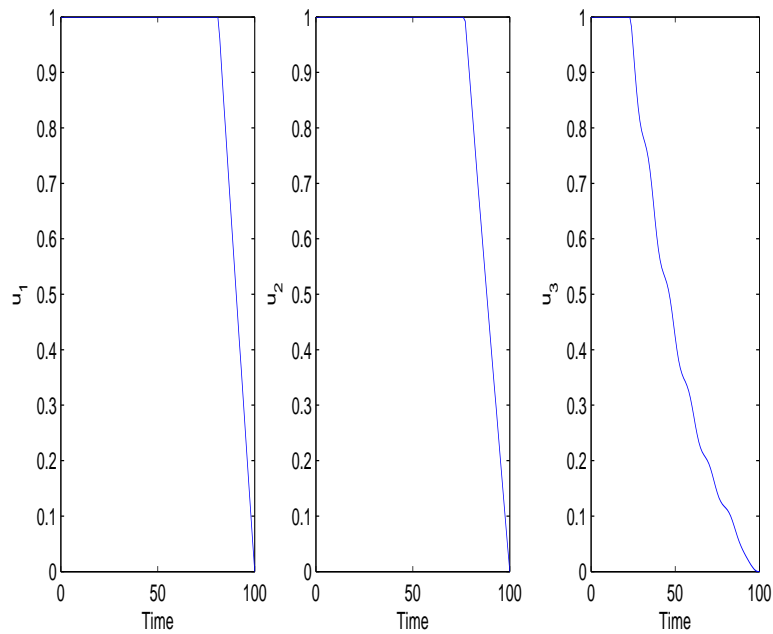
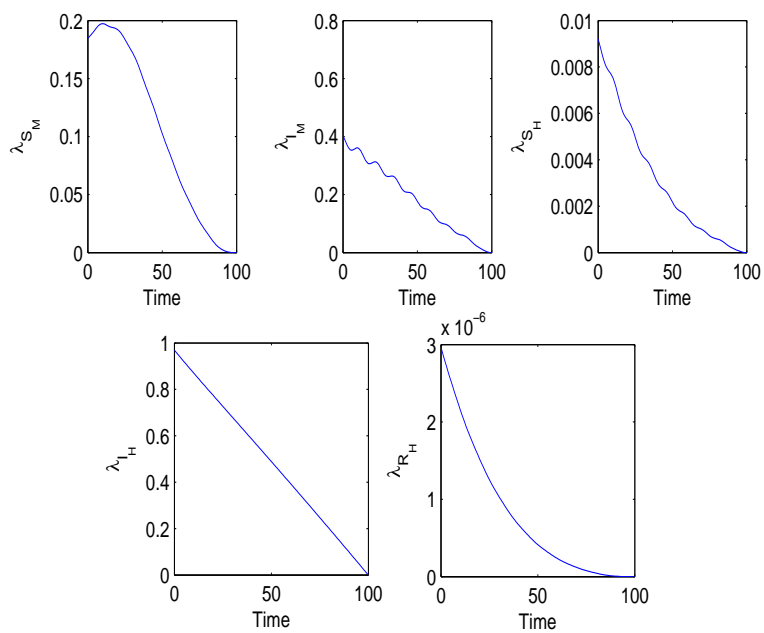Figure 8.12: Three control parameters.

Figure 8.13: Adjoint variables.

8.8   Conclusion

In this chapter, a mathematical model on Malaria disease has been developed considering time and control parameter (bed-nets, plantation of mosquito repellent plant and insecticide spray) dependent transmission rates from human to mosquito and mosquito to human where transmission rates vary periodically. Here, human population has been classified into three subpopulations such as susceptible human, infected human, recovered human and also mosquito population to be classified into two subpopulations such as susceptible mosquito and infected mosquito. Theoretically, it has been proved that the system is free of disease or disease persist under some conditions stated in **Theorem 8.4** and **Theorem 8.5** which are also verified numerically. From the analysis of the model, the following have been drawn.

(i) The solutions of the system (8.1) are bounded if $r^L > \lambda_2 u_3$.

(ii) In autonomous case in the absence of control parameters, our proposed system (8.1) undergoes a Hopf bifurcation with respect to recruitment rate of susceptible human (λ) and the disease transmission rate from infected human to mosquito (α). If we change the recruitment rate of susceptible human and the disease transmission rate from human to mosquito then a Hopf bifurcation occurs. Our study suggests that if the recruitment rate of susceptible human (λ) increases, the the system remains stable up to a threshold value $\lambda = \lambda^*$. But, the system becomes unstable above that threshold. Also, if the disease transmission rate from human to mosquito increases (α), then the autonomous system remains stable up to a threshold value $\alpha = \alpha^*$. But, the system becomes unstable above that threshold.

(iii) Here, different possible combinations of controls have been used and their effectiveness are compared by numerical simulations. From this numerical simulations, it is seen that using of all three controls always gives better results than the using single control and two controls together. From this simulation, it is concluded that if we want to use only one control then bed-nets will be very effective to reduce the infected human. Again, if we want to use two controls together then the use of bed-nets and cultivation of mosquito repellent plant be appropriate strategy to reduce the infected human from Malaria disease.

Gateway from Chapter 8 to Chapter 9
In Chapter 8, we have explored the dynamics of a vector borne fatal disease Malaria. Another important disease to study is Japanese Encephalitis which also makes several thousands of death in every year in India and other parts in the world due to lack of proper investigation and lack of suitable control strategies. The next chapter is devoted to study the dynamics of such system with appropriate control strategies.

Chapter 9

Stability and Bifurcation Analysis of Japanese Encephalitis Model with/without Effects of Some Control Parameters

9.1 Introduction

Japanese Encephalitis (JE) is a vector borne viral disease occurred in South Asia, Southeast Asia, East Asia and Pacific. The disease can cause irreversible neurologic damage. The JE Virus (JEV) is mainly transmitted by the mosquito Culex triataeniorrhynchus, which prefers to bread in the irrigated rice paddies [118]. Wading ardeid water birds (herons and egrets) serve as virus reservoirs, but the virus regularly spills over into the pigs, members of family of equidae (horses and donkeys) and humans [133]. The infected pigs acts as an amplifying host and a domestic pig rearing is an important risk factor in the transmission to humans. Bird's migration might play a role in dispersing JEV. Accidental transportation of vectors, human migration and international travel seem to be of little importance because viremia in humans is usually low and of short duration because humans are dead-end hosts. The transmission cycle of JE has been shown in Figure 9.1. The main pillar of JE control is the use of a live attenuated vaccine for humans [84]. Currently analyzable JE vaccines are relatively safe and effective but a drawback is that multiple doses are required. Effective delivery of the vaccines is poor, rural communities therefore remains a formidable challenge and compliance. The vaccination of pigs represents another potential strategy to control JE, but it is not widely used for two main

reasons. First the high turnover in pig population would require annual vaccination of newborn pigs which would be costly. Second the effectiveness of live attenuated vaccines is decreased in young pigs because of maternal antibodies. Environmental management for vector control such as alternative wetting and drying of rice fields (also known as intermittent irrigation) can substantially reduce vector breeding while saving water, increasing rice yields and reducing methane emission [240]. Environmental management measures are most viable if they are readily integrated into a broader approach of pest management and vector management [20, 64, 70, 142, 212, 213, 221, 235].
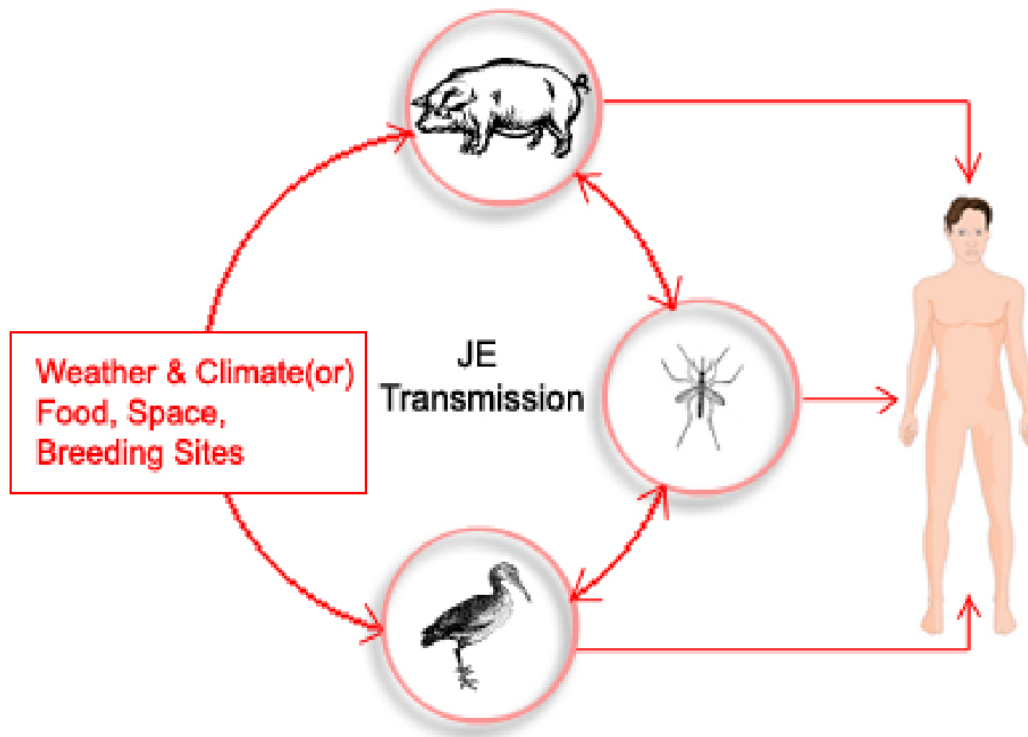
Figure 9.1: Represents the transmission cycle of JE virus.

A very few number of mathematical models of Japanese Encephalitis has been published but till now, the actual dynamics of this disease is unknown to us. At first, Mukhopadhyay et al. [158] studied a mathematical model on Japanese Encephalitis disease in which the stability properties of the model were discussed considering three populations such as vector, reservoir and human in 1993. Thereafter, some research papers on mathematical modelling of Japanese Encephalitis disease such as [159,164,232] have been developed. In India JE is a growing and alarming public health problem. In West Bengal of India since 1973, JE has been almost an annual event in the form of an epidemic or a small outbreak engulfing newer and newer rural areas. The district of Burdwan was the most affected area in West Bengal, probably forming a hyper-endemic zone. In the end of 2014, the

Japanese Encephalitis attack the North Bengal and the north-east part in India. Since the transmission dynamics of Japanese Encephalitis disease is till now unexplored, hence in this epidemic every year many people are died due to the attack of JE virus. So, the study is necessary about the JE virus transmission dynamics and its control strategy.

Though there are several research articles available on Japanese Encephalitis mathematical model but till now there exist some space in the literature which are as:

In most of the existing research papers, the carrying capacity of mosquito population has been considered as constant. But due to environmental changes that should be changed with respect to time. Also, some experimental studies prove that using some suitable control strategies we can restrict the breeding of mosquito and that will be very helpful to control Japanese Encephalitis disease. But in the existing literature no one consider control parameters to control the spread of Japanese Encephalitis disease.

To overcome these spaces we have developed a Japanese Encephalitis transmission model among mosquito, reservoir and human population. Here, a mathematical model on Japanese Encephalitis disease has been constructed by considering time dependent carrying capacity. The mosquito population has been classified into two subpopulations such as susceptible mosquito and infected mosquito. The reservoir population also be classified into three subpopulations such as susceptible reservoir, infected reservoir and recovered reservoir and the human population is divided into three subpopulations such as susceptible human, infected human and recovered human. Here, the existence of equilibrium points and their stabilities in the system have been explored theoretically as well as numerically. Also, it has found the conditions under which (i) the system will be free of disease and (ii) the disease persist uniformly. Then, the effectiveness of all control parameters has been checked numerically. Finally, Hopf bifurcations have been carried out considering constant environmental carrying capacity of mosquito without control parameters.

9.2   Model Formulation [1]

In this chapter, an interaction has been studied mathematically among mosquito, reservoir (pig, horse etc.) and human population in the Japanese Encephalitis disease. Here V_1 and V_2 have been considered as the susceptible and infected mosquito populations. Here Y_1, Y_2 and Y_3 denote the populations indicating the susceptible, infected and recovered reservoir respectively. Similarly, the susceptible, infected and recovered human are indicated by X_1, X_2 and X_3 respectively. Since Japanese Encephalitis (JE) is a vector

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borne viral disease and it is mainly transmitted by mosquito (Culex tritaeniorrhynchus) which prefers to bread in irrigated rice paddies and this atmosphere varies time to time in a year, so the environmental carrying capacity for mosquito denoted by K has been considered as a function of time. In this regard, we have considered $K(t)$ in the following form

$$K(t) = a + b sin(\omega t)$$

where a, b and ω are constants which has been shown in Figure 9.2.
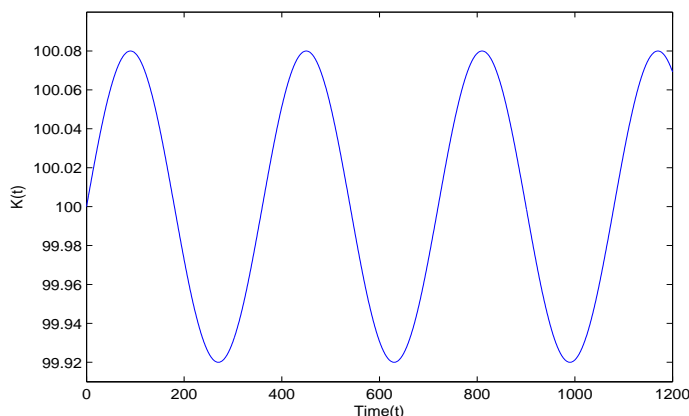

Figure 9.2: Environmental carrying capacity.

Here $r, \eta, \xi, \tau_0, \tau_1, u_1 (0 \leq u_1 \leq 1)$ and $u_2 (0 \leq u_2 \leq 1)$ be the growth rate of the susceptible mosquito population, per capita contact rate of infected reservoirs with the susceptible mosquito population, per capita loss of infectivity of the infected mosquito, effectiveness of intermittent irrigation system control parameter, effectiveness of the insecticide spray control parameter, intermittent irrigation system control parameter i.e., land cover and land use and insecticide spray control respectively. In infected mosquito population, α has been considered as the per capita natural death rate. Here, μ_1, β_1, f_1 and τ_2 denote the per capita natural birth and death rate of the susceptible reservoir, effective per capita contact rate of infective mosquito with reservoir population, per capita loss of immunity of recovered reservoir and effectiveness of the vaccination on the susceptible reservoir population respectively. To resist the replication of the encephalitis virus in the susceptible reservoir population, a vaccination control parameter $u_3 (0 \leq u_3 \leq 1)$ has been considered in this model. Here, γ_1 be the per capita recovery rate of the infected reservoir. Also $\mu_2, \mu_3, \beta_2, f_2, \tau_3$ and $u_4 (0 \leq u_4 \leq 1)$ be the recruitment rate of susceptible human, natural death rate of human, effectiveness of per capita contact rate of infective vectors with the human population, per capita loss of immunity rate of recovered human, effectiveness of the vaccination control parameter and the vaccination control parameter applied to the susceptible human respectively. Also, $\epsilon, \gamma_2, \tau_4$ and $u_5 (0 \leq u_5 \leq 1)$ be the disease related death rate, per capita recovery rate of infected human, effectiveness

of the treatment control parameter and the application of treatment control parameter respectively. Under the above considerations, a set of nonlinear differential equations of encephalitis disease model has been considered as follows:

$$
\begin{aligned}
\frac{dV_1}{dt} &= r(1 - V_1/K(t))V_1 - \eta Y_2 V_1 + \xi V_2 - \tau_0 u_1 V_1 - \tau_1 u_2 V_1 \\[2mm]
\frac{dV_2}{dt} &= \eta Y_2 V_1 - \alpha V_2 - \xi V_2 - \tau_1 u_2 V_2 \\[2mm]
\frac{dY_1}{dt} &= \mu_1 N_1 - \mu_1 Y_1 - \beta_1 V_2 Y_1 + f_1 Y_3 - \tau_2 u_3 Y_1 \\[2mm]
\frac{dY_2}{dt} &= \beta_1 V_2 Y_1 - \mu_1 Y_2 - \gamma_1 Y_2 \\[2mm]
\frac{dY_3}{dt} &= \gamma_1 Y_2 - \mu_1 Y_3 - f_1 Y_3 + \tau_2 u_3 Y_1 \\[2mm]
\frac{dX_1}{dt} &= \mu_2 - \mu_3 X_1 - \beta_2 V_2 X_1 + f_2 X_3 - \tau_3 u_4 X_1 \\[2mm]
\frac{dX_2}{dt} &= \beta_2 V_2 X_1 - \mu_3 X_2 - \epsilon X_2 - \gamma_2 X_2 - \tau_4 u_5 X_2 \\[2mm]
\frac{dX_3}{dt} &= \gamma_2 X_2 - \mu_3 X_3 - f_2 X_3 + \tau_3 u_4 X_1 + \tau_4 u_5 X_2
\end{aligned}
\right\}
\tag{9.1}
$$

subject to the initial conditions $V_1(0) \geq 0, V_2(0) \geq 0, Y_1(0) \geq 0, Y_2(0) \geq 0,$ $Y_3(0) \geq 0, X_1(0) \geq 0, X_2(0) \geq 0, X_3(0) \geq 0$ where $Y_1 + Y_2 + Y_3 = N_1$(constant).

9.3 Equilibrium Points and Basic Reproduction Number

Before derivation of the basic reproduction number of the system (9.1), it is necessary to show that the solution of the system (9.1) is bounded which is shown by the following theorem.

Theorem 9.1 All the solutions $(V_1(t), V_2(t), Y_1(t), Y_2(t), Y_3(t), X_1(t), X_2(t), X_3(t))$ are bounded, provided that $min\{\tau_0 u_1 + \tau_1 u_2, \alpha + \tau_1 u_2\} > 0$.

Proof. From the 3rd, 4th and 5th equations of system (9.1), it is obtained that

$$\frac{d}{dt}(Y_1 + Y_2 + Y_3) = \mu_1 N_1 - \mu_1(Y_1 + Y_2 + Y_3)$$

$$\frac{d}{dt}(Y_1 + Y_2 + Y_3) + \mu_1(Y_1 + Y_2 + Y_3) = \mu_1 N_1$$

Then, solving above we have

$$Y_1 + Y_2 + Y_3 = N_1 + C_1 e^{-\mu_1 t}$$

As $t \to \infty$ it is obtained that

$$Y_1 + Y_2 + Y_3 = N_1$$

So, it is obvious that $Y_1 \leq N_1, Y_2 \leq N_1$ and $Y_3 \leq N_1$ for all t.
Also, from the 6th, 7th and 8th equations of system (9.1), it is obtained that

$$\frac{dX_1}{dt} + \frac{dX_2}{dt} + \frac{dX_3}{dt} = \mu_2 - \mu_3(X_1 + X_2 + X_3) - \epsilon X_2$$

$$i.e., \frac{d(X_1 + X_2 + X_3)}{dt} + \mu_3(X_1 + X_2 + X_3) \leq \mu_2$$

$$i.e., X_1 + X_2 + X_3 \leq \frac{\mu_2}{\mu_3} + C_2 e^{-\mu_3 t}$$

As $t \to \infty$, it is found that

$$X_1 + X_2 + X_3 \leq \frac{\mu_2}{\mu_3}$$

which implies that X_1, X_2 and X_3 are bounded for $t \geq 0$.
Let us define a function W such that

$$W = V_1 + V_2 \tag{9.2}$$

Taking time derivative of above equation (9.2), it is obtained that

$$\frac{dW}{dt} = r(1 - \frac{V_1}{K(t)})V_1 - \tau_0 u_1 V_1 - \tau_1 u_2 V_1 - \alpha V_2 - \tau_1 u_2 V_2$$

After adding QW to the both sides of the above equation, it is obtained that

$$\frac{dW}{dt} + QW = r(1 - \frac{V_1}{K(t)})V_1 - \tau_0 u_1 V_1 - \tau_1 u_2 V_1 - \alpha V_2 - \tau_1 u_2 V_2 + Q(V_1 + V_2)$$

$$= r(1 - \frac{V_1}{K(t)})V_1 + (Q - \tau_0 u_1 - \tau_1 u_2)V_1 + (Q - (\alpha + \tau_1 u_2))V_2 \tag{9.3}$$

where Q has been chosen as a positive quantity in the following manner:

$$Q = min\{\tau_0 u_1 + \tau_1 u_2, \alpha + \tau_1 u_2\}$$

Now, according to our problem definition, the carrying capacity of mosquito population is given by

$$K(t) = a + bsin(\omega t)$$
$$i.e., \quad |K(t)| = |a + bsin(\omega t)|$$
$$i.e., \quad |K(t)| \leq |a| + |bsin(\omega t)|$$
$$i.e., \quad |K(t)| \leq |a| + |b|, \text{ since } |sin(\omega t)| \leq 1$$
$$i.e., \quad |K(t)| \leq K_1 \qquad \text{where } K_1 = |a| + |b|$$

Using these the above equation (9.3) can be written as

$$\frac{dW}{dt} + QW \quad \leq r(1 - \frac{V_1}{K(t)})V_1$$

$$\leq r(1 - \frac{V_1}{K_1})V_1 \text{ since } |K(t)| \leq K_1$$

$$\leq \frac{rK_1}{4} \text{ where } max\{r(1 - \frac{V_1}{K_1})V_1\} = \frac{rK_1}{4}$$

Then solving above it is obtained that

$$W \leq \frac{rK_1}{4Q} + C_3 e^{-Qt} \tag{9.4}$$

where C_3 is an integrating constant.

Now, when t tends to ∞, then for $Q > 0$ the equation (8.4) reduces to the following

$$W \leq \frac{rK_1}{4Q}$$

$$i.e., V_1 + V_2 \leq \frac{rK_1}{4Q}$$

which implies that V_1 and V_2 are bounded for $t \geq 0$.
Hence all the solutions of the system (9.1) are bounded.

Now, the system (9.1) has the following disease free equilibrium point
$E_0 = (V_1^0, 0, Y_1^0, 0, Y_3^0, X_1^0, 0, X_3^0)$, where $V_1^0 = (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r})K(t)$,
$Y_1^0 = \frac{N_1(\mu_1 + f_1)}{(\mu_1 + f_1 + \tau_2 u_3)}, Y_3^0 = \frac{\tau_2 u_3 N_1}{(\mu_1 + f_1 + \tau_2 u_3)}, X_1^0 = \frac{\mu_2(\mu_3 + f_2)}{\mu_3(\mu_3 + f_2 + \tau_3 u_4)}, X_3^0 = \frac{\mu_2 \tau_3 u_4}{\mu_3(\mu_3 + f_2 + \tau_3 u_4)}$
and one endemic equilibrium point $E^* = (V_1^*, V_2^*, Y_1^*, Y_2^*, Y_3^*, X_1^*, X_2^*, X_3^*)$

where $V_1^*, V_2^*, Y_1^*, Y_2^*, Y_3^*, X_1^*, X_2^*$ and X_3^* satisfies

$$V_2^* = \frac{\eta Y_2^* V_1^*}{\alpha + \xi + \tau_1 u_2}, Y_2^* = \frac{\beta_1 V_2^* Y_1^*}{\mu_1 + \gamma_1}, X_2^* = \frac{\beta_2 V_2^* X_1^*}{\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5}, Y_3^* = \frac{\gamma_1 Y_2^* + \tau_2 u_3 Y_1^*}{\mu_1 + f_1},$$

$$X_3^* = \frac{\gamma_2 X_2^* + \tau_3 u_4 X_1^* + \tau_4 u_5 X_2^*}{\mu_3 + f_2},$$

$$r(1 - V_1^*/K(t))V_1^* - \eta Y_2^* V_1^* + \xi V_2^* - \tau_0 u_1 V_1^* - \tau_1 u_2 V_1^* = 0,$$

$$\mu_1 N_1 - \mu_1 Y_1^* - \beta_1 V_2^* Y_1^* + f_1 Y_3^* - \tau_2 u_3 Y_1^* = 0 \text{ and}$$

$$\mu_2 - \mu_3 X_1^* - \beta_2 V_2^* X_1^* + f_2 X_3^* - \tau_3 u_4 X_1^* = 0$$

In epidemiology, the basic reproduction number R_0 is defined as the expected number of secondary infections that occur when one infective is introduced into a completely susceptible host population. First, we enumerate the compartments in our model from left to right i.e., susceptible mosquito (V_1)=Compartment1, infected mosquito (V_2)=Compartment2 and so on. Then by this method the new infection generation terms and the remaining transition terms denoted by two matrices F and V are as follows:

$$F = \left(\frac{\partial F_i(x)}{\partial x_j} \right)_{x=x_0} \text{ and } V = \left(\frac{\partial V_i(x)}{\partial x_j} \right)_{x=x_0}$$

where $F_i(x)$ denote the rate of appearance of new infection in compartment i and $V_i(x)$ is the net transfer rate (other than infection) of compartment i. In our model there are three stages for transmission of infection through (i) infected mosquito, (ii) infected reservoir and (iii) infected human. So, here $x = (x_2, x_4, x_7)$ where x_2 denotes infected mosquito (V_2), x_4 denotes infected reservoir (Y_2) and x_7 denotes infected human (X_2). The net transfer rate is given by $V_i = V_i^- - V_i^+$, where V_i^- is the rate of transfer of individuals out of compartment i, and V_i^+ is the rate of transfer of individuals into compartment i by means other than infection.

Then, the two matrix can be evaluated as follows

$$F = \begin{pmatrix} 0 & \eta V_1^0 & 0 \\ \beta_1 Y_1^0 & 0 & 0 \\ \beta_2 X_1^0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} (\alpha + \xi + \tau_1 u_2) & 0 & 0 \\ 0 & (\mu_1 + \gamma_1) & 0 \\ 0 & 0 & (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) \end{pmatrix}$$

Then, we evaluate the matrix

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\eta V_1^0}{\mu_1 + \gamma} & 0 \\ \frac{\beta_1 Y_1^0}{\alpha + \xi + \tau_1 u_2} & 0 & 0 \\ \frac{\beta_2 X_1^0}{\alpha + \xi + \tau_1 u_2} & 0 & 0 \end{pmatrix}$$

The basic reproduction number is the maximum eigenvalue [59] of the matrix FV^{-1} and it is evaluated as follows:

$$R_0 = \sqrt{\frac{\beta_1 \eta V_1^0 Y_1^0}{(\alpha + \xi + \tau_1 u_2)(\gamma_1 + \mu_1)}} \tag{9.5}$$

Lemma 9.1 The average value of R_0 in any time interval $[b_1, b_2]$ is denoted by R_0^1 and is defined by

$$R_0^1 = \sqrt{\frac{\beta_1 \eta (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r}) N_1 (\mu_1 + f_1)[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}]}{(\mu_1 + f_1 + \tau_2 u_3)(\alpha + \xi + \tau_1 u_2)(\gamma_1 + \mu_1)}}$$

Proof. The average value of $R_0^2(t)$ for the interval $b_1 \leq t \leq b_2$ can be computed as

$$\frac{1}{b_2 - b_1} \int_{b_1}^{b_2} R_0^2 dt = \frac{\beta_1 \eta Y_1^0 (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r})}{(\alpha + \xi + \tau_1 u_2)(\gamma_1 + \mu_1)(b_2 - b_1)} \int_{b_1}^{b_2} K(t) dt \qquad (9.6)$$

Now,

$$\int_{b_1}^{b_2} K(t) dt = \int_{b_1}^{b_2} (a + bsin(\omega t)) dt$$

$$= a(b_2 - b_1) - \frac{b}{\omega}(cos(\omega b_2) - cos(\omega b_1)) \qquad (9.7)$$

Using equation (9.7) in (9.6), it is obtained that

$$\frac{1}{b_2 - b_1} \int_{b_1}^{b_2} R_0^2 dt = \frac{\beta_1 \eta Y_1^0 (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r})}{(\alpha + \xi + \tau_1 u_2)(\gamma_1 + \mu_1)}[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}]$$

Then the average value of R_0 in any time interval $[b_1, b_2]$ is

$$R_0^1 = \sqrt{\frac{\beta_1 \eta (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r}) N_1 (\mu_1 + f_1)[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}]}{(\mu_1 + f_1 + \tau_2 u_3)(\alpha + \xi + \tau_1 u_2)(\gamma_1 + \mu_1)}} \qquad (9.8)$$

Hence the proof.

Hence from this expression, **Lemma 9.2** can be drawn as

Lemma 9.2 Three control parameters u_1, u_2 and u_3 have effects on the basic reproduction number R_0^1.

Lemma 9.3 The Basic reproduction number R_0^1 is a decreasing function with respect to the parameters u_1, u_2 and u_3 provided that $a > \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}, u_1 < \frac{\alpha + \xi + r}{\tau_0}$ and $\tau_0 u_1 + \tau_1 u_2 < r$.

Proof. From (9.8) differentiating R_0^1 with respect to u_1, u_2 and u_3 we have

$$\frac{d(R_0^1)^2}{du_1} = -\frac{\beta_1 \eta \tau_0 N_1 (\mu_1 + f_1)\left[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}\right]}{r(\alpha + \xi + \tau_1 u_2)(\mu_1 + \gamma_1)(\mu_1 + f_1 + \tau_2 u_3)} \qquad (9.9)$$

$$\frac{d(R_0^1)^2}{du_2} = -\frac{\tau_1 (\alpha + \xi + r - \tau_0 u_1)\beta_1 \eta (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r}) N_1 (\mu_1 + f_1)[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}]}{(r - \tau_0 u_1 - \tau_1 u_2)(\alpha + \xi + \tau_1 u_2)^2 (\mu_1 + f_1 + \tau_2 u_3)(\mu_1 + \gamma_1)} \qquad (9.10)$$

$$\frac{d(R_0^1)^2}{du_3} = -\frac{\beta_1 \eta \tau_2 (1 - \frac{(\tau_0 u_1 + \tau_1 u_2)}{r}) N_1 (\mu_1 + f_1)\left[a - \frac{b(cos(\omega b_2) - cos(\omega b_1))}{\omega (b_2 - b_1)}\right]}{(\alpha + \xi + \tau_1 u_2)(\mu_1 + \gamma_1)(\mu_1 + f_1 + \tau_2 u_3)^2} \qquad (9.11)$$

Now, from equation (9.9) it is observed that $\frac{d(R_0^1)^2}{du_1}$ will be negative if $a > \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}$. Since all the parameters are positive and from equation (9.10) it is also observed that $\frac{d(R_0^1)^2}{du_2}$ will be negative if $u_1 < \frac{\alpha+\xi+r}{\tau_0}$, $\tau_0 u_1 + \tau_1 u_2 < r$ and $a > \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}$. Similarly, from equation (9.11), $\frac{d(R_0^1)^2}{du_3}$ will be a deceasing function with respect to u_3 if $\tau_0 u_1 + \tau_1 u_2 < r$ and $a > \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}$.

Lemma 9.4 The value of R_0^1 will be less than one with the application of single control parameter of u_1, u_2 and u_3 provided that $\frac{r}{\tau_0}\left[1 - \frac{(\alpha+\xi)(\mu_1+\gamma_1)\omega(b_2-b_1)}{\beta_1\eta N_1(cos(\omega b_2)-cos(\omega b_1))}\right] < u_1 \le 1$,

$\frac{\beta_1\eta N_1\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right] - (\alpha+\xi)(\mu_1+\gamma_1)}{\left[\tau_1(\mu_1+\gamma_1) + \frac{\beta_1\eta\tau_1 N_1}{r}\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]\right]} < u_2 \le 1$,

$\frac{\beta_1\eta N_1(\mu_1+f_1)\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right] - (\mu_1+f_1)(\mu_1+\gamma_1)(\alpha+\xi)}{\tau_2(\alpha+\xi)(\mu_1+\gamma_1)} < u_3 \le 1$.

Proof. At first, we consider the application of single control parameter u_1 to the system (9.1) to reduce the encephalitis disease. So, putting $u_2 = u_3 = 0$ in equation (9.8) it is obtained that

$$(R_0^1)^2 = \frac{\beta_1\eta(1 - \frac{\tau_0 u_1}{r})N_1[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}]}{(\alpha+\xi)(\gamma_1+\mu_1)}$$

Now, the value of $(R_0^1)^2$ will be less than one if

$$\frac{\beta_1\eta(1 - \frac{\tau_0 u_1}{r})N_1[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}]}{(\alpha+\xi)(\gamma_1+\mu_1)} < 1$$

$$(1 - \frac{\tau_0 u_1}{r}) < \frac{(\alpha+\xi)(\gamma_1+\mu_1)}{\beta_1\eta N_1[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}]}$$

$$u_1 > \frac{r}{\tau_0}\left[1 - \frac{(\alpha+\xi)(\mu_1+\gamma_1)\omega(b_2-b_1)}{\beta_1\eta N_1(cos(\omega b_2)-cos(\omega b_1))}\right]$$

$$\frac{r}{\tau_0}\left[1 - \frac{(\alpha+\xi)(\mu_1+\gamma_1)\omega(b_2-b_1)}{\beta_1\eta N_1(cos(\omega b_2)-cos(\omega b_1))}\right] < u_1 \le 1, \text{ since } 0 \le u_1 \le 1$$

In the similar way, it can be proved that

$$\frac{\beta_1\eta N_1\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right] - (\alpha+\xi)(\mu_1+\gamma_1)}{\left[\tau_1(\mu_1+\gamma_1) + \frac{\beta_1\eta\tau_1 N_1}{r}\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]\right]} \le u_2 \le 1$$

and

$$\frac{\beta_1\eta N_1(\mu_1+f_1)\left[a - \frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right] - (\mu_1+f_1)(\mu_1+\gamma_1)(\alpha+\xi)}{\tau_2(\alpha+\xi)(\mu_1+\gamma_1)} < u_3 \le 1$$

So it is concluded that system (9.1) can be disease free either by application of u_1 or u_2 or u_3 under the above conditions. But if for some parametric values the conditions are not full-filled, then system cannot be disease free through application of only one control. In that case two or more controls may be used simultaneously which is shown in **Lemma 9.5**.

Lemma 9.5 The value of R_0^1 will be less than one with the application of two or three control parameter simultaneously $(u_1, u_2), (u_2, u_3), (u_1, u_3)$ and (u_1, u_2, u_3) provided that

$$u_2 > \frac{\beta_1\eta(1-\frac{\tau_0 u_1}{r})N_1\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]-(\alpha+\xi)(\mu_1+\gamma_1)}{\tau_1\left[(\mu_1+\gamma_1)+\frac{\beta_1\eta}{r}N_1\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]\right]},$$

$$u_2 > \frac{\beta_1\eta N_1(\mu_1+f_1)\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]-(\alpha+\xi)(\mu_1+\gamma_1)(\mu_1+f_1+\tau_2 u_3)}{\tau_1\left[(\mu_1+\gamma_1)(\mu_1+f_1+\tau_2 u_3)+\frac{\beta_1\eta}{r}N_1(\mu_1+f_1)\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]\right]},$$

$$u_1 > \frac{r}{\tau_0}\left[1-\frac{(\mu_1+f_1+\tau_2 u_3)(\alpha+\xi)(\mu_1+\gamma_1)}{\beta_1\eta N_1(\mu_1+f_1)\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]}\right] \text{ and}$$

$$u_3 > \frac{\beta_1\eta\left(1-\frac{(\tau_0 u_1+\tau_1 u_2)}{r}\right)N_1(\mu_1+f_1)\left[a-\frac{b(cos(\omega b_2)-cos(\omega b_1))}{\omega(b_2-b_1)}\right]-(\mu_1+f_1)(\mu_1+\gamma_1)(\alpha+\xi+\tau_1 u_2)}{\tau_2(\alpha+\xi+\tau_1 u_2)(\mu_1+\gamma_1)}.$$

Proof. Proof is similar as **Lemma 9.4**.

9.4 Threshold Dynamics

In this section, it is shown that if $R_0 < 1$ then the disease free equilibrium point $(V_1^0, 0, Y_1^0, 0, Y_3^0, X_1^0, 0, X_3^0)$ is locally asymptotically stable and hence the disease dies out. Next, we show that if $R_0 > 1$, then V_2, Y_2 and X_2 uniformly persist and hence the disease also persists. Consequently, the basic reproduction number R_0 is the threshold parameter between the extinction and the uniform persistence of the disease.

Theorem 9.2 The system (9.1) around $E_0(V_1^0, 0, Y_1^0, 0, Y_3^0, X_1^0, 0, X_3^0)$ is locally asymptotically stable provided that $(\mu_1+\gamma_1)(\alpha+\xi+\tau_1 u_2) > \eta V_1^0\beta_1 Y_1^0$ and $(\mu_1+\gamma_1+\mu_3+\epsilon+\gamma_2+\tau_4 u_5)[(\mu_1+\gamma_1)(\mu_3+\epsilon+\gamma_2+\tau_4 u_5)+(\alpha+\xi+\tau_1 u_2)(\mu_3+\epsilon+\gamma_2+\tau_4 u_5+\alpha+\xi+\tau_1 u_2+\mu_1+\gamma_1)] > \eta V_1^0\beta_1 Y_1^0(\alpha+\xi+\tau_1 u_2+\mu_1+\gamma_1)$.

Proof. From the second, fourth and sixth equation of system (9.1), we have

$$\frac{dx}{dt} = Bx$$

where $x = (V_2, Y_2, X_2)^t$ and

$$B = \begin{pmatrix} -(\alpha+\xi+\tau_1 u_2) & \eta V_1^0 & 0 \\ \beta_1 Y_1^0 & -(\mu_1+\gamma_1) & 0 \\ \beta_2 X_1^0 & 0 & -(\mu_3+\epsilon+\gamma_2+\tau_4 u_5) \end{pmatrix}$$

Then, the characteristic equation of B is

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

where $a_1 = (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5 + \alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1)$, $a_2 = (\mu_1 + \gamma_1)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) + (\alpha + \xi + \tau_1 u_2)(\mu_1 + \gamma_1 + \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - \eta V_1^0 \beta_1 Y_1^0$ and $a_3 = (\mu_1 + \gamma_1)(\alpha + \xi + \tau_1 u_2)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - \eta V_1^0 \beta_1 Y_1^0(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$.

Now, all the roots of the above characteristic equation be negative for real roots and have negative real parts for complex roots

$$\text{if } a_1 > 0, a_3 > 0 \text{ and } a_1 a_2 - a_3 > 0$$

$$i.e., \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5 + \alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1 > 0$$

$$i.e., (\mu_1 + \gamma_1)(\alpha + \xi + \tau_1 u_2)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) > \eta V_1^0 \beta_1 Y_1^0(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$$

$$i.e., \frac{\eta V_1^0 \beta_1 Y_1^0}{(\mu_1 + \gamma_1)(\alpha + \xi + \tau_1 u_2)} < 1$$

$$i.e., R_0 < 1$$

and

$$(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5 + \alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1)\Big\{(\mu_1 + \gamma_1)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$$

$$+(\alpha + \xi + \tau_1 u_2)(\mu_1 + \gamma_1 + \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - \eta V_1^0 \beta_1 Y_1^0\Big\} > \Big\{(\mu_1 + \gamma_1)(\alpha + \xi + \tau_1 u_2)$$

$$-\eta V_1^0 \beta_1 Y_1^0\Big\}(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$$

$$i.e., (\mu_1 + \gamma_1 + \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)\Big[(\mu_1 + \gamma_1)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$$

$$+(\alpha + \xi + \tau_1 u_2)(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5 + \alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1)\Big]$$

$$> \eta V_1^0 \beta_1 Y_1^0(\alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1)$$

Therefore, if the above relation holds then the value of R_0 will be less than one and the system will be free of disease.

Hence the theorem.

Theorem 8.3 The disease of system (9.1) uniformly persistent i.e., $V_2 > 0, Y_2 > 0$ and $X_2 > 0$ provided that

$$\lim_{t \to \infty} \inf \Big(V_1(t), V_2(t), Y_1(t), Y_2(t), Y_3(t), X_1(t), X_2(t), X_3(t)\Big) \geq c$$

for a positive constant c.

Proof. Similar to [76, 236].

9.5 Global Stability Analysis

Here, we shortly describe the general method developed by Li and Muldowney [131] for a global stability of a nonlinear system around its interior equilibrium point. Here, we consider an autonomous dynamical system:

$$\dot{x} = f(x) \tag{9.12}$$

where $f : D \to R^n, D \subset R^n$ be an open set and simply connected and $f \in C^1(D)$ which is the space of continuously differentiable function on domain D. Each solution $x(t)$ of this differential equation (9.12) is uniquely determined by its initial value $x(0) = x_0$. Let x^* be an equilibrium point of (9.12). Then we can say that x^* is said to be globally asymptotically stable in D if it is locally stable and all trajectories in D converge to x^*. The main interest in this section is to derive a condition for global stability of an interior equilibrium point x^* in the proposed model. Now, for the proposed problem we have assumed the following conditions

(*i*) D is simply connected;

(*ii*) there is a compact absorbing set $K \subset D$;

(*iii*) x^* is the only equilibrium of (9.12) in D.

Under the above conditions, an equilibrium point x^* is globally asymptotically stable in D by geometric approach [131], provided that

$$\limsup_{t \to \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0))) ds < 0 \tag{9.13}$$

where $\mu(B)$ is calculated as follows:

Now, a matrix $P(x)$ is choosen in such a way that it will be a nonsingular $\binom{n}{2} \times \binom{n}{2}$ matrix valued function $x \to P(x)$ defined in C^1 on D. It is considered that

$$B = P_f P^{-1} + P J^{[2]} P^{-1}$$

where the matrix P_f is $(P_{ij}(x))_f$ is given by $(P_{ij}(x))_f = \nabla P_{ij}.f(x)$.

The jacobian matrix $J^{[2]}$ is the second additive compound matrix of $\binom{n}{2} \times \binom{n}{2}$ order for a $n \times n$ the jacobian matrix J defined by $J = (J_{ij})_n = \left(\frac{\partial f_i}{\partial x_j}\right)_n$. Therefore, in our model for $n = 3$, it is as follows

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

Now, it is considered that the Lozinskii measure μ of the matrix B with respect to a matrix norm $|.|$ in R^N where $N = \binom{n}{2}$, is defined by

$$\mu(B) = \lim_{h \to 0} \frac{|I + hB| - 1}{h}$$

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By using the above discussion, we want to derive the conditions under which the proposed model system (9.1) is globally stable around its interior equilibrium point x^* which is here (E^*). Firstly, we consider the subsystem of (9.1) as follows

$$
\left.\begin{aligned}
\frac{dV_2}{dt} &= \eta Y_2 V_1 - \alpha V_2 - \xi V_2 - \tau_1 u_2 V_2 \\[2mm]
\frac{dY_2}{dt} &= \beta_1 V_2 Y_1 - \mu_1 Y_2 - \gamma_1 Y_2 \\[2mm]
\frac{dX_2}{dt} &= \beta_2 V_2 X_1 - \mu_3 X_2 - \epsilon X_2 - \gamma_2 X_2 - \tau_4 u_5 X_2
\end{aligned}\right\} \tag{9.14}
$$

Then the Jacobian matrix for the system (9.14) is

$$
J = \begin{pmatrix}
-\alpha - \xi - \tau_1 u_2 & \eta V_1 & 0 \\
\beta_1 Y_1 & -(\mu_1 + \gamma_1) & 0 \\
\beta_2 X_1 & 0 & -(\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)
\end{pmatrix}
$$

Now, its second additive compound matrix $J^{[2]}$ is given by

$$
J^{[2]} = \begin{pmatrix}
-m & 0 & 0 \\
0 & -n & \eta V_1 \\
\beta_2 X_1 & \beta_1 Y_1 & -k
\end{pmatrix}
$$

where $m = (\alpha + \xi + \tau_1 u_2 + \mu_1 + \gamma_1), n = (\alpha + \xi + \tau_1 u_2 + \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$ and $k = (\mu_1 + \gamma_1 + \mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$.

Next, we choose a nonsingular matrix valued function $P(x)$ in the following way

$$
P(x) = P(V_2, Y_2, X_2) = diag\left(\frac{V_2}{X_2}, \frac{V_2}{X_2}, \frac{V_2}{X_2}\right) \text{ where } x = (V_2, Y_2, X_2)^t
$$

The matrix valued function P_f can be evaluated as

$$
P_f = \frac{\partial P}{\partial x} = diag\left(\frac{\dot{V_2}}{X_2} - \frac{V_2}{X_2^2}\dot{X_2}, \frac{\dot{V_2}}{X_2} - \frac{V_2}{X_2^2}\dot{X_2}, \frac{\dot{V_2}}{X_2} - \frac{V_2}{X_2^2}\dot{X_2}\right)
$$

Now, it follows that $P_f P^{-1} = diag\{\frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2}, \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2}, \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2}\}$ and $PJ^{[2]}P^{-1} = J^{[2]}$ so that

$$
B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}
$$

where

$$
B_{11} = \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} - m, B_{12} = \begin{bmatrix} 0 & 0 \end{bmatrix}, B_{21} = \begin{bmatrix} 0 & \beta_2 X_1 \end{bmatrix}^t
$$

$$\text{and } B_{22} = \begin{pmatrix} \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} - n & \eta V_1 \\ \beta_1 Y_1 & \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} - k \end{pmatrix}$$

Now, we consider the norm in R^3 as

$$|(u, v, w)| = max\{|u|, |v| + |w|\}$$

and

$$\mu(B) \leq \sup\{g_1, g_2\} = \sup\{\mu_1(B_{11}) + |B_{12}|, \mu_1(B_{22}) + |B_{21}|\} \tag{9.15}$$

where $|B_{21}|, |B_{12}|$ are matrix norms with respect to the L^1 vector norm and μ_1 denotes the Lozinskii measure with respect to the L^1 norm. Now,

$$\mu_1(B_{11}) = \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} - m, |B_{12}| = max\{0, 0\} = 0, |B_{21}| = \beta_2 X_1$$

$$\mu_1(B_{22}) = \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} + max\{-n + \beta_1 Y_1, -k + \eta V_1\}$$

The general expression of g_1 and g_2 for the above system are thus

$$g_1 = \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} - m$$

$$g_2 = \frac{\dot{V_2}}{V_2} - \frac{\dot{X_2}}{X_2} + \beta_2 X_1 + max\{-n + \beta_1 Y_1, -k + \eta V_1\}$$

Again, from the system (9.1), it is obtained that

$$\dot{X_2} = \beta_2 V_2 X_1 - (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)X_2$$

$$\frac{\dot{X_2}}{X_2} = \frac{\beta_2 V_2 X_1}{X_2} - (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5)$$

Therefore using these in above it is obtained that

$$g_1 = \frac{\dot{V_2}}{V_2} - \frac{\beta_2 V_2 X_1}{X_2} + (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - m$$

$$g_2 = \frac{\dot{V_2}}{V_2} - \frac{\beta_2 V_2 X_1}{X_2} + (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) + \beta_2 X_1 + max\{-n + \beta_1 Y_1, -k + \eta V_1\}$$

Therefore, from equation (9.15) we have

$$\begin{aligned} \mu(B) \quad \leq \quad & \frac{\dot{V_2}}{V_2} - \frac{\beta_2 V_2 X_1}{X_2} + (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) \\ + \quad & max\{\beta_2 X_1 + max\{-n + \beta_1 Y_1, -k + \eta V_1\}, -m\} \end{aligned}$$

If the disease is uniformly persistence for $R_0 > 1$ then

$$\inf\left\{\lim_{t\to\infty}(V_1(t), V_2(t), Y_1(t), Y_2(t), Y_3(t), X_1(t), X_2(t), X_3(t))\right\} \geq c$$

then the above equation reduces to the following form

$$\mu(B) \leq \frac{\dot{V_2}}{V_2} - \beta_2 c + (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) + max\left\{\beta_2 c + max\{-n + \beta_1 c, -k + \eta c\}, -m\right\}$$

$$\leq \frac{\dot{V_2}}{V_2} - \left[\beta_2 c - (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - max\left\{\beta_2 c + max\{-n + \beta_1 c, -k + \eta c\}, -m\right\}\right]$$

$$(9.16)$$

Now, we define that

$$\mu_4 = \left[\beta_2 c - (\mu_3 + \epsilon + \gamma_2 + \tau_4 u_5) - max\left\{\beta_2 c + max\{-n + \beta_1 c, -k + \eta c\}, -m\right\}\right]$$

Using this the equation (9.16) reduces to the following

$$\mu(B) \leq \frac{\dot{V_2}}{V_2} - \mu_4$$

Then integrating the above from 0 to t we have

$$\int_0^t \mu(B)ds \leq \int_0^t \frac{\dot{V_2}}{V_2}dt - \mu_4 \int_0^t dt$$

$$i.e., \quad \int_0^t \mu(B)ds \leq [logV_2(t)]_0^t - \mu_4 t$$

$$i.e., \quad \frac{1}{t}\int_0^t \mu(B)ds \leq \frac{1}{t}log\frac{V_2(t)}{V_2(0)} - \mu_4$$

Therefore, we have

$$\lim_{t\to\infty}\sup\sup\frac{1}{t}\int_0^t \mu(B)ds < -\mu_4 < 0, \text{ provided that } \mu_4 > 0 \qquad (9.17)$$

for all $(V_2(0), Y_2(0), X_2(0)) \in K$.

Then, the equation (9.17) proves that the system (9.14) is globally asymptotically stable when $R_0 > 1$ and $\mu_4 > 0$.

Since the system (9.14) is globally asymptotically stable hence it can be written as

$$V_2(t) \to V_2^*, Y_2(t) \to Y_2^* \text{ and } X_2(t) \to X_2^* \text{ as } t \to \infty$$

Again, from the first equation of system (9.1) we have

$$\frac{dV_1}{dt} = r(1 - V_1/K(t))V_1 - \eta Y_2 V_1 + \xi V_2 - \tau_1 u_2 V_1$$

and its limit form is given by

$$\frac{dV_1}{dt} = r(1 - V_1/K(t))V_1 - \eta Y_2^* V_1 + \xi V_2^* - \tau_0 u_1 V_1 - \tau_1 u_2 V_1$$

$$\frac{dV_1}{dt} = V_1 \left\{ r - \eta Y_2^* + \xi V_2^* - \tau_0 u_1 - \tau_1 u_2 - \frac{rV_1}{K(t)} \right\}$$

$$\frac{dV_1}{dt} \leq V_1 \left\{ r - \eta Y_2^* + \xi V_2^* - \tau_0 u_1 - \tau_1 u_2 - \frac{rV_1}{K_1} \right\}, \text{ where } |K(t)| \leq K_1$$

$$\frac{dV_1}{dt} \leq \frac{r}{K_1} V_1 \left\{ M - V_1 \right\}$$

where $M = \frac{K_1}{r} \left[r - \eta Y_2^* - \tau_0 u_1 - \tau_1 u_2 \right]$.

Now, its solution is given by

$$V_1 \leq \frac{M l_2}{l_2 + e^{-\frac{Mrt}{K_1}}} \text{ where } l_2 \text{ is arbitrary constant.}$$

From this it is seen that when $t \to \infty$ then

$$V_1 \to M, \text{ provided that } M > 0$$
$$V_1 \to V_1^* \text{ where } V_1^* = M$$

Similarly, it can be shown that

$$Y_1 \to Y_1^*, Y_3 \to Y_3^*, X_1 \to X_1^*, X_3 \to X_3^*$$

This proves that the system (9.1) is globally asymptotically stable around E^*.

9.6 Numerical Simulation

To check the feasibility of our analysis regarding stability conditions of the system (9.1), we have conducted some numerical computation using MATLAB by choosing the following set of parametric values:
$r = 2.0$, $K(t) = 1000 + 0.01 sin(\pi t/180)$, $\eta = 0.15$, $\xi = 0.01$, $\alpha = 0.1$, $\mu_1 = 1/20$, $\beta_1 = 0.0001$, $f_1 = 0.3$, $\gamma_1 = 3.0$, $\mu_2 = 50$, $\mu_3 = 1/65$, $f_2 = 0.3$, $\beta_2 = 0.003$, $\epsilon = 1/45$, $\gamma_2 = 0.1$, $u_1 = u_2 = u_3 = u_4 = u_5 = 0$. For the above set of parametric values with different initial values such as $(26, 730), (60, 750)$ and $(65, 530)$ we draw the phase portrait of the system (9.1) which is shown in Figure 9.3. From this figure, it is observed that all the trajectories initiated from different initial values converge to the equilibrium point $(33.72, 651)$ which indicates that the endemic equilibrium point is stable.
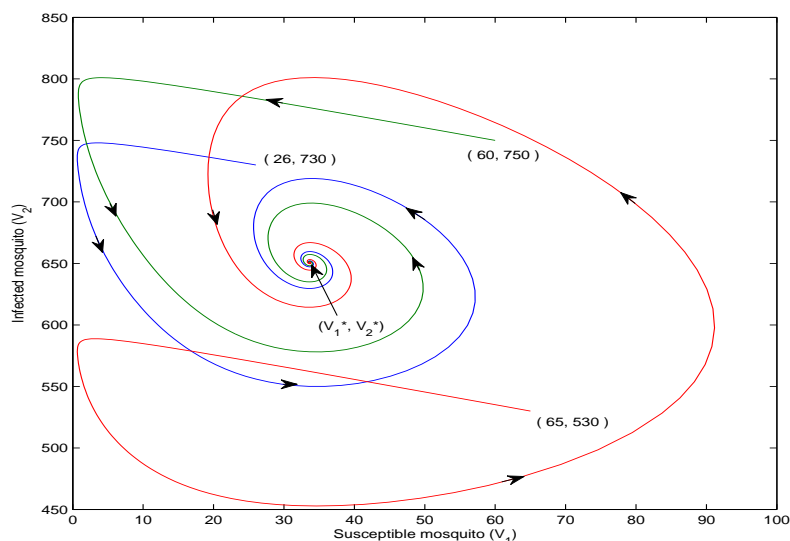
Figure 9.3: Nonlinear stability of (V_1^*, V_2^*) in $V_1 - V_2$ plane.

Without Control

In this section, we have explored some characteristics numerically for the following problems, regarding the stability conditions for the system (9.1).

Problem 1: In this problem, the following parametric values have been considered: $r = 1.0$, $K(t) = 100 + 0.08sin(\frac{\pi}{180}t)$, $\eta = 0.00021$, $\xi = 0.0001$, $\alpha = 0.3$, $\mu_1 = 1/20$, $\beta_1 = 0.0001$, $f_1 = 0.4$, $\gamma_1 = 4.5$, $\mu_2 = 10$, $\mu_3 = 1/65$, $f_2 = 0.4$, $\beta_2 = 0.0003$, $\epsilon = 1/45$, $\gamma_2 = 4.5$.

For these parametric values the conditions stated in **Theorem 9.2** are satisfied, so the disease free solution around E_0 of the system (9.1) will be locally asymptotically stable. Again, using the above set of parametric values, we have $R_0^1 = 0.35$ which is less than one. Hence, this implies that disease tend to extinct which is also observed by Figure 9.4.

Problem 2: Again, in this problem it is considered that $r = 0.6$, $K(t) = 1000 + 0.08sin(\frac{\pi}{180}t)$, $\eta = 0.0021$, $\xi = 0.001$, $\alpha = 0.3$, $\mu_1 = 1/16$, $\beta_1 = 0.01$, $f_1 = 0.4$, $\gamma_1 = 2.0$, $\mu_2 = 20$, $\mu_3 = 1/65$, $f_2 = 0.2$, $\beta_2 = 0.003$, $\epsilon = 1/30$, $\gamma_2 = 0.5$.

For the above parametric values, the reproduction number R_0^1 is 1.11 which is grater than one. So, the system (9.1) for such parameters is endemic according to **Theorem 9.3** which is also depicted by Figure 9.5 where it has been shown that infected mosquito population, infected reservoir population as well as infected human population do not vanish as time increases. It is observed that ultimately these are going to the said endemic equilibrium point $(45.83, 88.89, 643.2, 273.4, 1183.0, 516.6, 29.79, 620.6)$. Again, in this numerical simulation, we have $\mu_4 = 0.0035$ which is grater than zero.

Hence, according to discussion in **Section 9.5**, the endemic equilibrium point
$(45.83, 88.89, 643.2, 273.4, 1183.0, 516.6, 29.79, 620.6)$ is also globally asymptotically stable.
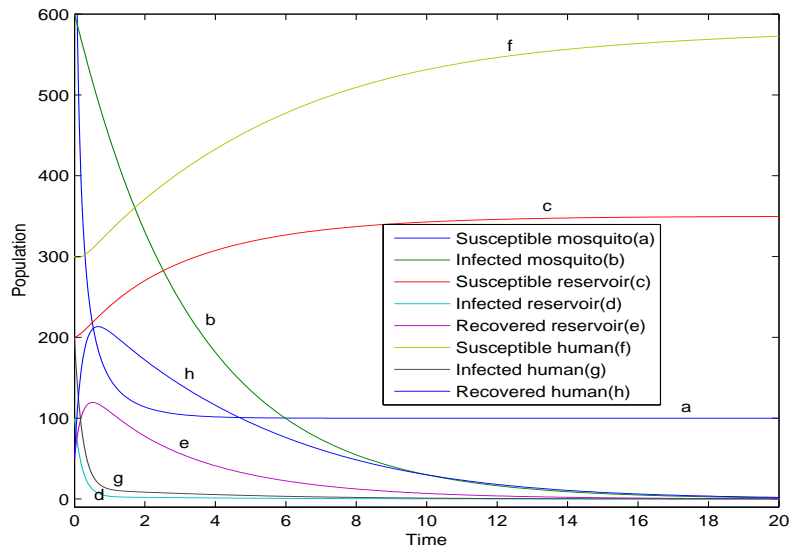
Figure 9.4: Represents disease free equilibrium point without control.
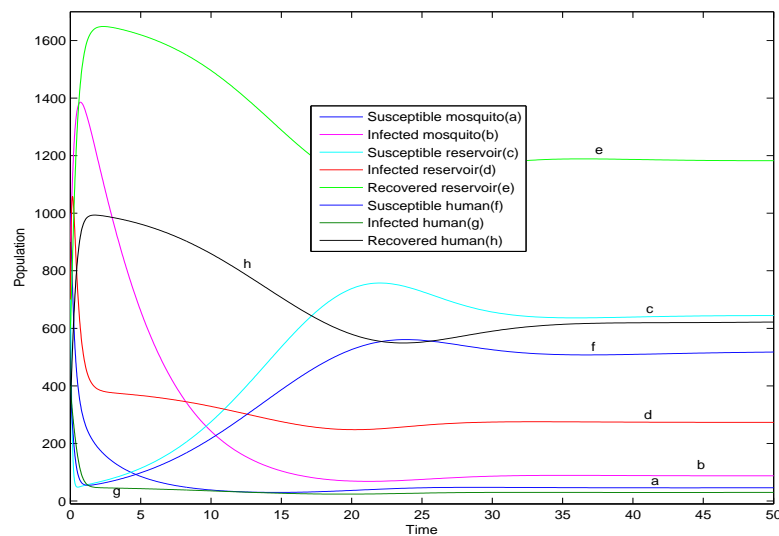
Figure 9.5: Represents endemic equilibrium point without control.

With Control

Problem 3: Here the same set of parametric values has been considered in **Problem 2.** including five control parameters such as: $u_1 = 0.5, u_2 = 0.5, u_3 = 0.5, u_4 = 0.5, u_5 = 0.5$ with full effectiveness i.e., $\tau_0 = \tau_1 = \tau_2 = \tau_3 = \tau_4 = \tau_5 = 1.0$ in the system.

For the above set of parametric values, the system (9.1) is disease free since the reproduction number $R_0^1 = 0.1920$ which is less than one. In Figure 9.6 it has been shown that infected mosquito, infected reservoir and infected human vanish as t tends to infinity.
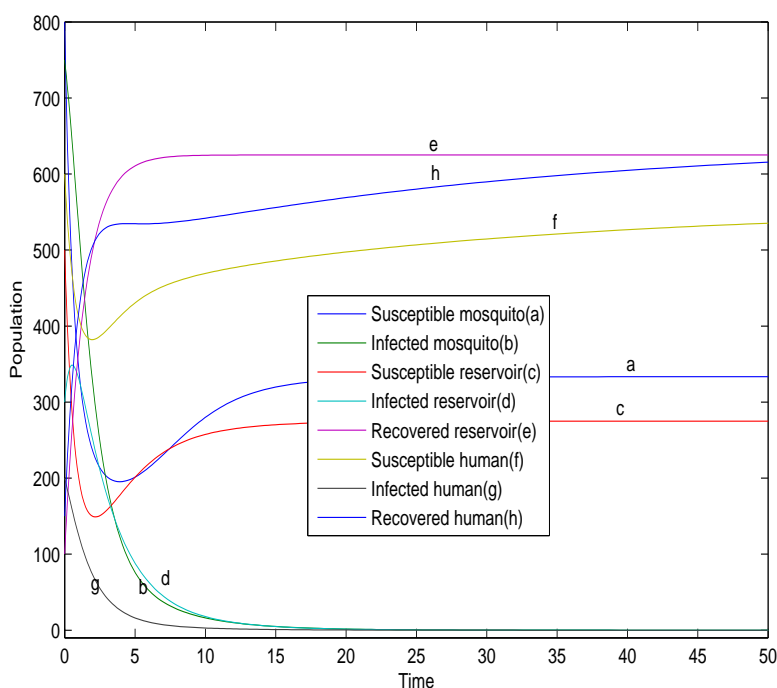
Figure 9.6: Represents the mosquito and human populations with control $R_0^1 < 1$.

Problem 4: For this problem parametric values have been considered as
$r = 1.2$, $K(t) = 1000 + 0.08sin(\frac{\pi}{180}t), \eta = 0.0021, \xi = 0.0001, \alpha = 0.3, \mu_1 = 1/16,$
$\beta_1 = 0.001, \ f_1 = 0.4, \gamma_1 = 4.5, \mu_2 = 150, \mu_3 = 1/65, f_2 = 0.4, \ \beta_2 = 0.0003, \ \epsilon = 1/45,$
$\gamma_2 = 4.5.$

The above parametric values have been used to illustrate the effectiveness of all control parameters on the system to reduce the infected humans from the Japanese Encephalitis disease. Also different results have been given in **Table 9.1** due to different combinations of application of all control parameters.

Table 9.1: Disease extinction or disease persistent

Effectiveness of controls	Values of control parameters	R_0^1	Results
$\tau_0 = 1.0$	$u_1 = 1.0, u_2 = u_3 = u_4 = u_5 = 0.0$	0.4522	Disease free
$\tau_1 = 1.0$	$u_2 = 1.0, u_1 = u_3 = u_4 = u_5 = 0.0$	0.2173	Disease free
$\tau_2 = 1.0$	$u_3 = 1.0, u_1 = u_2 = u_4 = u_5 = 0.0$	0.6229	Disease free
$\tau_3 = 1.0$	$u_4 = 1.0, u_1 = u_2 = u_3 = u_5 = 0.0$	1.1077	Disease persist
$\tau_4 = 1.0$	$u_5 = 1.0, u_1 = u_2 = u_3 = u_4 = 0.0$	1.1077	Disease persist
$\tau_3 = 1.0, \tau_4 = 1.0$	$u_4 = 1.0, u_5 = 1.0, u_1 = u_2 = u_3 = 0.0$	1.1077	Disease persist
$\tau_0 = \tau_3 = \tau_4 = 1.0$	$u_1 = 1.0, u_4 = 1.0, u_5 = 1.0, u_2 = u_3 = 0.0$	0.4522	Disease free

For the above set of parametric values from **Table 9.1** the following observations have been made:

(i) when the control combinations are ($u_4 = 1.0, u_1 = u_2 = u_3 = u_5 = 0.0$), ($u_5 = 1.0, u_1 = u_2 = u_3 = u_4 = 0.0$) and ($u_4 = 1.0, u_5 = 1.0, u_1 = u_2 = u_3 = 0.0$), then the values of R_0^1 is grater than one for each such combination i.e., in these situations the disease persists in the system. Therefore, we can say that

(a) the system remains endemic if only one of the following control parameters such as use of vaccination to the susceptible human (u_4) and treatment of the infected human (u_5) are used in the model.

(b) if we apply one or more control parameters such as u_4, u_5 and (u_4, u_5) in the model then the system remains also endemic.

(ii) when the control combinations are ($u_1 = 1.0, u_2 = u_3 = u_4 = u_5 = 0.0$), ($u_2 = 1.0, u_1 = u_3 = u_4 = u_5 = 0.0$) and ($u_3 = 1.0, u_1 = u_2 = u_4 = u_5 = 0.0$) then the values of R_0^1 is less than one i.e., in such situations the system becomes disease free. Since this set of parametric values satisfies the **Lemma 9.4**.

So, from the above numerical simulations, it is concluded that the control parameters u_1, u_2 and u_3 are more effective than u_4,u_5 and (u_4, u_5).

The Figure 9.7 represents the value of the reproduction number with respect to the control parameters u_1, u_2 and u_3 for the parametric values used in **Table 9.1**. From this figure it is concluded that the reproduction number (R_0^1) is a decreasing function with respect to the above mentioned parameters. Hence the **Lemma 9.3** is also verified.
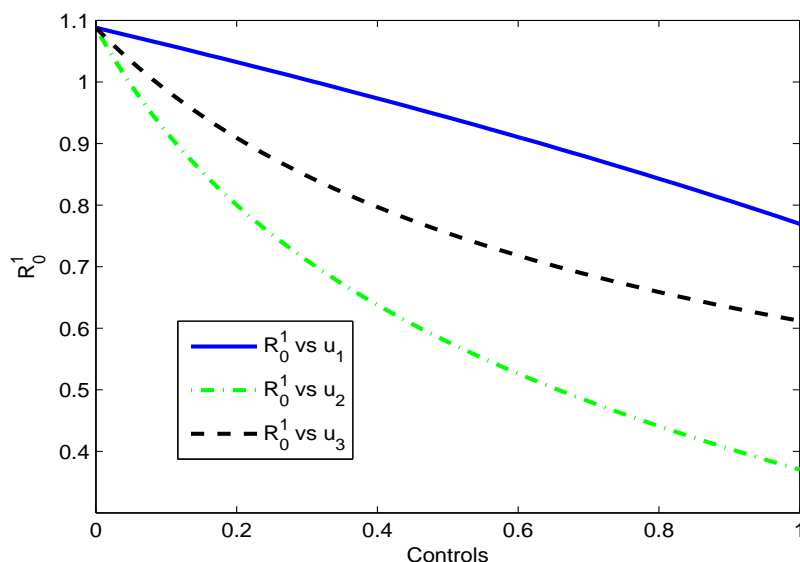
Figure 9.7: Represents the value of R_0^1 with respect to control parameters u_1, u_2 and u_3.

With Hopf bifurcation of the System

In this section, environmental carrying capacity of mosquito population has been considered as a constant without any control parameter i.e., $K(t) = a$ and $u_1 = u_2 = u_3 = u_4 = u_5 = 0$.

Problem 5: For this case the following parametric values have been considered as:
$K = 5.0$, $\xi = 0.02$, $\alpha = 0.2$, $\mu_1 = 0.1$, $f_1 = 0.03$, $f_2 = 0.04$, $\gamma_1 = 0.1$, $\mu_2 = 100$, $\mu_3 = 0.2$, $\beta_2 = 0.03$, $\epsilon = 0.002$, $\gamma_2 = 0.2$, $N_1 = 500$.

For this parametric values, it is seen that our proposed system undergoes Hopf bifurcation with respect to the parameter r (intrinsic growth rate of susceptible mosquito), η (per capita contact rate of infected reservoirs with the susceptible mosquito population) and β_1 (per capita contact rate of infective mosquito with susceptible reservoir population). If we change the value of r, η and β_1 from 0.1 to 0.5, 0.02 to 0.08 and 0.05 to 0.08 then the Hopf bifurcation occurs which have been shown in Figure 9.8, Figure 9.9 and Figure 9.10 respectively. It is clear from the bifurcation diagram that for lower values of $\eta(= 0.0242)$, $\beta_1(= 0.0638)$ and higher value of $r(= 0.384)$ the our proposed system remains stable but, above the threshold value of $\eta(= 0.0242)$, $\beta_1(= 0.0638)$ and lower values of $r(= 0.384)$ our proposed system losses its stability and periodic solution arises through Hopf bifurcation.
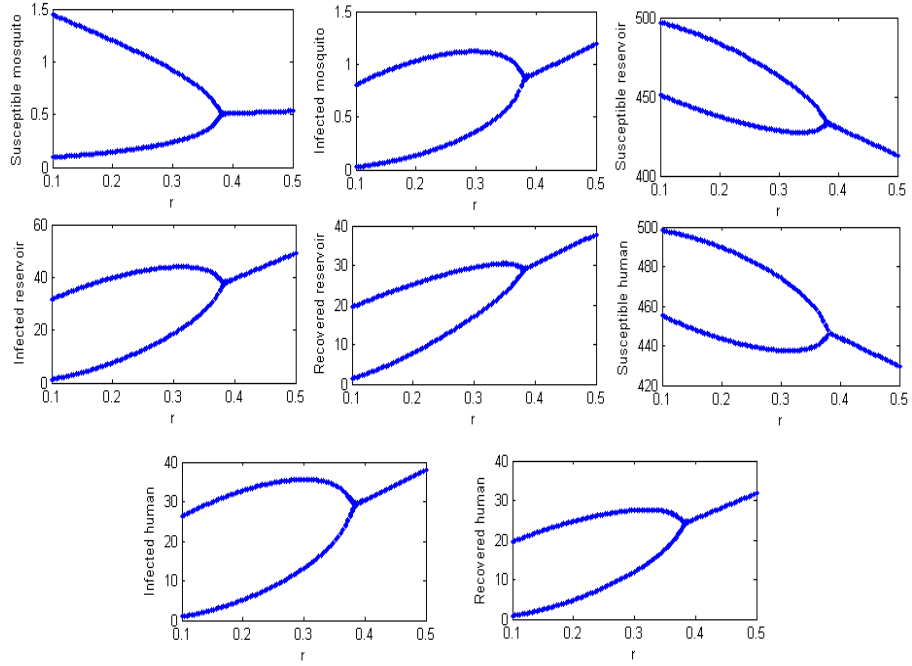
Figure 9.8: Represents bifurcation diagram with respect to r.
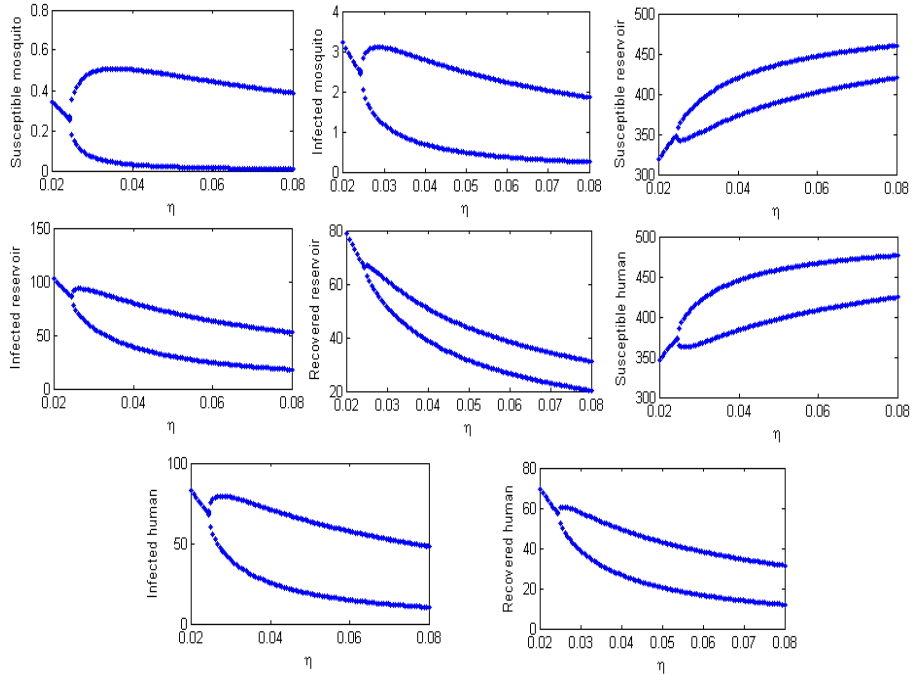
Figure 9.9: Represents bifurcation diagram with respect to η.
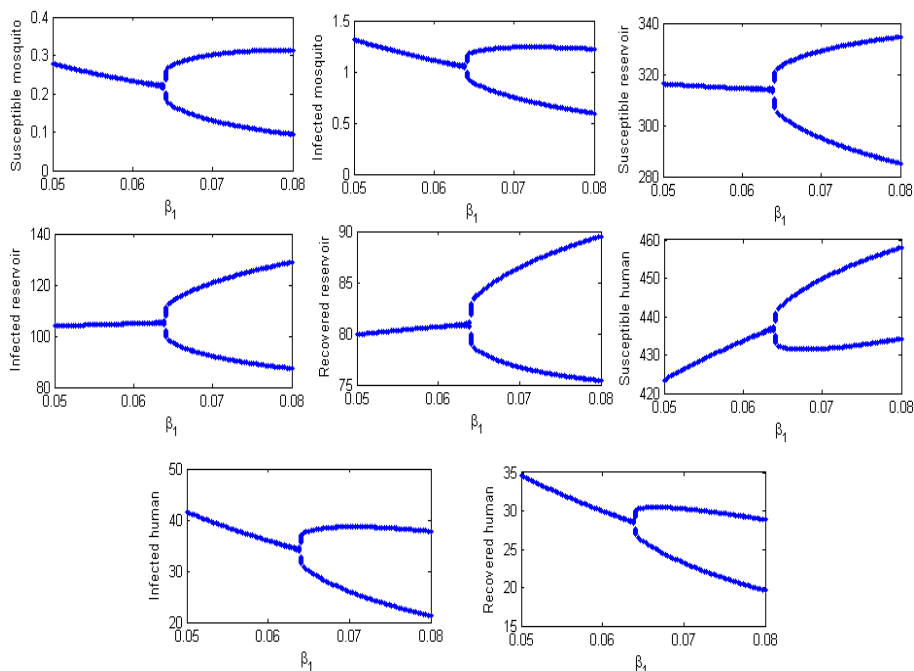
Figure 9.10: Represents bifurcation diagram with respect to β_1.

9.7 Conclusion

In this concluding part we would like to mention that this Japanese Encephalitis model has been derived depending on time dependent carrying capacity and some control parameters. The existence of local stability of disease free equilibria of the model has been analyzed. The condition under which the global stability of endemic equilibria of the system exist has been derived. Theoretically, we have found the required conditions under which system will be disease free and disease persist along with numerical verification also.

From the analysis of this model the following have been drawn

(i) The solutions of the system (9.1) are bounded if $min\{\tau_0 u_1 + \tau_1 u_2, \alpha + \tau_1 u_2\} > 0$.

(ii) The basic reproduction number is a decreasing function with respect to u_1, u_2 and u_3 under some conditions.

(iii) Our proposed system can be disease free with the application of single control of u_1, u_2 and u_3 under some conditions.

(iv) It is also shown that if one control cannot make the system disease free then two or three controls make the system disease free under some conditions.

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(v) The disease free solutions and endemic solutions of our proposed system will be locally asymptotically stable and globally asymptotically stable under certain conditions.

(vi) For a certain set of parametric values from the **Table 9.1** it is concluded that any one control among intermittent irrigation system (u_1), use of pesticide to kill the mosquito larvae (u_2) and the application of vaccination on the reservoir population (u_3) make the system disease free.

(vii) Our proposed system in the absence of control parameters with constant environmental carrying capacity of mosquito undergoes a Hopf bifurcation with respect to some parameters. If we change the intrinsic growth rate of susceptible mosquito, per capita contact rate of infected reservoirs with the susceptible mosquito population and per capita contact rate of infective mosquito with susceptible reservoir population, then a Hopf bifurcation occurs. Our study suggests that if the intrinsic growth rate of susceptible mosquito increases, the the system remains unstable up to a threshold value r^*. But, the system becomes stable above that threshold value. Similarly, if per capita contact rate of infected reservoirs with the susceptible mosquito population (η) and per capita contact rate of infective mosquito with susceptible reservoir population (β_1) increase, then our proposed system remains stable up to a threshold value η^* and β_1^* respectively. But, the system becomes unstable above that thresholds.

Part IV

Summary, Extension and Bibliography

Chapter 10

Summary and Future Research

Work

In this dissertation, some innovative Ecological and Epidemiological problems have been considered and solved.

In **Chapter 2**, the first model of this thesis represents the dynamics of a prey-predator model interacting among three species such as Phytoplankton, Zooplankton and Fish. It is considered that the growth rate of Phytoplankton growth rate is logistic and it produces some toxin which makes some death on Zooplankton population. Again, it is also assumed that Zooplankton consumes only Phytoplankton and Fish consumes Phytoplankton as well as Zooplankton. The equilibrium points of our proposed system are determined and the stability of this system have been discussed around these equilibrium points. After that Hopf bifurcation analysis has been done with respect to the consumption rate of Zooplankton (β) and releasing rate of toxin substances (ρ) produced by unit biomass of Phytoplankton. Finally, sensitivity analysis on two critical parameters R_0 and R_1 involved in the stability of the equilibrium points are also carried out.

Chapter 3 also deals with a prey predator mathematical model where prey population is divided into two subpopulations such as juvenile prey and adult prey. It is assumed that the growth rate of the juvenile prey depends on the adult prey population and is also assumed that juvenile prey becomes adult after staying sometimes on the juvenile stage. In this model, it is considered that predator consumes both juvenile prey and adult prey. An anti-predator behavior has been considered on adult prey population. Due to this behavior, the adult prey makes a defense against the predator to save its infants from predation making some deaths on the predator population. Then, different equilibrium points are determined and stability of the system around these equilibrium points has been done. Hopf bifurcation analysis has been carried out with respect to the

anti predator behavior term (η). Comparison of results with and without anti predator behavior has been resulted analytically as well as numerically.

In **Chapter 4**, the impacts of an additional food on a harvested three species prey predator system have been analyzed. Among three species, one species is prey, other two species are middle predator and top predator. Here, it is assumed that the middle predator can consume only prey and the top predator can consume both prey and middle predator. The harvesting of top predator is considered in this model. It is assumed that some constant amount of additional food is to be supplied to the top predator population. Due to this, the predation pressure on the prey and middle predator population must decrease. Then, the different equilibrium points are determined and the stability of the system around these equilibrium has been carried out. Hopf bifurcation analysis have been investigated with respect to the consumption rate of prey to the top predator (a_3) and the harvesting effort (e). Finally, some numerical simulation has been presented for better understanding the dynamics of the model.

In **Chapter 5**, the effects of additional food on a three species such as prey, predator and super predator have been discussed. In this model, it is assumed that the predator population only consumes the prey population and the super predator consumes only predator. A constant rate of refuge has been considered on the predator population. Also, harvesting on the super predator population has been considered. It is also assumed that a constant amount of additional food be supplied to the super predator population. Henceforth, the predator population is also benefited from this additional food in a little amount. Then, Hopf bifurcation analysis has been done with respect to the predation rate of predator to the super predator (a_2). An optimal harvesting problem has been formulated and solved by using Pontryagin's maximum principle. The effects on the the optimal harvesting rate have been shown with respect to the additional food. Finally, some numerical results are given to substantiate our theoretical results.

In **Chapter 6**, a Cholera disease transmission model has been developed. In this model, the total human population is divided into three subpopulations such as susceptible human, infected human and recovered human and total bacterial population has been divided into three subpopulations such as hyper-infectious, low-infectious Vibrio Cholerae and bacteriophage population. It is considered that the susceptible human becomes infected by hyper-infectious and low-infectious Vibrio Cholerae. It is also assumed that the hyper-infectious Vibrio Cholerae grows through infected human feces, vomiting etc. and the growth of low-infectious Vibrio Cholerae depends on the hyper-infectious Vibrio Cholerae, because hyper-infectious becomes low-infectious after certain times. It is considered that the bacteriophage reduces the density of both hyper-infectious and low-infectious bacterium. Then determining the different equilibrium including disease free and endemic, the stability of the system has been investigated. Here, the required conditions for which the system will be disease free and disease persistent have been explored. Finally, some numerical results are presented to validate our analytical findings.

In **Chapter 7**, a Cholera disease transmission model has been developed considering all parametric values in the model to be fuzzy uncertainty. Here, the model consists of the interaction between human populations to be divided into three subpopulations such as susceptible human, infected human and recovered human along with one bacterial population i.e., Vibrio Cholerae in the environment. It is assumed that the susceptible human becomes infected through food and drinking water contaminated by the Vibrio Cholerae in the environment. The bacterium Vibrio Cholerae are released in the environment through the feces and vomiting of an infected human. Then, a mathematical model is formulated considering interactions among all populations and fuzziness of all parameters. After that the disease free and endemic equilibrium points are determined in the system along with the showing of the stability of the model. The change of equilibrium points with the variation of uncertainty has been investigated. Different solution graphs are given in the numerical section for better understanding the dynamics of our proposed model.

In **Chapter 8**, a Malaria disease transmission model has been developed. Here, total mosquito population is divided into two subpopulations such as susceptible mosquito and infected mosquito and also total human population is divided into three subpopulations such as susceptible human, infected human and recovered human. It is assumed that the growth rate of susceptible mosquito is logistic and susceptible mosquito becomes infected through the bite of infected human. Again, the disease will be spread in the human population when an infected mosquito bites a susceptible human. To eradicate the Malaria disease from human population, three types of control parameters such as bed-nets, insecticide spray and use of citronella plant based product have been considered. Again, it is considered that the disease transmission rate from human to mosquito and mosquito to human are control parameters and time dependent. Here, death rate of mosquito is depended on a control parameter. Then the disease free and endemic equilibrium have been determined. The stability of the system around these equilibrium has been investigated. An optimal control problem of the Malaria disease has been constructed and solved by Pontryagins maximum principle. The Hopf bifurcation has been analyzed with respect to disease transmission rate from human to mosquito (α) and recruitment rate of susceptible human (λ) for autonomous case of the system.

Chapter 9 discusses the derivation of the transmission of Japanese Encephalitis disease among mosquito, reservoir and human populations. The growth rate and carrying capacity of mosquitoes have been considered as logistic and time dependent. It is assumed that the susceptible mosquito becomes infected through the bite of it an infected reservoir and then the susceptible human becomes infected through the bite of infected mosquito. To eradicate the disease from human population, some control parameters such as intermittent irrigation system, use of pesticide to kill the mosquito larvae, application of vaccination on the reservoir, treatment of infected human and application of vaccination to the human have been used. Then the stability of the system around dis-

ease free and endemic equilibrium has been investigated. The Hopf bifurcation analysis have been done with respect to growth rate of susceptible mosquito, per capita contact rate of infected reservoirs with the susceptible mosquito and per capita contact rate of infective mosquito with reservoir respectively. Finally, the numerical simulation has been given to support our analytical results with the effectiveness of using different control strategies.

Future Research Scope

Following modifications can be made in the models described in the thesis for future extension: (*i*) In Model-2.1, the mathematical formulation is quite general and hence the present models can be extended to include ratio dependent functional response, time delay, harvesting on the fish species etc. (*ii*) Model- 3.1 can be extended to include time delay, refuge, harvesting on the predator population etc. (*iii*) Model-4.4 can be extended to include ratio dependent functional response, time delay, refuge, additional food to both top predator, middle predator and prey etc. (*iv*) Model-5.1 can further be extended in several ways like harvesting on both super predator and predator, time delay, anti-predator behavior and others. (*v*) Model-6.1 can be extended by introducing new factors which are responsible for disease transmission, suitable control strategies, time delay on the Vibrio Cholerae in the bacterial population etc. (*vi*) Model-7.1 can be extended by introducing optimal control, time delay, Holling type II or III type functional response etc. (*vii*) Model-8.1 can be extended by consideration of some new type of control strategies, time delay etc. (*viii*) Model-9.1 can further be extended in several ways like optimal control, time delay, time dependent transmission rates etc.

Chapter 11

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