

**STABILITY ANALYSIS OF PREY-PREDATOR SYSTEM
USING TAKAGI-SUGENO APPROACH**

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REQUIREMENTS FOR THE AWARD OF THE DEGREE OF

**DOCTOR OF PHILOSOPHY
IN
MATHEMATICS**

BY
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UNDER THE SUPERVISION OF
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CERTIFICATE

This is to certify that the thesis entitled “**STABILITY ANALYSIS OF PREY-PREDATOR SYSTEM USING TAKAGI-SUGENO APPROACH**”, submitted to the Department of Mathematics, National Institute of Technology, Warangal, is a record of bonafide research work carried out by **Ms. KHUSHBU SINGH**, Roll No. 701964, for the award of Degree of Doctor of Philosophy in Mathematics under my supervision. The contents of the thesis have not been submitted elsewhere for the award of any degree or diploma.

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DECLARATION

This is to certify that the work presented in the thesis entitled “**STABILITY ANALYSIS OF PREY-PREDATOR SYSTEM USING TAKAGI-SUGENO APPROACH**”, is a bonafide work done by me under the supervision of **Prof. K. Kaladhar**, Assistant Professor, Department of Mathematics, National Institute of Technology, Warangal and has not been submitted elsewhere for the award of any degree or diploma.

I declare that this written submission represents my ideas in my own words and where others' ideas or words have been included, I have adequately cited and referenced the original sources. I also declare that I have adhered to all principles of academic honesty and integrity and have not misrepresented or fabricated or falsified any idea / data / fact / source in my submission. I understand that any violation of the above will be a cause for disciplinary action by the Institute and can also evoke penal action from the sources which have thus not been properly cited or from whom proper permission has not been taken when needed.

Khushbu Singh

Roll No. 701964

Date: _____

Dedicated

to

Lord Shiva

and

My beloved parents

Shri. Lal Bahadur and Smt. Savitri

&

My Teachers

Who made me what I am today

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Abstract

Predator-Prey model is a relationship between two species living in the same space. It gives the effect on population between the two species. When two species are living in same habitat they share some resources such as food resource and ecological niche. A predator-prey interaction has been described firstly by two pioneers Lotka and Volterra in two independent works. After them, more realistic prey-predator models were introduced by Holling suggesting three kinds of functional responses for different species to model the phenomena of predation.

Almost all of the physical dynamical systems in real life cannot be represented by linear differential equations. The non-linear model is analyzed with the help of Takagi-Sugeno Fuzzy model. The fuzzy model proposed by Takagi and Sugeno is described by fuzzy IF-THEN rules which represents local input-output relations of a nonlinear system. This has motivated the work in this thesis, where an attempt has been made to study the stability of Lotka-Volterra predator-prey system with fuzzy impulsive control.

The thesis has four parts, which consists of ten chapters. **Part-I** consists of a single chapter (chapter 1) which gives an introduction to the problems discussed in this thesis and it provides motivation to the study carried out. A survey of pertinent literature is presented to show the significance of the problems considered. **Part-II** contains three chapters, 2, 3 and 4, which deals with the stability of interaction dynamics of two and three species prey - predator system without infection. **Part-III** deals with the the stability of prey and predator system with infection. It consists of five chapters, namely 5, 6, 7, 8, and 9.

In all the above chapters, mathematical models are considered to study the relationship among preys and predators. We have two, three species Lotka-Volterra predator-prey models with imprecise biological parameters. To improve the model's reality we analyze the global and asymptotic stability of this model with the help of the Takagi-Sugeno (T-S) model. The T-S impulsive control model and the fuzzy impulsive control models were used to explore the stability of the Lotka-Volterra predator-prey system. The impulsive control technique, which is analyzed in the framework of the fuzzy systems based on T-S model, is found appropriate for very complex and non-linear system with impulsive effects.

Part-IV consists of a single chapter 10, which presents the summary of the thesis with main conclusions and point out various problems which are yet to be solved in this area of research.

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Nomenclature

Notations	Physical Significance	S.I.unit
$a'_i s$	half-saturation constants	$kg.m^{-3}$
B	predator's birth rate	t^{-1}
$C, C'_i s$	conversion rate	$kg t^{-1}$
D	predator's natural death rate	t^{-1}
e	intra-specific competition	not specific (depends on frequency, intensity, or magnitude of interaction.)
h_1, h_2	help between the preys	not specific
$m, m'_i s$	death rate of predators	t^{-1}
$P, P'_i s$	predation rate of predators	$x.t^{-1}$
$r, r'_i s$	growth rate of prey	t^{-1}
r_{12}	the interaction of predator with prey	not specific
r_{21}	prey interaction with predator	not specific
t	time	t
$x(t)$	population of preys	$x.m^{-2}$
$y(t)$	population of predators	$y.m^{-2}$
α	ratio between capture rate of the second prey and the first prey	<i>dimensionless</i>
$\beta, \beta'_i s$	infection transmission	t^{-1}
λ	ratio between handling time of the predator per second prey item and first prey item	<i>dimensionless</i>
ω	diagonal matrix	$n.n$
δ_j	impulsive distance	m
ϵ	constant scalar	dimensionless
$\lambda(\alpha)$	maximum eigen value	dimensionless
μ	no. of fuzzy rules	dimensionless

Part I

Introduction

Chapter 1

Introduction

1.1 Preliminary

All living things require energy. However living things get their energy in different methods. Sometimes one individual consumes another individual. No species of animals lives in complete isolation. Since all animals must eat to live, all must interact, if not with other animals then with plants. No two species can exist in the same niche without ending up in the competition i.e. if two species share the same niche then they would end up in the competition, so the result of competition will be either win/loss or partition. In the case of partition both the species are living in the same habitat but limiting their resources. In the case of win/loss, one will win and one will lose. So the species which win will exist and one which lose will extinct. The one which wins is called **PREDATOR** and one which lose is called **PREY**.

1.2 Prey - Predator Model

The Prey - Predator model is a relationship between two species living in the same space. It gives the effect on the population between the two species. It provides

information about competition between two species living in the same ecosystem.

- **PREDATOR** is an animal that hunts, kills and eats other animals for food. Predator species need to be adapted for efficient hunting if they are to catch enough food to survive.
- **PREY** is a term used to describe organisms that predator kill for food. Prey species must be well adapted to escape predators for their species to continue.

1.3 Lotka - Volterra Prey - Predator Model

The idea to predict the outcome of competition based on the impact of one species on another is given by Lotka-Volterra model. A predator-prey interaction has been described firstly by two pioneers Lotka and Volterra in two independent works [1, 2]. After them, more realistic prey-predator models were introduced by Holling suggesting three kinds of functional responses for different species to model the phenomena of predation. It shows the relationship between predator and prey. The Lotka-Volterra equations are a pair of first order non-linear differential equations used to describe the dynamics of biological system in which two species interact one as predator and the other as prey. There has always been an unique interest in the study of population evolution, beginning with populations of a single species and progressing to more realistic models where various species coexist and communicate with one another in the same ecosystem. Between these, we can find models those look at predator-prey relationships, symbiosis, or competitive connections. Since the well-known Lotka-Volterra model was developed and resolved the major issues with ecological processes [3]. Lotka and Volterra made the first breakthrough in contemporary mathematical ecology for a predator-prey competing species. Following Lotka and Volterra's pioneering work on the predator-prey model, the latest mathematical ecology has attained an essential position in analytical biology. Hence, the

mathematical models are frequently used by applied mathematicians to analyze the intricate interactions between predators and prey. Therefore, the classical ecological models of interacting populations typically focused on two species.

1.3.1 Assumptions

- Let $x(t)$ and $y(t)$ be the population of prey and predator species at time t .
- In the absence of predators, the prey population will grow naturally.

Mathematical model for population change of prey is:

$$\frac{dx}{dt} = ax, a > 0 \quad (1.1)$$

- In the absence of prey, the predator population will decrease at the natural rate. Mathematical model for population change of predator is:

$$\frac{dy}{dt} = -Py, P > 0 \quad (1.2)$$

- The presence of both predators and prey is beneficial to the growth of predator species and is harmful to the growth of prey species, i.e. the predator species increases and the prey species decreases at a rate proportional to the product of the two populations.

1.3.2 Basic equations

With the above assumptions, the system of non-linear first-order O.D.E are

$$\frac{dx}{dt} = ax - bxy \quad (1.3)$$

$$\frac{dy}{dt} = qxy - Py \quad (1.4)$$

where a, b, P, q are positive constants, $\frac{dx}{dt}$, $\frac{dy}{dt}$ represent the instantaneous growth rate of the two populations, ax represents the exponential growth of the prey when the prey is assumed to have an unlimited food supply and reproduce exponentially unless subject to predation, bxy represents the rate of the predation upon the prey, qxy represents the growth of predator populations, Py represents the loss rate of the predators due to either natural death or emigration.

- A predator prey model is an essential tool in ecology and specifically for our understanding of interacting populations in the natural environment.
- Predator- prey models are arguably the building blocks of the bio and eco-systems, as bio-masses are grown out of their resource masses. Species compete, evolve, and disperse simply for the purpose of seeking resources to sustain their struggle for their existence.
- Models of competitive interaction of the predator - prey are widely used for the analysis of economic processes and phenomena. Like enterprizes, industries, brands, products, and technologies can compete with each other in time.

1.4 Epidemiology

The study of disease transmission in animals is known as epidemiology. Disease's impact on eco-systems is a significant topic from both a mathematical and an ecological point of view. As a result, ecologists and academicians have been focusing more and more on the creation of key tools, as well as experimental ecology, to characterize how ecological species are infected. The consequence of infection in predator-prey model with disease in prey and predator has been investigated enormously in last few years by many researchers.

In the study of infectious disease transmission and control, mathematical models have become indispensable tools. Most infectious disease transmission models are based on Kermack and Mckendrick's initial SIR model [4]. When exposed to contagious individuals, sensitive becomes infectious. Scientists have recently focused a lot of emphasis on epidemiological models. Numerous scientists, Anderson [5], Hethcote [6] exclusively addressed single-species models in classical epidemiological frameworks. This is because sick species are less active and so more easily caught as shown in [7, 8, 9, 10].

1.5 Takagi - Sugeno Model

Almost all of the physical dynamical systems in real life cannot be represented by linear differential equations. Apart from the traditional methods like Direct approach for solving the non-linear system of equations recent Fuzzy method approaches have been developed. The solution of non-linear systems by classical methods is not easy due to its non-linearity, analytical complexity, chaotic behavior, etc. Hence, the T-S method is very much effective to analyze the non-linear models. The fuzzy model proposed by Takagi and Sugeno is described by fuzzy IF-THEN rules which represents local input-output relations of a nonlinear system. The main feature of a Takagi-Sugeno fuzzy model is to express the local dynamics of each fuzzy implication (rule) by a linear system model. It develops a systematic approach to generate fuzzy rules from a given input-output data set. Until recently, less work has been done on the stability of Lotka-Volterra predator-prey system with fuzzy impulsive control. T-S method is very much useful as it is less time consuming and easy to solve complex systems. We can easily analyze the stability of the complex systems using T-S method as given in [11, 12, 13].

The Takagi-Sugeno (T-S) approach is a method used in fuzzy logic modeling to approximate complex nonlinear systems using a set of fuzzy if-then rules. This

approach is particularly advantageous when dealing with systems that are difficult to model using traditional mathematical techniques, such as predator-prey models, which often involve nonlinear interactions and uncertainties. The Takagi-Sugeno approach offers several advantages for improving the realism of predator-prey models by effectively handling nonlinear relationships, incorporating uncertainty, providing linguistic interpretability, adapting to data, and handling multiple input variables. These features make it a valuable tool for modeling and understanding complex ecological systems.

The Takagi-Sugeno (T-S) fuzzy impulsive control model is a framework that combines fuzzy logic with impulsive control techniques to address complex, nonlinear control systems.

1.6 Fuzzy Impulsive Control

Most plants in engineering, science, and industries have inherent non-linearity and are difficult to design and control using general nonlinear systems. In order to overcome this kind of difficulties, many researchers have developed various schemes, among which a successful approach is fuzzy impulsive control combined with the linguistic knowledge representation. For instance, one can see temperature control in rapid thermal processing [14], the control of a flexible robot system [15], an automated highway system [16]. In parallel with these practical applications, theoretical researches with respect to fuzzy control have been performed to include many control issues. Stability analysis is certainly one of the most important issues that theoretic efforts have focused on.

Table 1.1: Various impacts of the Lotka-Volterra Prey-Predator and Takagi-Sugeno Models

Lotka-Volterra Prey-Predator Model	Takagi-Sugeno Model
Population Dynamics: The model provides a mathematical framework for understanding how predator and prey populations interact over time.	Control Systems: The TS model is extensively used in designing controllers for nonlinear systems, providing a flexible approach to handle systems where linear control methods are insufficient.
Stability Analysis: By analyzing the model, ecologists can study the conditions under which predator-prey systems are stable or unstable, leading to insights into ecosystem stability and resilience.	Engineering Applications: In robotics, the TS model helps in path planning, navigation, and control, allowing robots to handle uncertain and dynamic environments effectively.
Mathematical Ecology: The model has spurred the development of mathematical ecology, a field that uses mathematical approaches to study ecological systems.	Signal Processing: TS models are used in signal processing for noise reduction and filtering, enhancing the quality of signals in various applications like audio processing and telecommunications.
Epidemiology: Analogous models are used to study the spread of infectious diseases.	Environmental Systems: TS models are used to model and predict climate systems, which are inherently nonlinear and complex.
Agriculture: The model helps in understanding the dynamics of agricultural pests and their natural predators, guiding the development of sustainable pest management practices.	Industrial Applications: In industries such as chemical manufacturing, the TS model is used for process control, optimizing production processes, and maintaining product quality.
Lotka-Volterra predator-prey model highlight the need for more realistic, adaptable, and integrative approaches.	Until recently, less work has been done on the stability of Lotka-Volterra predator-prey system with TS fuzzy impulsive control.

Addressing these gaps will not only advance theoretical understanding but also enhance the practical applicability of these models across various fields.

1.7 Literature Survey

Lotka-Volterra was the pioneer in the subject of bio-mathematics. Volterra proposed the differential equation in 1925 to answer the problem of rapid changes in prey and predator populations. The predator-prey system has been used in a variety of fields and has played a significant role in bio-mathematics. Further, the stability of the predator-prey system is being given more attention [17]. The Lotka-Volterra

model is a type of interference competition in which two species' per capita growth rates are projected to be reduced by each other. Li and his team [18] investigated the Lotka-Volterra predator-prey system's impulsive control and discovered the necessary conditions for asymptotic stability using Lyapunov functions [19, 20, 21].

Following Lotka and Volterra pioneering work, the prey-predator concept has become a prominent and significant research subject in applied sciences. Later Kermack and McKendrick [4] extended their contribution to the mathematical theory of epidemics. Over the last few decades, there has been an increasing interest in studying the effects of illnesses in prey-predator systems (Haque and Venturino [22]). To represent the complicated interaction between interacting prey and predators, good number of prey-predator models have been proposed and thoroughly investigated in the real-world environment species. In view of significance, Mahapatra and Santra [23] studied the prey-predator model for optimal harvesting with prey refuge. Liu and Liu [24] explained the behavior of a stochastic model of predation including three species of prey and predators intraguild. Recently, Hu et al. [25], outlined the behavior of a predator-prey model with a constant yield of prey.

Traditional models for ecology of inter-connected populations have mainly concentrated on two species. According to Price et al. [26], social behavior should depend on at least three trophic levels. The literature has looked into continuous time models of two interacting species in great detail [27]. These models can only display on two basic patterns mathematically, such as an approach to a limit cycle or a steady state [28]. However, it has been found that ecological groups in nature have extremely complicated dynamical tendencies. There are reports of more intricate patterns in three species continuous time models [29, 30, 31, 32].

In 1986, Anderson and May [33] introduced infectious disease transmission into a predator-prey model, assuming that the infection is only transmitted inside the prey species. The traditional Lotka-Volterra predator-prey model, in which infection

spreads among either the prey or the predator, was explored by Venturino [34]. However, a very less study have been done with infection in predator [35, 36]. Venturino examined the local properties of eco-epidemic models in predator-prey systems with disease only in the predator population. Haque and Venturino [37] investigated the importance of infectious disease in the Holling Tanner predator-prey model and found a number of interesting results. They came to the conclusion that disease in any species can be used as a biological control. Haque and Venturino [38] looked at the function of transmissible disease in predator species using ratio-dependent functional responses. Pada [39] recently examined predator-prey changing in presence of infection in the predator and demonstrated local stability analysis at the equilibrium point using basic reproduction numbers [40].

In recent times many authors such as Haque [41] investigated and developed different predator-prey models in existence of disease. Recently, Nandadulal et al [42] performed a qualitative investigation of a fishery's bio-economic management in presence of some infection.

Venturino [43], Hethcote et al. [44], Yongzhen et al. [45] examined the standard Lotka-Volterra prey-predator model in which illness spreads among either the prey or the predator. Recently, Soufiane Bentout and Salih Djilali [46] suggested an age-structured predator-prey model in which infection evolves in the prey population. Nazmul and Samares Pal [47] proposed a predator-prey model that includes infection of the prey population and components such as fear, protection, and prey.

During recent years, mathematical modeling in ecology and epidemiology has grown to be one of the most essential tools available, since it is so helpful for interpreting and studying the systems' vital behavior. An important area of discussion in theoretical ecology has been the dynamic interaction between predators and their preys. The main element in models of predator-prey interaction is the predator's functional reaction on the population of prey, it explains how many preys are con-

sumed by a predator per unit time for specific prey-predator concentrations. Even there are other types of functional responses, the Lotka-Volterra functional response are the most significant and beneficial functional responses [48, 49].

One of the most intriguing areas in mathematical biology is the interaction between predators and prey. The well-known Lotka-Volterra predator-prey model is the first mathematical representation of the interaction between predators and prey [50], which is a two-species model. Some scholars have noted that population models with two species can't accurately capture the real world [51, 52], and models with three or more species can only depict a significant number of crucial behaviors. The advancement of mathematics also demonstrated that three-species food chain models have significantly more detailed features than two-species models [53, 54].

Since the impact of infectious diseases on the ecological system regulates population size, researchers have recently become more interested in the fusion of ecology and epidemiology. There are a lot of prey-predator models that have infectious infections. The dynamics of the prey-predator system with disease in the prey and predator populations were hypothesized and examined by Venturino [55, 56], Hsieh and Hsiao [57], Haque and Venturino [58], Haque et al. [59, 60], Xiao and Chen [61], Zhou et al. [62], Tewa [63], Hudson [64], recently, Deng [65] etc. Additionally, numerous research studies have explored the dynamic behavior of the predator-prey system with infection in the predator population. There are also several scholars who have studied eco-epidemic models where predator populations are infected through consuming prey (Anderson and May [33], Haderler and Freedman [66] etc). Additionally, some researchers have developed eco-epidemic models with optimal control [67].

In recent years, fuzzy impulsive theory has been applied to the stability analysis of the non-linear differential equations [68, 69, 70]. However, it should be admitted that the stability of fuzzy logic controller (FLC) is still an open problem. It is

well-known that the parallel distributed compensation technique has been the most popular controller design approach and belongs to a continuous input control way. It is important to point out that there exist many systems, like the predator-prey system, which cannot commonly endure continuous control inputs, or they have impulsive dynamical behavior due to abrupt jumps at certain instants during the evolving processes. In this sense, it is the same with communication networks, biological population management, chemical control, and so forth [71, 72, 73, 74, 75]. Hence, it is necessary to extend FLC and reflect these impulsive jump phenomena in the predator prey system. Until recently, few papers talk about the stability of Lotka-Volterra predator prey system with fuzzy impulsive control.

1.8 Thesis Summary

In view of the above discussion, it can be noted that until recently, a few researchers presented the stability of two-dimensional Lotka-Volterra predator-prey system with fuzzy impulsive control. So we have considered the Lotka-Volterra prey-predator population's model with and without disease. To improve the model's reality, we analyzed the global and asymptotic stability of this model with the help of the T-S approach [36]. Initially, using the T-S mathematical model and fuzzy impulsive control, the stability of the predator-prey system is examined with the help of theorems. Finally, the graphical solutions for the problems were presented.

The thesis has four parts and consists of ten chapters.

Part-I consists of a single **Chapter 1** which gives an introduction to the problems discussed in this thesis and it provides motivation to the study carried out. A survey of pertinent literature is presented to show the significance of the problems considered. The basic equations governing the Lotka-Volterra predator-prey model, which is relevant for the investigation presented in the thesis.

Part-II deals with the stability of prey and predator systems without infection.

This consists of three Chapters 2, 3 and 4. In each of these chapters, the Lotka-Volterra predator-prey model with imprecise biological parameters are considered. To improve the model's reality, we analyze the global and asymptotic stability with the help of the T-S model.

Chapter 2, presents a mathematical model to study the relationship between prey and predator. In this we have two species Lotka-Volterra predator-prey model with imprecise biological parameters. The asymptotic stability of the impulsive fuzzy system is shown by various stability theorems. Numerical example with prey and predator system with impulsive effects is given to illustrate the application of impulsive fuzzy control, and simulation results shows the effectiveness of the proposed method.

In **Chapter 3**, the three-dimensional Lotka-Volterra predator-prey system's stability has been examined using the Takagi-Sugeno (T-S) and the Fuzzy impulsive control model. The main focus of this chapter is to examine the stability of predator-prey model with one predator and two preys and to examine the interaction between the considered preys and the predator.

In **Chapter 4**, we investigate the interaction dynamics of one prey and two predators. The three-dimensional Lotka-Volterra prey-predator system's stability has been investigated by applying the Takagi-Sugeno (T-S) impulse control model and the Fuzzy impulse control.

Part-III deals with the stability of prey and predator system with infection. This part consists of five chapters, namely 5, 6, 7, 8, and 9. In all these chapters, the species are divided into two categories such as the susceptible species and the infected species. Then the stability analysis of the predator-prey model is analyzed using the Takagi-Sugeno (T-S) based fuzzy impulsive control. After creation of the design, the global stability as well as the fuzzy solutions are discussed via numerical recreations and graphical representations with suitable discussion to prove the applicability of

the considered system.

In **Chapter 5**, the study is based on a predator-prey model with an infection that affects only predator species. Predators are divided into two categories such as the susceptible predator and the infected predator, which are feeding on prey species. Numerical simulation provides global stability and the fuzzy solution.

Chapter 6, presents a mathematical model based on the predator-prey model with disease infection on the prey. Prey species are divided into two categories - susceptible and infected prey species. Here we present a disease that affects only prey species.

In **Chapter 7**, an emphasis is given to study the dynamical behavior of a prey-predator system in which disease infection is in both the prey and predator populations. Prey and Predators are divided into two categories - the susceptible and the infected. A system of four differential equations has been proposed and analyzed.

In **Chapter 8**, we took into account an eco-epidemic model with two preys and one predator, with the infectious disease infecting only the first prey population. The relationship connecting the second prey-predator is supposed to be represented by Lotka-Volterra's functional response.

In **Chapter 9**, we develop a set of ordinary differential equations that represents the dynamics of an ecosystem with two predators and one prey, but only the first predator is affected by an infectious disease. The global stability and the Fuzzy solution are carried out through numerical simulations and graphical representations with appropriate discussion for a better understanding of the dynamics of this proposed model.

The main conclusions of the earlier chapters and the directions in which further investigations may be carried out are indicated in **Part-IV, Chapter - 10**.

Part II

Population Dynamics of prey and predator system

Chapter 2

Stability Analysis of a T-S based intra-specific predator-prey competition model with Fuzzy Impulsive Control ¹

2.1 Introduction

For biologists concerned with the results of competitive interactions between species, the Lotka-Volterra model of intra-specific competition is a good place to start. The model's assumptions (for both species, such as carrying capacity and competition coefficients) may be unrealistic, but they require explanations. By changing the dynamics of one or both populations, several factors can influence the outcomes of competitive interactions. Many researchers have built models based on the premise that biological parameters are exactly identified, but the values of all the parameters are not always known exactly due to various factors such as a lack of knowl-

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edge, data limitations, variability in natural systems, environmental factors such as climate, habitat quality, and resource availability. The impact of above factors on predator and prey populations can be significant and can manifest in various ways: Lack of knowledge and data limitations can lead to uncertainty in parameter estimation, fluctuations in climate or habitat quality can affect prey abundance and distribution, impacting predator foraging behavior and population dynamics, environmental factors such as climate change and habitat degradation can alter ecosystem resilience, making predator and prey populations more vulnerable to disturbances and fluctuations. For dealing with these challenges, fuzzy set theory is a useful tool [76, 77].

In recent times, fuzzy impulsive concept has been used to analyze nonlinear differential equations as well [78, 68]. Many systems, such as predator-prey systems exhibit impulsive dynamical behavior, as a result of rapid changes at specific points during developing processes. As a result, the predator-prey system's fuzzy logic controller (FLC) must be increased, and these impulsive changes in the predator-prey system must be observed [79, 80, 81].

In this chapter, we have two species Lotka-Volterra predator-prey model with imprecise biological parameters. To improve the model's reality we analyze the global and asymptotic stability of this model with the help of T-S model [82, 83, 84] then presented the graphical solutions of the problem.

2.2 Model Formation

Inspired by the predator-prey relationship in Paul and Bhattacharya [85], the two species model considered as:

$$\begin{aligned}\frac{dx}{dt} &= x(r - r_{12}y - ex - q_1E_1) \\ \frac{dy}{dt} &= y(r_{21}x - m - q_2E_1)\end{aligned}\tag{2.1}$$

where x be the total population density of the prey, y be the predator total population density, t be the number of years, $x = x_0 > 0$, $y = y_0 > 0$ denote the density of prey and predator at time t respectively. The coefficients $r > 0$, $m > 0$ signifies the prey birth rate and predator death rates respectively. The coefficients $r_{12} > 0$, $r_{21} > 0$ gives the interaction between the species. Here q_1, q_2, E_1 are positive constants, and e be the intra-specific competition.

A matrix differential equation is stated as follows to analyze the system's stability:

$$\dot{x} = Ax + \phi(x)\tag{2.2}$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}(t) \\ \dot{y}(t) \end{pmatrix}, A = \begin{bmatrix} r - q_1E_1 & 0 \\ 0 & -m - q_2E_1 \end{bmatrix}, \phi(x) = \begin{bmatrix} -r_{12}xy - ex^2 \\ r_{21}xy \end{bmatrix}$$

2.3 T-S Fuzzy model with Impulsive effects

2.3.1 lemma

Let $\dot{x} = f(x(t))$, here the state variable is $x(t) \in R^n$, and $f \in C[R^n, R^n]$ fulfills the condition $f(0) = 0$, is a compact vector field defined in $W \subseteq R^n$. Using the techniques proposed by Tanaka and Wang [86]. We can build a fuzzy model for system (2.1) as shown below:

Control Rule i ($i = 1, 2, \dots, \mu$): IF $z_1(t)$ is M_{i1} , $z_2(t)$ is M_{i2} ... and $z_p(t)$ is M_{ip}

THEN $\dot{x}(t) = A_i x(t)$, where μ is no. of T-S fuzzy rules, $z_1(t), z_2, \dots, z_p(t)$ are the premise variables, each M_{ij} is a fuzzy set and $A_i \subseteq R^{n \times n}$ is a constant matrix.

Thus, the non-linear equations can be transformed into the following linear equations. If $x(t)$ is M_i then,

$$\begin{aligned} \dot{x}(t) &= A_i x(t), t \neq \tau_j \\ \Delta(x) &= k_{ij} x(t), t = \tau_j \\ i &= 1, 2, 3 \dots \mu; j = 1, 2, \dots \end{aligned} \quad (2.3)$$

where $A_i = \begin{bmatrix} r - q_1 E_1 - z_1 & 0 \\ z_2 & -m - q_2 E_1 \end{bmatrix}$, $i=1$ to 4, where the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 2 and z_1, z_2 are related to the values of $x(t) \in [0, d_1]$, $y(t) \in [0, d_2]$ (here $z_1 = ex + r_{12}y$, $z_2 = r_{21}y$). M_i , $x(t)$ and $A_i \in R^{2 \times 2}$, μ is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta x_{t=\tau_j} = x(\tau_j - \tau_{j-1})$.

With centre-average defuzzifier, the T-S fuzzy impulsive system as a whole may be written as:

$$\begin{aligned} \dot{x}(t) &= \sum_{i=1}^r h_i(z(t))(A_i x(t)); \quad t \neq \tau_j \\ \Delta(x) &= \sum_{i=1}^r h_i(z(t))K_{ij}; \quad t = \tau_j \end{aligned} \quad (2.4)$$

where, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$.

Evidently, $h_i(z(t)) \geq 0$, $\sum_{i=1}^r h_i(z(t)) = 1, i = 1, 2, \dots, r$

2.4 Stability Analysis

Now we'll look at the impulsive fuzzy system's numerous stability (2.4) by considering the following theorems [83].

2.4.1 Theorem

Assume that λ_i is maximum eigen value of $[A_i^T + A_i]$, ($i = 1, 2, 3 \dots r$) . Let $\lambda(\alpha) = \max_i \{\lambda_i\}$, $0 < \delta_j = \tau_j - \tau_{j-1} < \infty$ is impulsive distance [81]. If $\lambda(\alpha) \geq 0$ and there exists a constant scalar $\epsilon > 1$ and a semi-positive matrix P , such that

$$\ln(\epsilon \omega_j) + \lambda(\alpha) \delta_j \leq 0 \quad (2.5)$$

where

$$P = C^T C, \omega_j = \max_i \|C(I + K_{i,j})\| \quad (2.6)$$

then the system (2.4) is stable globally and asymptotically.

2.4.2 Theorem

Assume that λ_i is maximum eigen value of $[A_i^T + A_i]$, ($i = 1, 2, 3 \dots r$) . Let $\lambda(\alpha) = \max_i \{\lambda_i\}$, $0 < \delta_j = \tau_j - \tau_{j-1} < \infty$ is impulsive distance. If $\lambda(\alpha) < 0$ and a constant scalar $0 \leq \epsilon < -\lambda(\alpha)$

such that

$$\ln(\omega) - \epsilon \delta_j \leq 0 \quad (2.7)$$

where

$$P = C^T C, \omega_j = \max_i \|C(I + K_{i,j})\| \quad (2.8)$$

then the system (2.4) is stable globally and exponentially.

2.5 Numerical Simulation

By using fuzzy impulsive T-S design model on (2.2), the membership functions [86] obtained as:

$L_1 = \frac{z_1}{(ed_1 + r_{12}d_2)}$, $L_2 = \frac{(ed_1 + r_{12}d_2) - z_1}{(ed_1 + r_{12}d_2)}$, $L_3 = \frac{z_2}{r_{21}d_2}$, $L_4 = \frac{d_2r_{21} - z_2}{r_{21}d_2}$ and the matrices A_1, A_2, A_3, A_4 are:

$$A_1 = \begin{bmatrix} r - q_1E_1 - ed_1 - d_2r_{12} & 0 \\ r_{21}d_2 & -m - q_2E_1 \end{bmatrix},$$

$$A_2 = \begin{bmatrix} r - q_1E_1 - ed_1 - d_2r_{12} & 0 \\ 0 & -m - q_2E_1 \end{bmatrix},$$

$$A_3 = \begin{bmatrix} r - q_1E_1 & 0 \\ r_{21}d_2 & -m - q_2E_1 \end{bmatrix},$$

$$A_4 = \begin{bmatrix} r - q_1E_1 & 0 \\ 0 & -m - q_2E_1 \end{bmatrix}$$

and the Defuzzification can be represented as:

$$\dot{x}(t) = \sum_{i=1}^r W_i(z(t))(A_i x(t)) \quad (2.9)$$

where -

$$W_1(z(t)) = L_1(z_1(t)) * L_3(z_2(t)), W_2(z(t)) = L_1(z_1(t)) * L_4(z_2(t)),$$

$$W_3(z(t)) = L_2(z_1(t)) * L_3(z_2(t)), W_4(z(t)) = L_2(z_1(t)) * L_4(z_2(t))$$

This Fuzzy model exactly represents the non-linear system (2.2) in the region $[0,0.5] \times [0,0.5]$.

2.6 Results and discussion

In this section, the global stability of the considered intra-specific competition predator-prey model (2.1) is discussed. Because of biological systems are complicated, nonlinear, and unpredictable, fuzzy logical methods with linguistic descriptions should be used to represent them. We have studied the system (2.1) numerically using MATHEMATICA software to get better insight of the proposed model.

Calculations were carried by taking the values of the parameters [85] at $r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, $q_2=0.5$ in (2.3) to get the eigen values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ as explained in the theorems (2.4.1, 2.4.2). It is found that $\max(\lambda_i) = \lambda(\alpha) = -0.50$, then we have chosen $\text{diag}[-0.84, -0.84]$ as impulsive control matrix, such that $\omega = \|I + K\| = 0.16$. It is noted that the system (2.3) is stable globally (2.4.2) when $\epsilon=0.4$, $\delta=0.5$ (at those above values, $\ln(\omega) - \epsilon\delta_j = -2.032 < 0$). Further, it is noted that the predator-prey model is unstable (2.4.1) when $r=3$, $m=3$, $E_1=1$, $e=0.005$, $r_{12}=0.3$, $q_1=0.2$, $q_2=0.5$, $r_{21}=0.1$, $q_1=0.01$, $q_2=0.02$, $d_1=0.5$, $d_2=0.5$, since $\max(\lambda_i) = \lambda(\alpha) = 5.9$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.20 > 0$.

The impact of the various parameters on prey-predator system (2.1) with T-S fuzzy impulsive control model is presented in figs. 2.1 - 2.8.

The dynamical behavior of the two species population (x, y) under the influence of intra-specific competition parameter (e) with impulsive control can be seen in fig. 2.1 at $r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, and $q_2=0.5$ [87]. This figure clearly exhibits that the population of prey decreases with an increase in (e) whereas the predator population reduces to zero.

The dynamical pattern of prey- predator population (x, y) by varying prey max time (d_1) parameter under fuzzy impulsive control can be noted in fig. 2.2 at $r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_2=0.5$, $q_1=0.2$, and $q_2=0.5$. It is observed

from this figure that, the prey population increases and predator population becomes stable (zero) as (d_1) decreases.

The trend of two species (x, y) population with respect to predator max time (d_2) is presented in fig. 2.3 by taking $r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $d_1=0.5$, $e=0.2$, $q_1=0.2$, $q_2=0.5$. It is clear from this figure that, the prey population increases with an increase in predator max time (d_2) .

The impact of interaction parameter (r_{12}) on prey-predator system is shown in fig. 2.4 at $r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, $q_2=0.5$. This figure clearly displays that decrease in the interaction of predator with prey leads to increase in the prey population.

The effectiveness of interaction parameter (r_{21}) on prey-predator population (x, y) is presented in fig. 2.5 by fixing the other parameters at $r=2.5$, $m=3$, $r_{12}=0.3$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, and $q_2=0.5$. This figure shows that, increase in the prey interaction with predator population doesn't have any influence on predator-prey system. This is due to the fact that, interaction level of preys over predators is negligible.

The dynamical behavior of the prey-predator population (x, y) with prey birth rate (r) is explained in fig. 2.6 by taking the other parameters at $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, and $q_2=0.5$. It is found from this figure that, increase in prey birth rate leads to increase in prey population.

The nature of prey-predator (x, y) population with the effect of predator's death rate (m) is shown in fig. 2.7 by considering the values of the other parameters at $r=2.5$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, and $q_2=0.5$. It is clear from this figure that, increase in the death rate of predator population leads to increase in the prey population and decrease in the predator population.

Finally, the nature of two species (x, y) population (without impulsive control) is presented in fig. 2.8 by fixing all the parameters obtained from T-S fuzzy model at

$r=2.5$, $m=3$, $r_{12}=0.3$, $r_{21}=0.1$, $E_1=15$, $e=0.2$, $d_1=0.5$, $d_2=0.5$, $q_1=0.2$, $q_2=0.5$ and initial conditions $x(0) = 5, y(0) = 5, t = 10$. The figure clearly shows how the prey and predator populations reach stability.

2.7 Conclusions

In this chapter we analyzed the stability of a two species competition model with fuzzy impulsive control by T-S fuzzification. The main results of this study are as follows:

- The population of prey increases with an increase in intra-specific competition (e), whereas the predator population reduces to zero.
- The prey population increases and predator population becomes stable (zero) as prey max time (d_1) decreases.
- The prey population is directly proportional to predator max time (d_2).
- Increase in the prey interaction with predator population doesn't have any impact on predator-prey system. This is due to the fact that, interaction level of preys over predators is negligible.
- Effect of prey birth rate is to increase prey population.
- It has been observed that the death rate of predator population leads to increase in the prey population and decrease in the predator population.

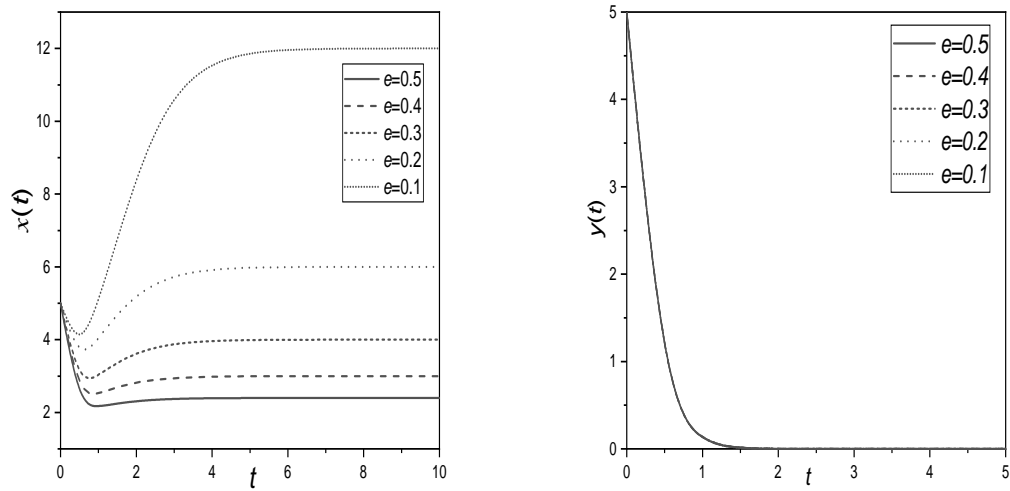


Figure 2.1: Influence of intra-specific competition parameter (e) on prey-predator system under impulsive control

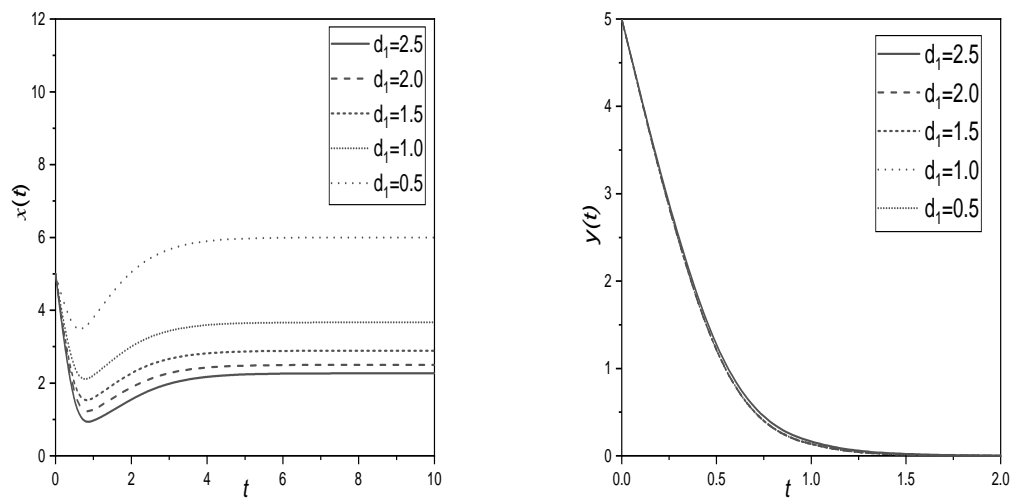


Figure 2.2: Influence of prey max time (d_1) on prey-predator system under impulsive control

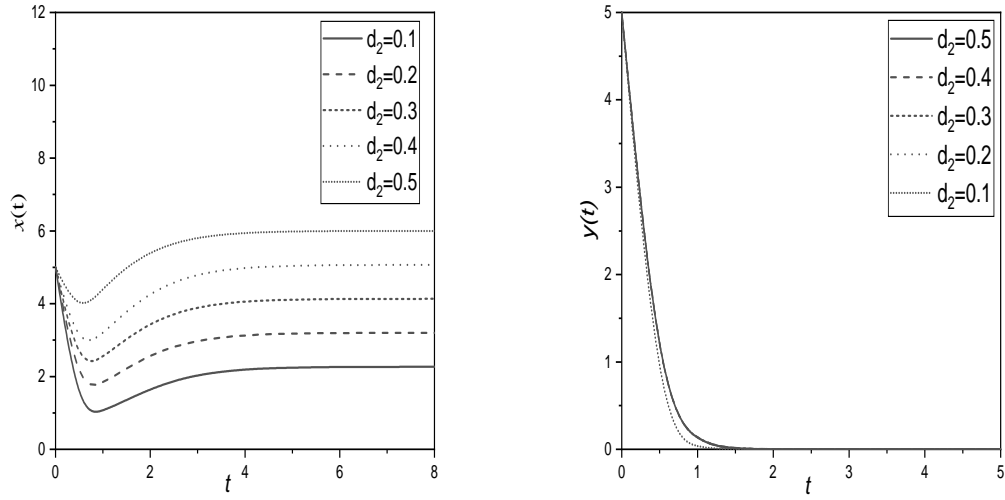


Figure 2.3: Influence of predator max time (d_2) on prey-predator system under impulsive control

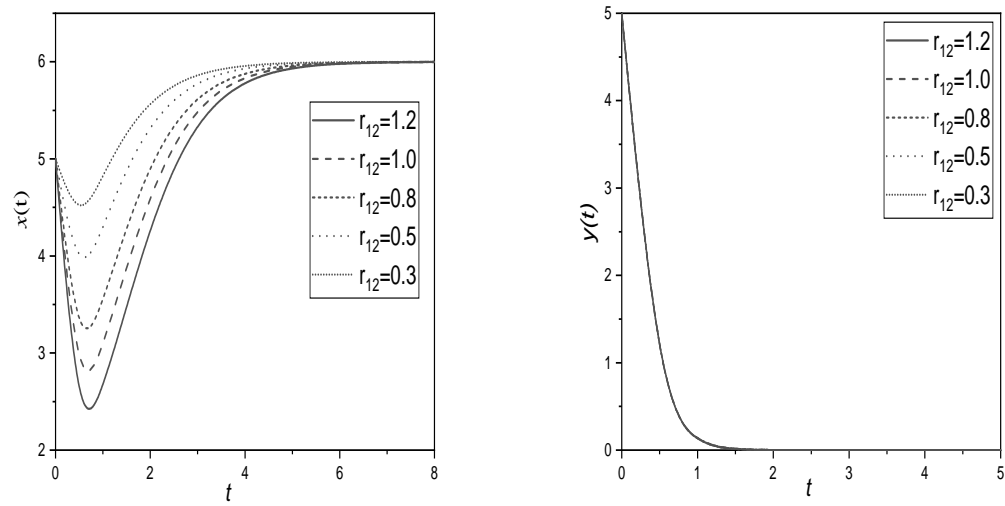


Figure 2.4: Influence of r_{12} on prey-predator system under impulsive control

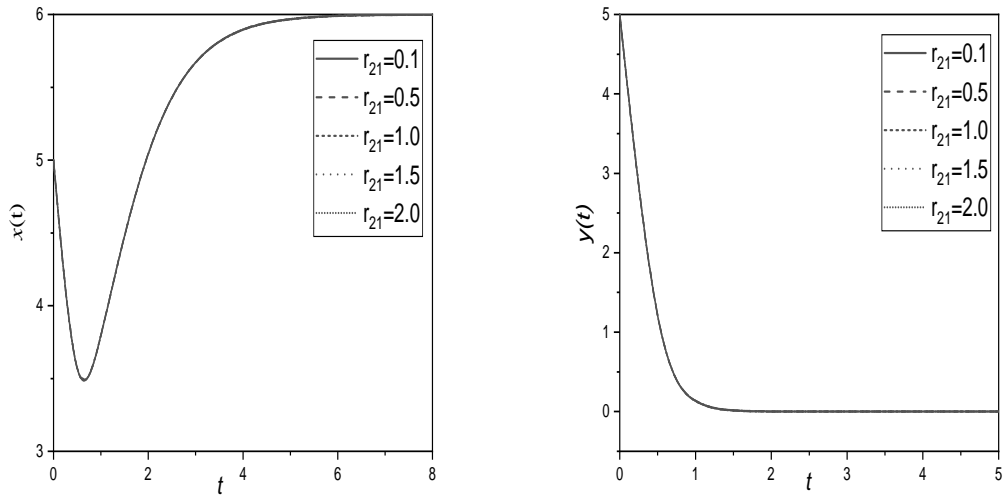


Figure 2.5: Influence of r_{21} on prey-predator system under impulsive control

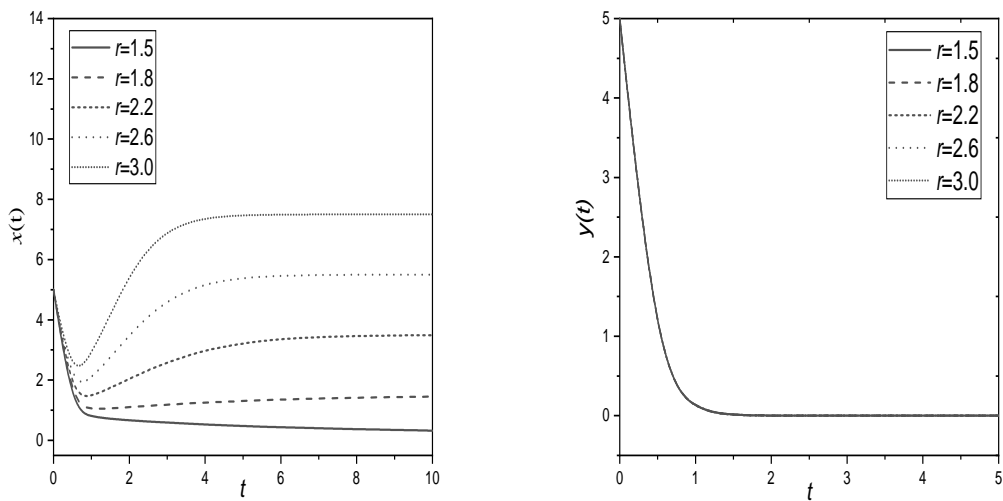


Figure 2.6: Influence of birth rate of prey (r) on prey-predator system under impulsive control

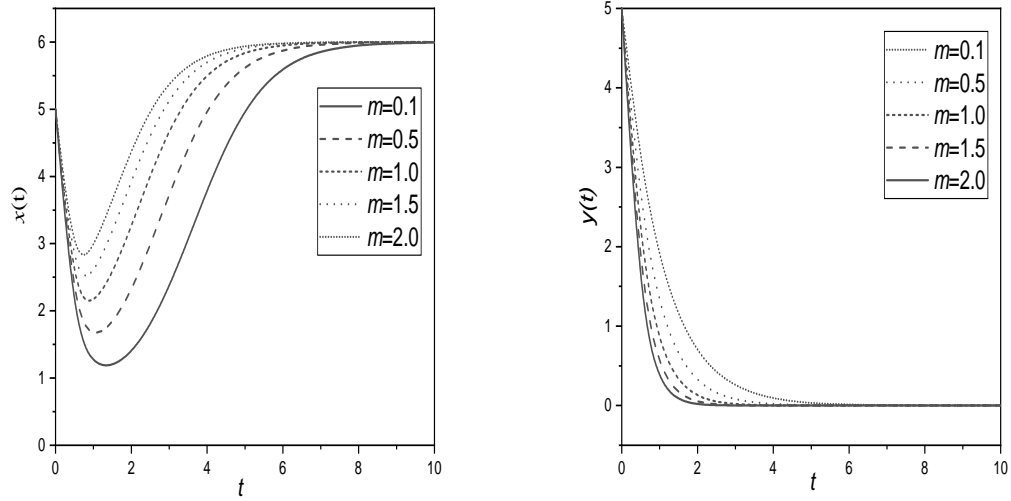


Figure 2.7: Influence of death rate of predator (m) on prey-predator system under impulsive control

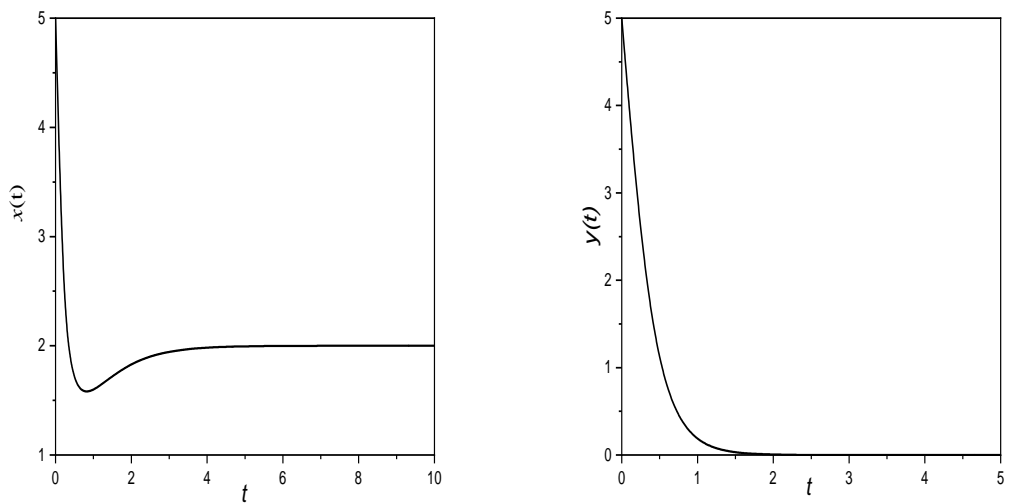


Figure 2.8: Plot of predator-prey system without impulsive control

Chapter 3

Stability Analysis of Prey-Predator model with two preys and one predator using Fuzzy Impulsive Control ¹

3.1 Introduction

For a very long period, theoretical ecology was completely ignored the astounding dynamical behaviors of three-species models. Of course, both theorists and experimenters faced a significant number of new challenges as a result of the growing number of differential equations and dimensions. Additionally, this concept has to be examined because certain three-species communities have recently drawn a lot of interest. Three-species systems, such as two prey, one predator [88, 89, 90, 91], plant, herbivore, parasite, and plant, pest, and predator, are therefore becoming more prevalent in several disciplines of ecology [92, 93, 94].

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The main focus of this chapter is to examine interaction between one predator and two preys. To improve the model's reality we analyze the global and asymptotic stability [82, 95] of this model with the help of the T-S approach, then presented the graphical solutions of the problem.

3.2 Model Formation

Our mathematical model is based on the following assumptions-

- The overall population density of the first and second preys are represented by x_1 and x_2 .
- y is the predator's overall population density.
- In the second population of prey, there is no intra-specific interaction.
- Since the second prey's growth is exponential, there is a huge supply of it when there is no predator around.
- Holling type - II functional response regulates the first prey and predator interaction.
- First prey population grows logistically in the absence of any predator.

Based on the aforementioned hypotheses, the following model is proposed, which includes a set of non-linear differential equations.

$$\begin{aligned}
 \frac{dx_1}{dt} &= r_1x_1 - ex_1^2 - \frac{P_1x_1z}{a + \lambda\alpha x_2 + x_1} + h_1x_1x_2y \\
 \frac{dx_2}{dt} &= r_2x_2 - P_2x_2y + h_2x_1x_2y \\
 \frac{dy}{dt} &= \frac{C_1P_1x_1y}{a + \lambda\alpha x_2 + x_1} + C_2P_2x_2y - my
 \end{aligned} \tag{3.1}$$

where all the parameters are positive with the initial conditions $x_1 = x_{10} > 0$, $x_2 = x_{20} > 0$, $y = y_0 > 0$, x_{10} , x_{20} , y_0 are the initial value of the populations. Here r_1 is intrinsic growth rate of first prey, e is intra-specific competition parameter, r_2 is the intrinsic growth rate of second prey, P_1 is the predation rate of first prey, m is the natural death rate of predator, P_2 is the predation rate of second prey, C_1 is the conversion rate of first prey to predator, C_2 is the conversion rate of second prey to predator, a is the half-saturation constant, λ is the ratio between handling time of the predator per second prey item and handling time of the predator per first prey item, α is the ratio between capture rate of the second prey and capture rate of the first prey, h_1, h_2 are the coefficients of help between two preys.

A matrix differential equation is stated as follows to analyze the system's stability:

$$\dot{x} = Ax + \phi(x) \quad (3.2)$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}_1(t) \\ \dot{x}_2(t) \\ \dot{y}(t) \end{pmatrix}, A = \begin{bmatrix} r_1 & 0 & 0 \\ 0 & r_2 & 0 \\ 0 & 0 & -m \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex_1^2 - \frac{P_1 x_1 y}{a + \lambda \alpha x_2 + x_1} + h_1 x_1 x_2 y \\ -P_2 x_2 y + h_2 x_1 x_2 y \\ \frac{C_1 P_1 x_1 y}{a + \lambda \alpha x_2 + x_1} + C_2 P_2 x_2 y \end{bmatrix}$$

3.3 T-S Fuzzy model with Impulsive effects

3.3.1 lemma

Let $\dot{x} = f(x(t))$, here the state variable is $x(t) \in R^n$, and $f \in C[R^n, R^n]$ fulfills the condition $f(0) = 0$, is a compact vector field defined in $W \subseteq R^n$. Using the techniques proposed by Tanaka and Wang [86]. We can build a fuzzy model for system (2.1) as shown below:

Control Rule i ($i = 1, 2, \dots, \mu$): IF $z_1(t)$ is M_{i1} , $z_2(t)$ is M_{i2} ... and $z_p(t)$ is M_{ip}

THEN $\dot{x}(t) = A_i x(t)$, where μ is no. of T-S fuzzy rules, $z_1(t), z_2, \dots, z_p(t)$ are the premise variables, each M_{ij} is a fuzzy set and $A_i \subseteq R^{n \times n}$ is a constant matrix.

The non-linear equations can be transformed into the following linear equations.

If $x(t)$ is M_i then

$$\begin{aligned} \dot{x}(t) &= A_i x(t), t \neq \tau_j \\ \Delta(x) &= K_{ij} x(t), t = \tau_j \\ i &= 1, 2, 3 \dots r; j = 1, 2, \dots \end{aligned} \quad (3.3)$$

where $A_i = \begin{bmatrix} r_1 - z_1 - P_1 z_2 + h_1 z_3 & 0 & 0 \\ h_2 z_3 & r_2 - z_4 & 0 \\ C_1 P_1 z_2 & C_2 z_4 & -m \end{bmatrix}$, $i=1$ to 4, where the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 4 and z_1, z_2, z_3, z_4 are related to the values of $x_1(t) \in [0, d_1]$, $x_2(t) \in [0, d_2]$, $y(t) \in [0, d_3]$, (here $z_1 = ex_1$, $z_2 = \frac{y}{a + \lambda \alpha x_2 + x_1}$, $z_3 = x_2 y$, $z_4 = P_2 y$). $M_i, x(t), A_i \in R^{3 \times 3}$, r is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta(x)|_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

3.4 Numerical Simulation

By using fuzzy impulsive T-S design model on (3.1), the membership functions as given in [96], obtained as

$$\begin{aligned} M_1 &= \frac{z_1}{ed_1}, M_2 = \frac{ed_1 - z_1}{ed_1}, N_1 = \frac{z_2}{\frac{d_3}{a + \lambda \alpha d_2 + d_1}}, N_2 = \frac{\frac{d_3}{(a + \lambda \alpha d_2 + d_1)} - z_2}{\frac{d_3}{(a + \lambda \alpha d_2 + d_1)}}, K_1 = \frac{z_3}{d_2 d_3}, \\ K_2 &= \frac{d_2 d_3 - z_3}{d_2 d_3}, L_1 = \frac{z_4}{P_2 d_3}, L_2 = \frac{P_2 d_3 - z_4}{d_3}, \text{ and the matrices } A'_i \text{ s are calculated} \\ \text{using } A_i &= \begin{bmatrix} r_1 - z_1 - P_1 z_2 + h_1 z_3 & 0 & 0 \\ h_2 z_3 & r_2 - z_4 & 0 \\ C_1 P_1 z_2 & C_2 z_4 & -m \end{bmatrix}, i = 1 \text{ to } 16, \text{ where the matrices} \end{aligned}$$

A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 4 and, the Defuzzification can be represented as:

$$\dot{x}(t) = \sum_{i=1}^r h_i(z(t))(A_i x(t)) \quad (3.4)$$

here h'_i s are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i=1$ to 16, $j=1$ to 4,

This Fuzzy model exactly represents the non-linear system in the region $[0,5] \times [0,10] \times [0,10]$.

$$\begin{aligned} \frac{dx_1}{dt} &= r_1 x_1 - e x_1^2 - \frac{P_1 x_1 z}{a + \lambda \alpha x_2 + x_1} + h_1 x_1 x_2 y \\ \frac{dx_2}{dt} &= r_2 x_2 - P_2 x_2 y + h_2 x_1 x_2 y \\ \frac{dy}{dt} &= \frac{C_1 P_1 x_1 y}{a + \lambda \alpha x_2 + x_1} + C_2 P_2 x_2 y - m y \end{aligned} \quad (3.5)$$

3.5 Results and discussion

In this section, the global stability of the considered intra-specific competition predator-prey model (3.1) is discussed. We have studied the system (3.1) numerically using MATHEMATICA software to get better insight of the proposed model. Calculations were carried by taking the values of the parameters at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, in (3.3) to get the eigen values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ as explained in the theorems ([83]). It is found that $\max(\lambda_i) = \lambda(\alpha) = 41.6$, then we have chosen $\text{diag}[-0.84, -0.84]$ as impulsive control matrix, such that $\omega = \|I + K\| = 0.01$. It is noted that the system (3.3) is stable globally when $\epsilon=1.5$, $\delta_j=0.1$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -0.039 < 0$). Further, it is observed that the prey-predator model is unstable when $r_1 = 2.5$, $e = 1$, $r_2 = 1$, $C_1 = 0.8$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.4$, $h_2 = 0.1$, $\lambda = 0.2$, $\alpha = 0.3$, $P_1 = 3.5$, $P_2 = 2$, $d_1 = 5$, $d_2 = 10$, $d_3 = 10$, since $\max(\lambda_i) = \lambda(\alpha) = 86.18$,

$$\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 4.419 > 0 \text{ for } \omega = 0.99, \epsilon=1.5, \delta_j=0.1.$$

e	r_1	r_2	P_1	P_2	C_1	C_2	m	a	h_1	h_2	α	λ	d_1	d_2	d_3	$\max(\lambda_i)=\lambda(\alpha)$	$\ln(\epsilon\omega) + \lambda(\alpha)\delta_j$	conclusion
2.5	0.5	0.5	1.5	1.0	0.4	0.1	1.0	1.0	0.2	0.05	0.15	0.1	10	10	10	41.6	-0.039	stable
2.0	0.5	0.5	1.5	1.0	0.5	0.1	1.0	1.0	0.05	0.05	0.1	0.1	10	10	10	13.07	-2.89	stable
1.5	0.1	0.1	2.5	1.0	1.0	0.5	1.0	1.0	0.1	0.2	0.15	0.1	10	10	10	32.56	-0.943	stable
1.0	2.5	1.0	3.5	2.0	0.8	0.1	1.0	1.0	0.4	0.1	0.3	0.2	10	10	10	86.18	4.419	unstable

Table 3.1: Stability of the system at various parameters

Table 3.1 presents the stability of the system at various values of the present study.

The impact of the various parameters on prey-predator system (3.1) with T-S fuzzy impulsive control model is presented in figs. 3.1 - 3.9.

The impact of predation coefficient of second prey (P_2) parameter on prey-predator population (x_1, x_2, y) under fuzzy impulsive control can be noted in fig. 3.1 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure shows that increase in predation coefficient of second prey leads to increase in first prey population and predator population whereas decrease in second prey population.

The change on prey-predator populations (x_1, x_2, y) by varying first prey max time (d_1) is shown in fig. 3.2 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_2 = 10$, $d_3 = 10$. This figure clearly displays that, the second prey population increases as d_1 decreases.

The dynamical change on prey-predator populations (x_1, x_2, y) by varying second prey max time (d_2) on prey-predator system is shown in fig. 3.3 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_3 = 10$. This figure shows that, the second prey and predator population decreases as d_2 increases.

The nature on prey-predator populations (x_1, x_2, y) by varying predator max time (d_3) is shown in fig. 3.4 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$. It is observed from this figure that, as d_3 decreases, the first and second prey population decreases whereas predator population increases.

The effect of coefficient of help between prey (h_1) on prey-predator system is shown in fig. 3.5 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure clearly displays that decrease in h_1 leads to decrease in first prey population.

The effectiveness by varying predation coefficient of first prey (P_1) parameter on prey- predator population (x_1, x_2, y) under fuzzy impulsive control can be noted in fig. 3.6 at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure clearly displays that increase in predation coefficient of first prey leads to increase in first prey population.

The effect of intra-specific competition (e) on prey-predator system is shown in fig. 3.7 at $r_1 = 0.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure clearly displays that decrease in intra-specific competition between prey-predator leads to increase in second prey population.

The effect of intrinsic growth rate of second prey (r_2) on prey-predator system is shown in fig. 3.8 at $r_1 = 0.5$, $e = 2.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$, $\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure shows that an increase in r_2 leads to increase in the second prey population.

Finally, the nature of three species (x_1, x_2, y) population (without impulsive control) is presented in fig. 3.9 by fixing all the parameters obtained from T-S fuzzy model at $r_1 = 0.5$, $e = 2.5$, $r_2 = 0.5$, $C_1 = 0.4$, $C_2 = 0.1$, $m = 1$, $h_1 = 0.2$, $h_2 = 0.05$,

$\lambda = 0.1$, $\alpha = 0.15$, $P_1 = 1.5$, $P_2 = 1$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, and initial conditions $x_1(0) = 5$, $x_2(0) = 10$, $y(0) = 10$, and $t = 10$. The figure clearly shows how the prey and predator populations reach stability.

3.6 Conclusions

In this chapter, we have constructed a mathematical model of two prey one predator population. The main results of this study are as follows:

- The effect of intra-species competition is to decrease the rate of population growth as population density increases.
- An increase in the prey growth rate causes a rise in the prey population.
- Predator population grows as predator maximum time decreases, but prey population decreases.
- The second prey max time (d_2) is inversely proportional to the second prey and predator population.
- Growth in the predation of second prey, leads to growth in the population of first prey and reduction in the population of second prey.
- As predator max time (d_3) rises, the first prey population rises and the predator population declines.
- Enhance in the help between two preys leads to increase in the population of first prey.
- Rise in the predation of first prey, leads to rise in the population of first prey.

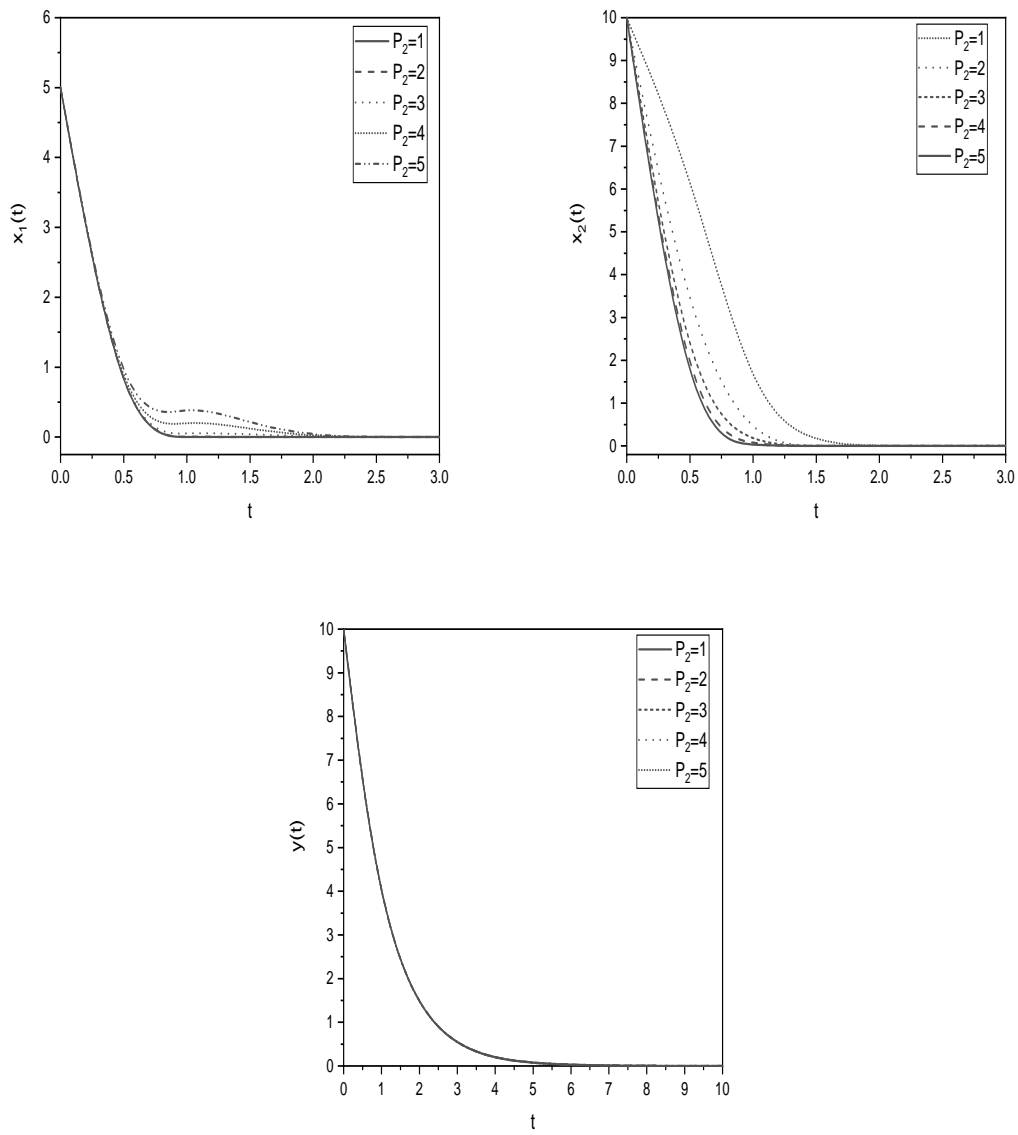


Figure 3.1: Effect of predation coefficient of second prey (P_2) on prey-predator system under impulsive control

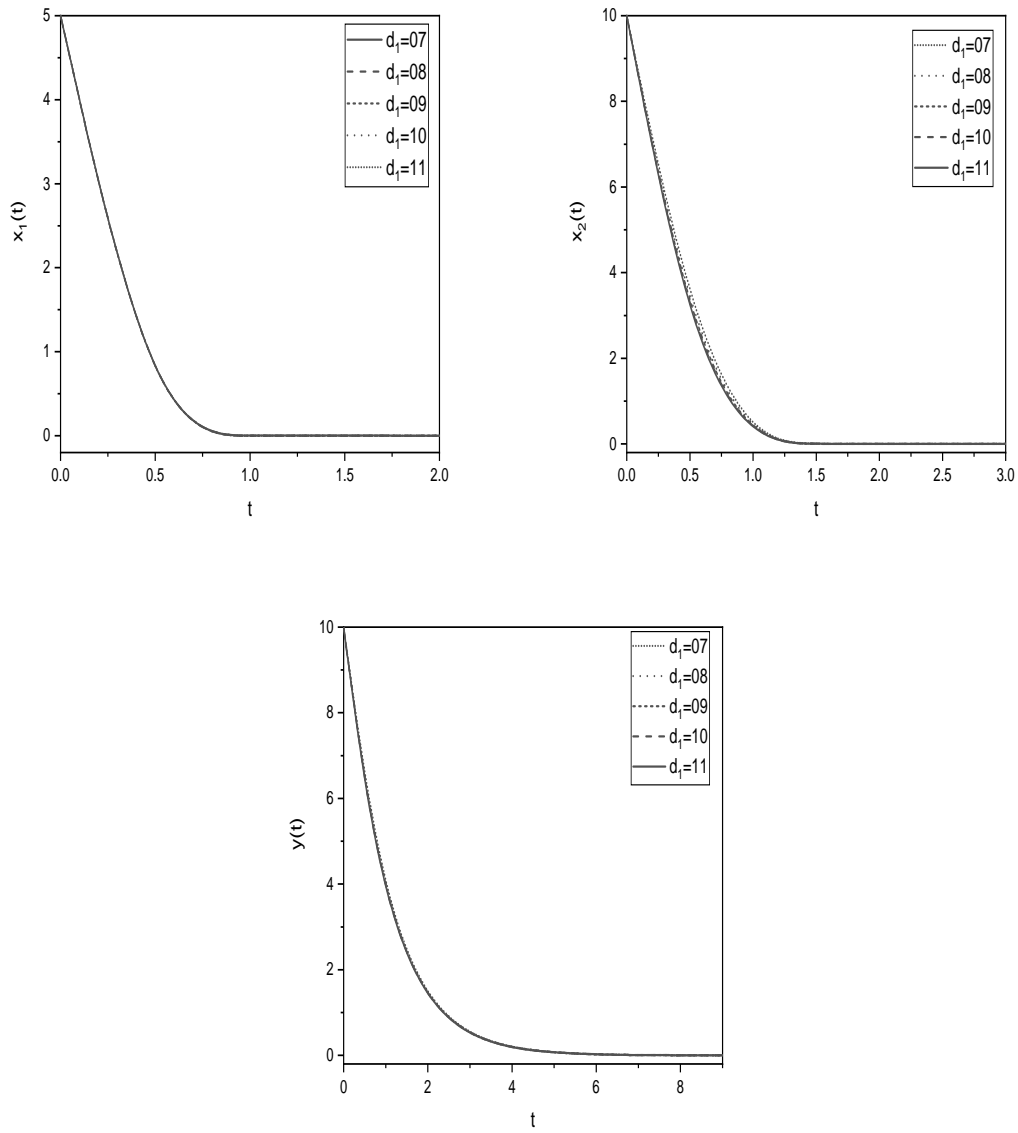


Figure 3.2: Effect of first prey max time (d_1) on prey-predator system under impulsive control

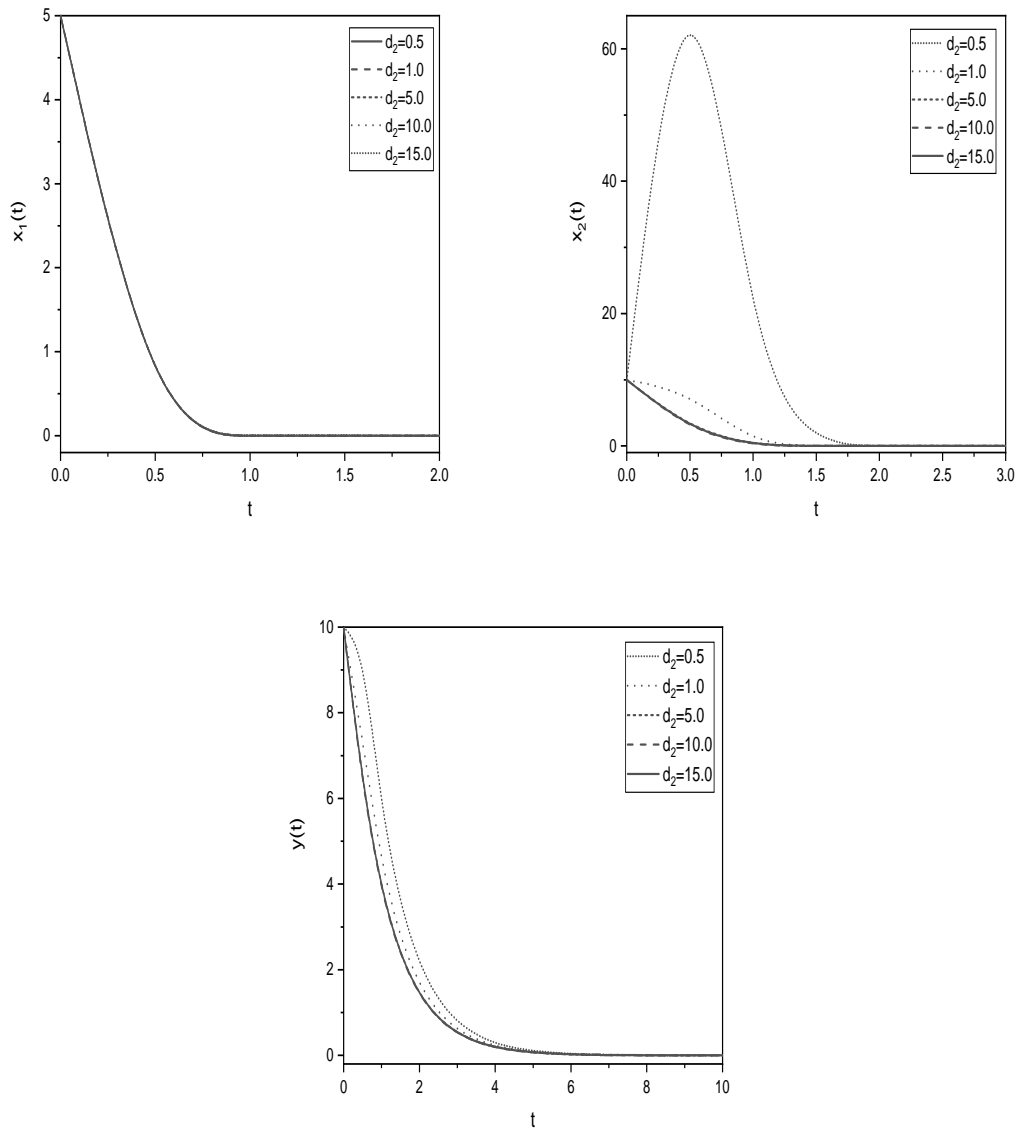


Figure 3.3: Effect of second prey max time (d_2) on prey-predator system under impulsive control

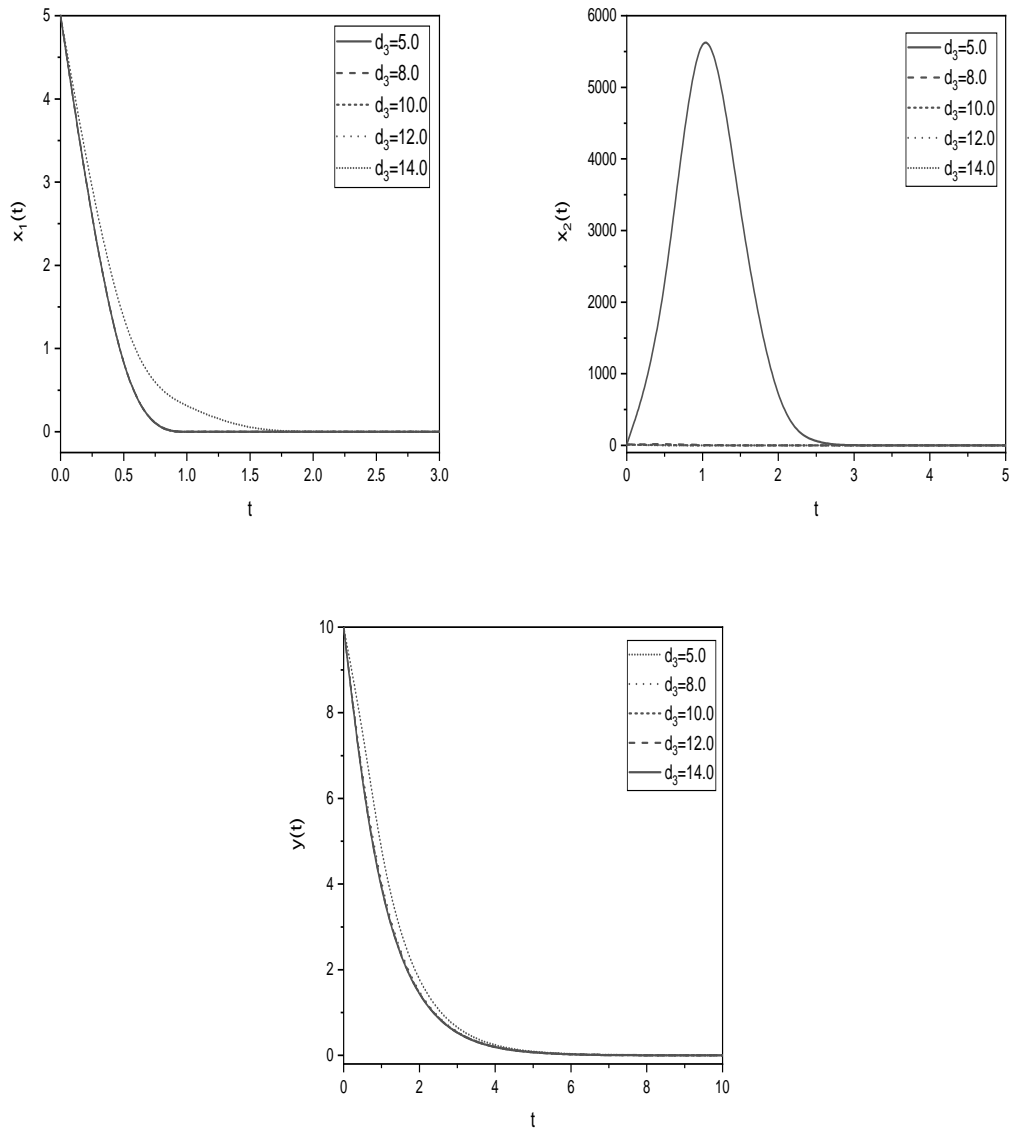


Figure 3.4: Effect of predator max time (d_3) on prey-predator system under impulsive control

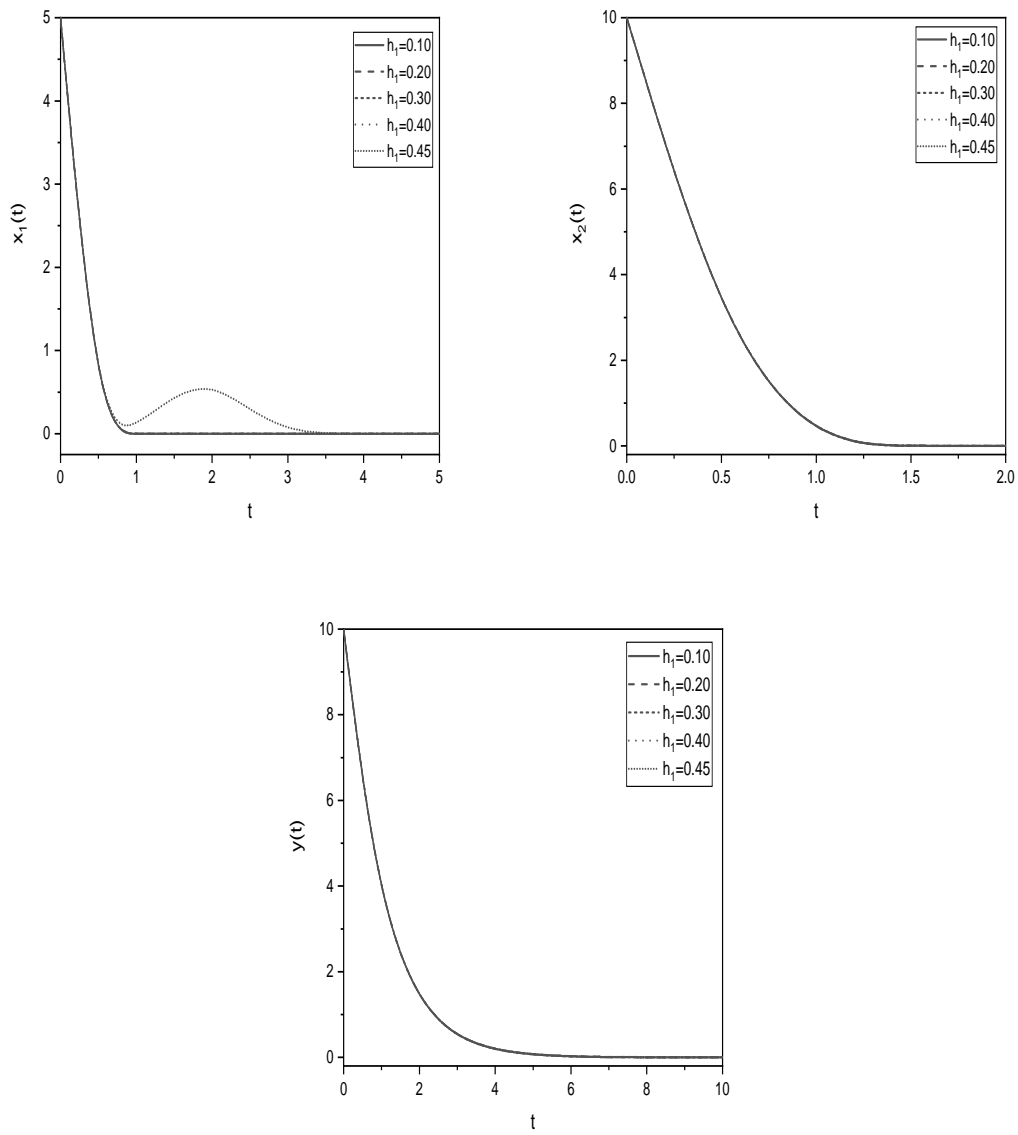


Figure 3.5: Effect of coefficient of help between prey (h_1) on prey-predator system under impulsive control

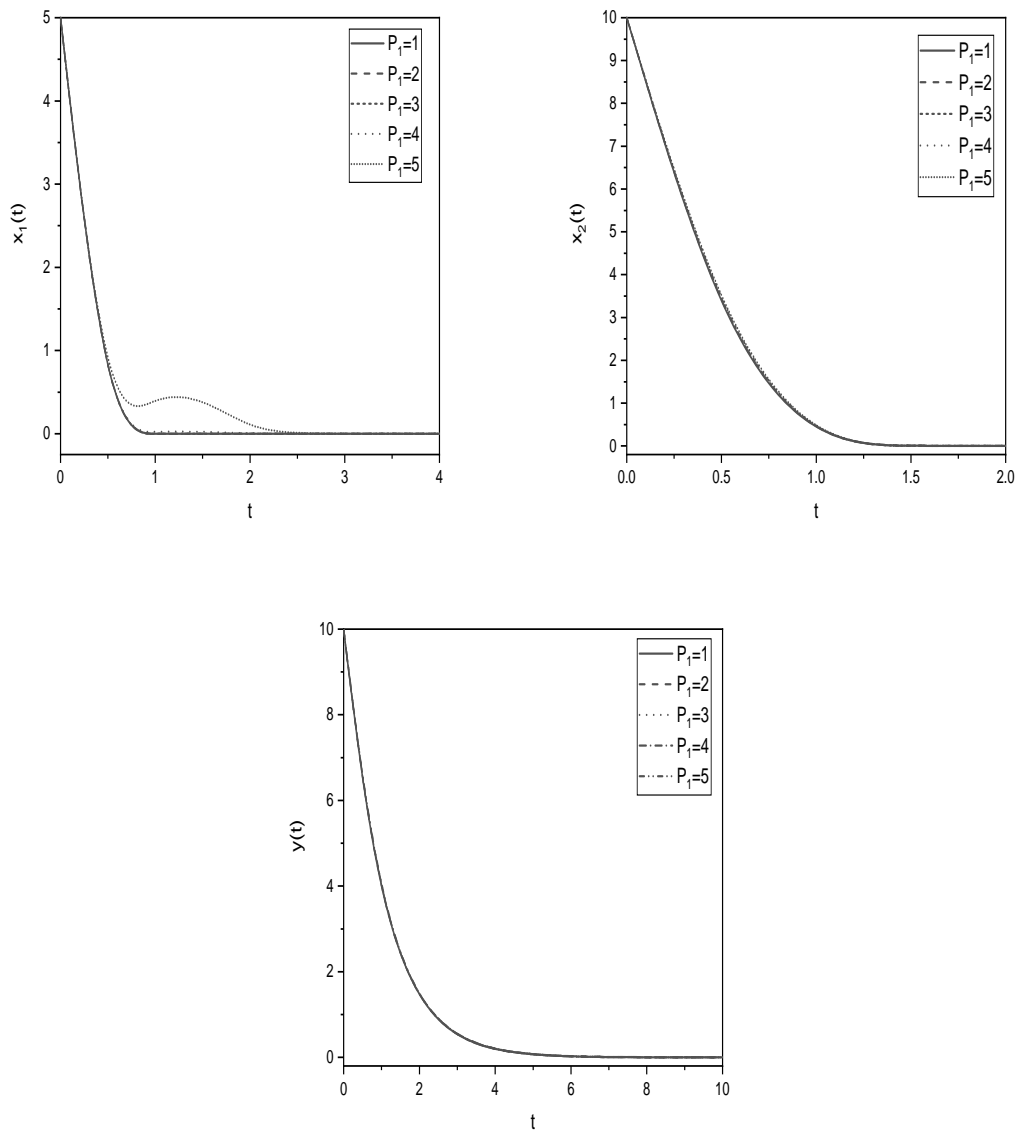


Figure 3.6: Effect of predation coefficient of first prey (P_1) on prey-predator system under impulsive control

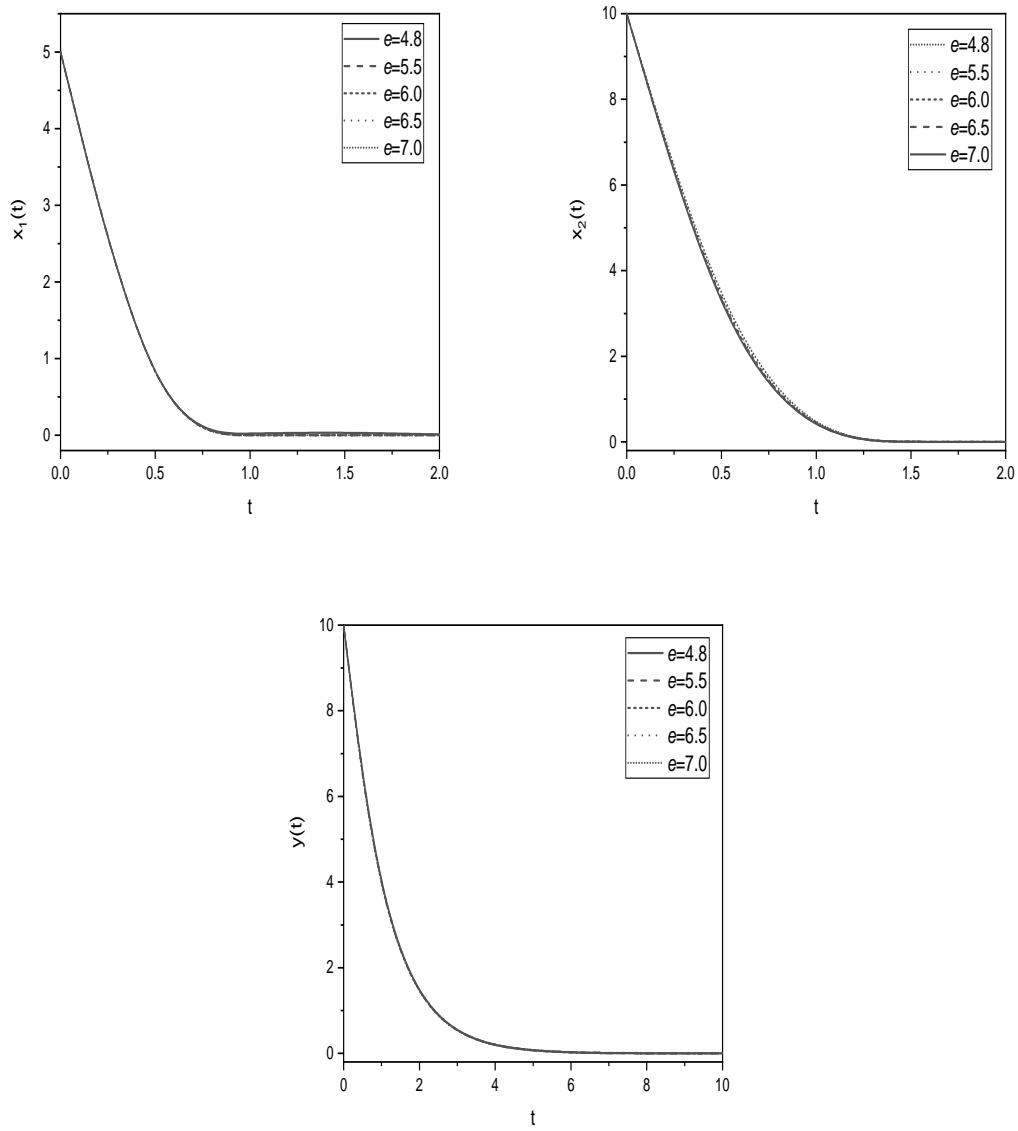


Figure 3.7: Effect of intra-specific competition (e) on prey-predator system under impulsive control

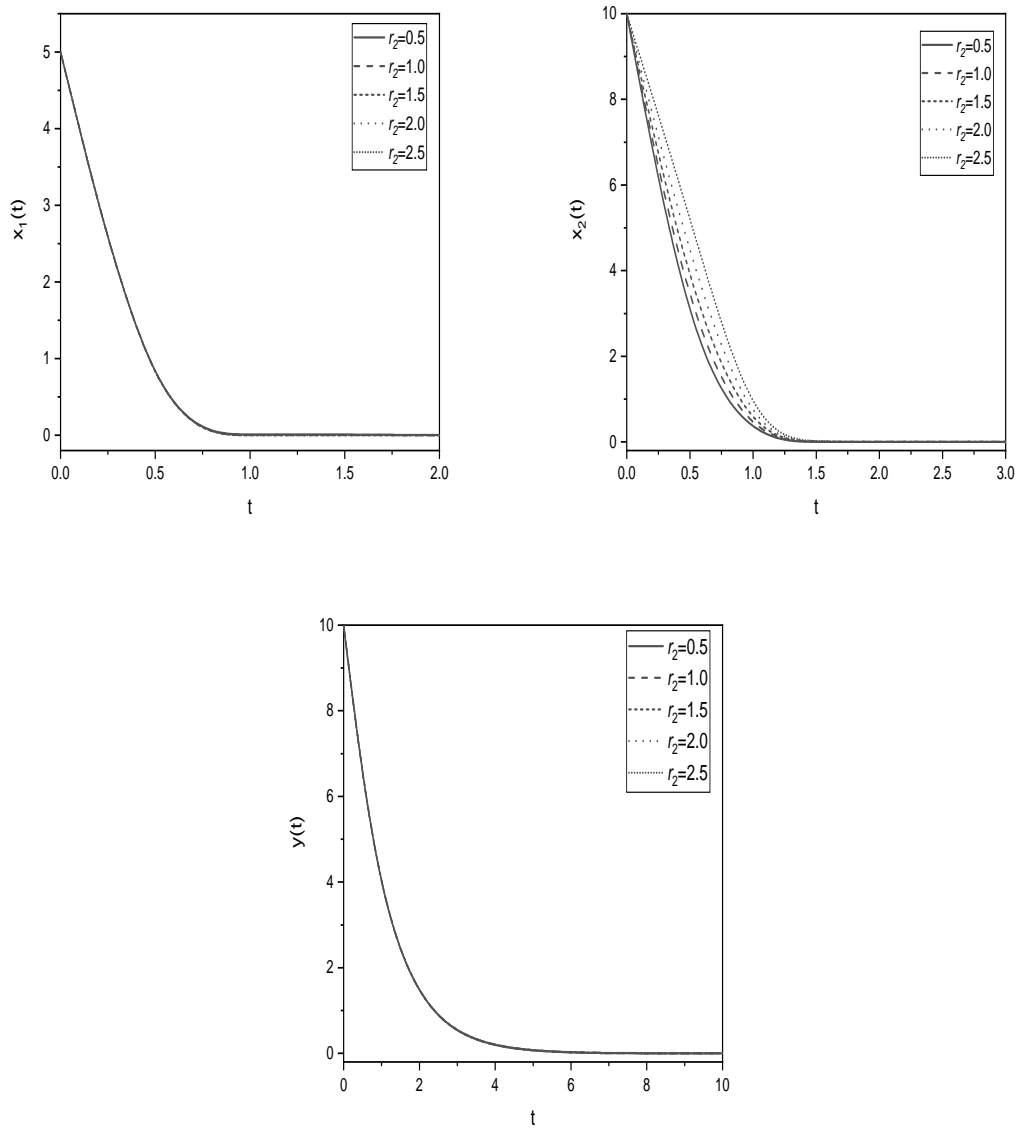


Figure 3.8: Effect of intrinsic growth rate of second prey (r_2) on prey-predator system under impulsive control

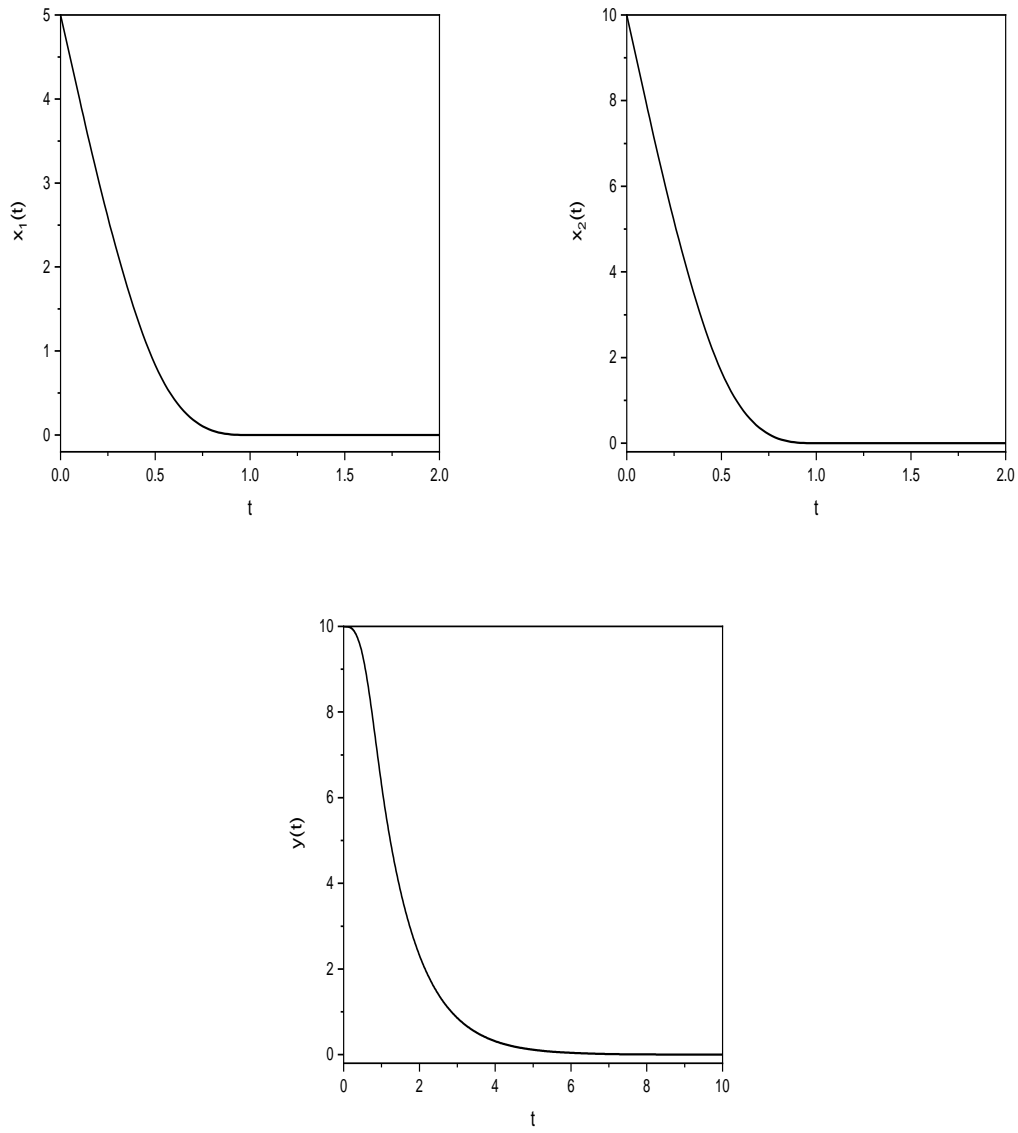


Figure 3.9: plot of predator-prey system without impulsive control

Chapter 4

Population Dynamic study of interaction between two Predators and one Prey ¹

4.1 Introduction

In recent years, the interest in fuzzy control has grown significantly. This has been largely stimulated by the fuzzy control has had in its many applications. It has been made known that numerous fundamental concerns still need to be resolved despite the apparent achievement. The validity and application of any control design approach depend on a number of factors, including stability analysis, systematic design, and performance comparison [97, 98].

In the previous chapter, we have with two preys and one predator model. In this chapter, we take into Lotka-Volterra predator-prey model with one prey and two predators. We examine the global and asymptotic stability to strengthen the reality of the model as given in [82, 95] by means of the T-S model, then provided the graphical representations for the problem by examination. The stability of the

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Lotka-Volterra predator-prey system with fuzzy impulse control has not yet been extensively studied in any literature. Therefore, with the help of fuzzy impulse control and the T-S mathematical model, the stability of the prey-predator system is studied [99, 100, 101].

4.2 Model Formulation

These presumptions serve as the foundation for our mathematical model.

- The total population density of the prey is indicated by x .
- Let the first predator overall population density is denoted by y_1 .
- The second predator overall population density is denoted by y_2 .

Based on the above considerations, we propose the following model using the system of non-linear differential equations.

$$\begin{aligned}\frac{dx}{dt} &= rx - ex^2 - \frac{P_1 y_1 x}{a_0 + x} - \frac{P_2 y_2 x}{a_0 + x} \\ \frac{dy_1}{dt} &= \frac{C_1 P_1 y_1 x}{a_0 + x} - \frac{C_1 P_1 y_1 y_2}{a_1 + x} - m_1 y_1 \\ \frac{dy_2}{dt} &= \frac{C_2 P_2 y_2 x}{a_0 + x} - \frac{C_2 P_2 y_1 y_2}{a_2 + x} - m_2 y_2\end{aligned}\tag{4.1}$$

where all the variables $(r, e, P_1, P_2, C_1, C_2, m_1, m_2, a_0, a_1, a_2) > 0$ and x_0, y_{10}, y_{20} are initial populations with $x = x_0 > 0, y_1 = y_{10} > 0, y_2 = y_{20} > 0$. Here, r is the internal production growth rate of prey, e represents the intra-species competition, first predator's predation rate is P_1 , second predator's predation rate is P_2 , C_1 is the first predator conversion rate after eating prey, C_2 is the second predator conversion rate after eating prey, first predator's mortality rate is m_1 , second predator's mortality rate is m_2 , and a_0, a_1 , and a_2 are the half-saturation constants.

The following describes a matrix differential equation, to analyze the system's stability:

$$\dot{x} = Ax + \phi(x) \quad (4.2)$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}(t) \\ \dot{y}_1(t) \\ \dot{y}_2(t) \end{pmatrix}, A = \begin{bmatrix} r & 0 & 0 \\ 0 & -m_1 & 0 \\ 0 & 0 & -m_2 \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex^2 - \frac{P_1 y_1 x}{a_0 + x} - \frac{P_2 y_2 x}{a_0 + x} \\ \frac{C_1 P_1 y_1 x}{a_0 + x} - \frac{C_1 P_1 y_1 y_2}{a_1 + x} \\ \frac{C_2 P_2 y_2 x}{a_0 + x} - \frac{C_2 P_2 y_1 y_2}{a_2 + x} \end{bmatrix}$$

4.3 Fuzzy Takagi-Sugeno approach with impacts of impulse

The non-linear equations can be transformed into the following linear equation as explained in the earlier (chapter-2).

If $x(t)$ is M_i then

$$\dot{x}(t) = A_i x(t), t \neq \tau_j \quad (4.3)$$

$$\Delta(x) = K_{ij} x(t), t = \tau_j \quad (4.4)$$

$$i = 1, 2, 3 \dots r; j = 1, 2, \dots \quad (4.5)$$

$$\text{where } A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 & 0 & 0 \\ C_1 z_2 & -C_1 z_4 - m_1 & 0 \\ C_2 z_3 & -C_2 z_5 & -m_2 \end{bmatrix}, i = 1 \text{ to } 31, \text{ where the ma-}$$

trices A_i 's are generated using maximum and minimum values of z'_k 's; $k = 1$ to 5 and z_1, z_2, z_3, z_4, z_5 are related to the values of $x(t) \in [0, d_1], y_1(t) \in [0, d_2], y_2(t) \in [0, d_3]$ (here $z_1 = ex, z_2 = \frac{P_1 y_1}{a_0 + x}, z_3 = \frac{P_2 y_2}{a_0 + x}, z_4 = \frac{P_1 y_2}{a_1 + x}, z_5 = \frac{P_2 y_2}{a_2 + x}$) . $M_i, x(t), A_i$

$\in R^{3 \times 3}$, r is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulse instant, $\Delta(x) |_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

4.4 Numerical Simulations

The membership functions for the fuzzy impulse (4.2) Takagi-Sugeno design model were produced as follows [96]:

$$\begin{aligned} M_1 &= \frac{z_1}{ed_1}, M_2 = \frac{ed_1 - z_1}{ed_1}, N_1 = \frac{z_2}{\frac{P_1 d_2}{a_0 + d_1}}, N_2 = \frac{\frac{P_1 d_2}{a_0 + d_1} - z_2}{\frac{P_1 d_2}{a_0 + d_1}}, K_1 = \frac{z_3}{\frac{P_2 d_3}{a_0 + d_1}}, \\ K_2 &= \frac{\frac{P_2 d_3}{a_0 + d_1} - z_3}{\frac{P_2 d_3}{a_0 + d_1}}, L_1 = \frac{z_4}{\frac{P_1 d_3}{a_1 + d_1}}, L_2 = \frac{\frac{P_1 d_3}{a_1 + d_1} - z_4}{\frac{P_1 d_3}{a_1 + d_1}}, O_1 = \frac{z_5}{\frac{P_2 d_3}{a_2 + d_1}}, O_2 = \frac{\frac{P_2 d_3}{a_2 + d_1} - z_5}{\frac{P_2 d_3}{a_2 + d_1}} \end{aligned}$$

and the matrices A'_k s are calculated using

$$A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 & 0 & 0 \\ C_1 z_2 & -C_1 z_4 - m_1 & 0 \\ C_2 z_3 & -C_2 z_5 & -m_2 \end{bmatrix}, i = 1 \text{ to } 31,$$

where the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 5 and, the Defuzzification is characterized by:

$$\dot{x}(t) = \sum_{k=1}^r h_k(z(t))(A_k x(t)) \quad (4.6)$$

here h'_i s are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i=1$ to 31 , $j=1$ to 5

This Fuzzy model is a suitable representation of the non-linear system in the region $[0,5] \times [0,10] \times [0,10]$.

$$\begin{aligned}
\frac{dx}{dt} &= rx - ex^2 - \frac{P_1 y_1 x}{a_0 + x} - \frac{P_2 y_2 x}{a_0 + x} \\
\frac{dy_1}{dt} &= \frac{C_1 P_1 y_1 x}{a_0 + x} - \frac{C_1 P_1 y_1 y_2}{a_1 + x} - m_1 y_1 \\
\frac{dy_2}{dt} &= \frac{C_2 P_2 y_2 x}{a_0 + x} - \frac{C_2 P_2 y_1 y_2}{a_2 + x} - m_2 y_2
\end{aligned} \tag{4.7}$$

4.5 Results and discussion

This section describes the global stability of the considered intra-species predator-prey competition model (4.2). Given that they are complex, nonlinear, and unpredictable, natural systems should be characterized using a fuzzy logical technique combined with communication description. We have studied the system (4.1) numerically using MATHEMATICA software to get better insight of the proposed model.

The calculations were performed by taking the parameter values, at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $C_1 = 2.0$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $P_2 = 2.0$, $d_2 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$, $d_3 = 10$, $C_2 = 1.5$, in (4.3) to find the eigen values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ as stated in the theorems ([83]). We discover that $\max(\lambda_i) = \lambda(\alpha) = 4.0$, then we decided that $\text{diag}[-0.84, -0.84]$ as impulse control matrix, such that $\omega = \|I + K\| = 0.16$. The system is acknowledged that (4.3) is stable globally when $\epsilon = 2.8$, $\delta_j = 0.12$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -0.3229 < 0$). Additionally, it is noted that the prey-predator model is unstable when $r = 7.0$, $e = 3.0$, $P_1 = 2.0$, $P_2 = 3.0$, $C_1 = 6.0$, $C_2 = 1.0$, $m_1 = 1.0$, $m_2 = 1.5$, $a_0 = 15$, $a_1 = 10$, $a_2 = 5.0$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, since $\max(\lambda_k) = \lambda(\alpha) = 14.0$, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.877 > 0$ for $\omega = 0.16$, $\epsilon = 2.8$, $\delta_j = 0.12$.

The stability of the system for various study-related parameters is shown in Table 4.1

Figures 4.1 - 4.10 show how different factors affect prey-predator system 4.1 with

r	e	P_1	P_2	C_1	C_2	m_1	m_2	a_0	a_1	a_2	d_1	d_2	d_3	$\max(\lambda_k)=\lambda(\alpha)$	$\ln(\epsilon\omega) + \lambda(\alpha)\delta_l$	conclusion
2.0	0.05	1.0	2.0	2.0	1.5	1.0	0.7	10.0	10.0	20.0	10.0	10.0	10.0	4.0	-0.3229	stable
3.3	0.05	1.0	2.0	2.0	1.5	1.0	0.7	10.0	10.0	20.0	10.0	10.0	10.0	6.6	-0.0109	stable
3.0	0.5	1.0	1.0	2.0	1.5	1.5	0.5	8.0	10.0	5.0	10.0	10.0	10.0	6.0	-0.0829	stable
7.0	3.0	2.0	3.0	6.0	1.0	1.0	1.5	15.0	10.0	5.0	10.0	10.0	10.0	14.0	0.877	unstable

Table 4.1: System stability under diverse conditions

T-S Fuzzy impulse Control Model.

Fig 4.1 depicts how intra-species competition (e) affects the prey-predator population at $r = 2.0$, $P_1 = 1.0$, $P_2 = 2.0$, $d_2 = 10$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$, $d_3 = 10$. The effect of intra-species competition is to decrease the rate of population growth as population density increases.

The effect of changing the prey maximum time (d_1) on the prey-predator population is depicted in fig. 4.2 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_3 = 10$. This figure clearly shows that, first predator population increases as d_1 increases because there is more food for predators.

Fig 4.3 illustrates the dynamic shift on the prey-predator population caused by adjusting the first predator maximum time (d_2) in the prey-predator system $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $P_2 = 2.0$, $d_3 = 10$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$. This graph demonstrates how the predator population rises when d_2 falls.

The performance of prey-predator population (x, y_1, y_2) by varying second predator max time (d_3) is shown in fig. 4.4 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$. This graph shows that the predator population increases as d_3 declines.

The changes on prey-predator system with varying half saturation constant (a_0) is given in fig. 4.5 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $d_3 = 10$, $P_2 = 2.0$,

$C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$. This figure clearly displays that decrease in half saturation constant (a_0) indicate that predators can achieve half of their maximum consumption rate at a lower prey density, which means they are more efficient in utilizing the preys and hence predator population rises.

The consequence of half saturation constant (a_1) on prey-predator population is shown in fig. 4.6 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_2 = 20$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This figure clearly displays that the drop in half saturation constant (a_1) leads to increase in predator population.

The impact of half saturation constant (a_2) on prey-predator population is given in fig. 4.7 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $d_1 = 10$, $d_3 = 10$. This figure clearly exhibit that decrease in half saturation constant (a_2) leads to increase in predator population.

The impact of mortality rate of first predator (m_1) on prey-predator population is shown in fig. 4.8 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $d_3 = 10$, $P_2 = 2.0$, $C_2 = 1.5$, $m_2 = 0.7$, $a_0 = 10$, $C_1 = 2.0$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$. This figure clearly displays that decrease in mortality rate of first predator causes rise in first predator population.

The impact of mortality rate of second predator (m_2) on prey-predator population is shown in fig. 4.9 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $d_2 = 10$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$, $d_3 = 10$. This figure clearly displays that decrease in mortality rate of second predator causes an increase in predator population.

Fig 4.10 illustrates how changing the first predator's (P_1) predation coefficient affected the population of prey and predators in the fuzzy impulse control at $r = 2.0$,

$e = 0.05$, $P_2 = 2.0$, $C_1 = 2.0$, $C_2 = 1.5$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_1 = 10$, $a_2 = 20$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This graph clearly displays that rise in predation coefficient of first predator causes to increase in first predator population.

By setting all of the variables from the Takagi-Sugeno fuzzy model, the nature of three species' populations (without impulse control) is finally depicted in fig 4.11 at $r = 2.0$, $e = 0.05$, $P_1 = 1.0$, $C_2 = 1.5$, $P_2 = 2.0$, $C_1 = 2.0$, $m_1 = 1.0$, $m_2 = 0.7$, $a_0 = 10$, $a_2 = 20$, $d_1 = 10$, $a_1 = 10$, $d_2 = 10$, $d_3 = 10$ when $x(0) = 5$, $y_1(0) = 10$, $y_2(0) = 10$, and $t = 10$. This graph clearly exhibit how populations of prey and predators achieve to their stability.

4.6 Conclusions

In this study, a prey-predator population model is developed with two predator and one prey population. The main findings of this study are as follows:

- The population of predators increases as intra-specific competition declines.
- Predators become less in number as prey first predator max time (d_2) and second predator max time (d_3) increase.
- The half-saturation constants are inversely proportional to predators population.
- Decrease in mortality rate causes an increase in predator population.

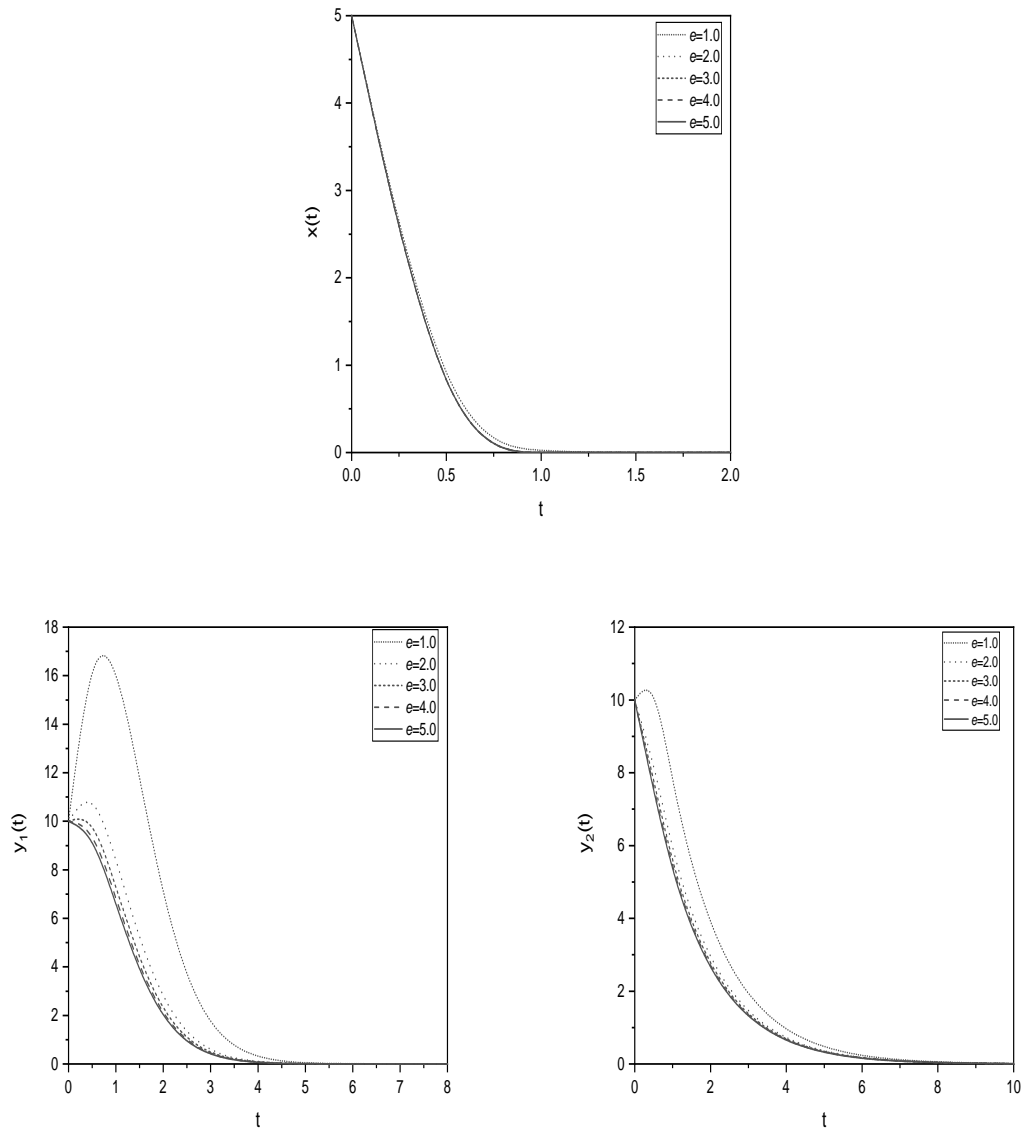


Figure 4.1: Figure depicting intra-species competition (e) effects on the prey-predator population while impulse control is present

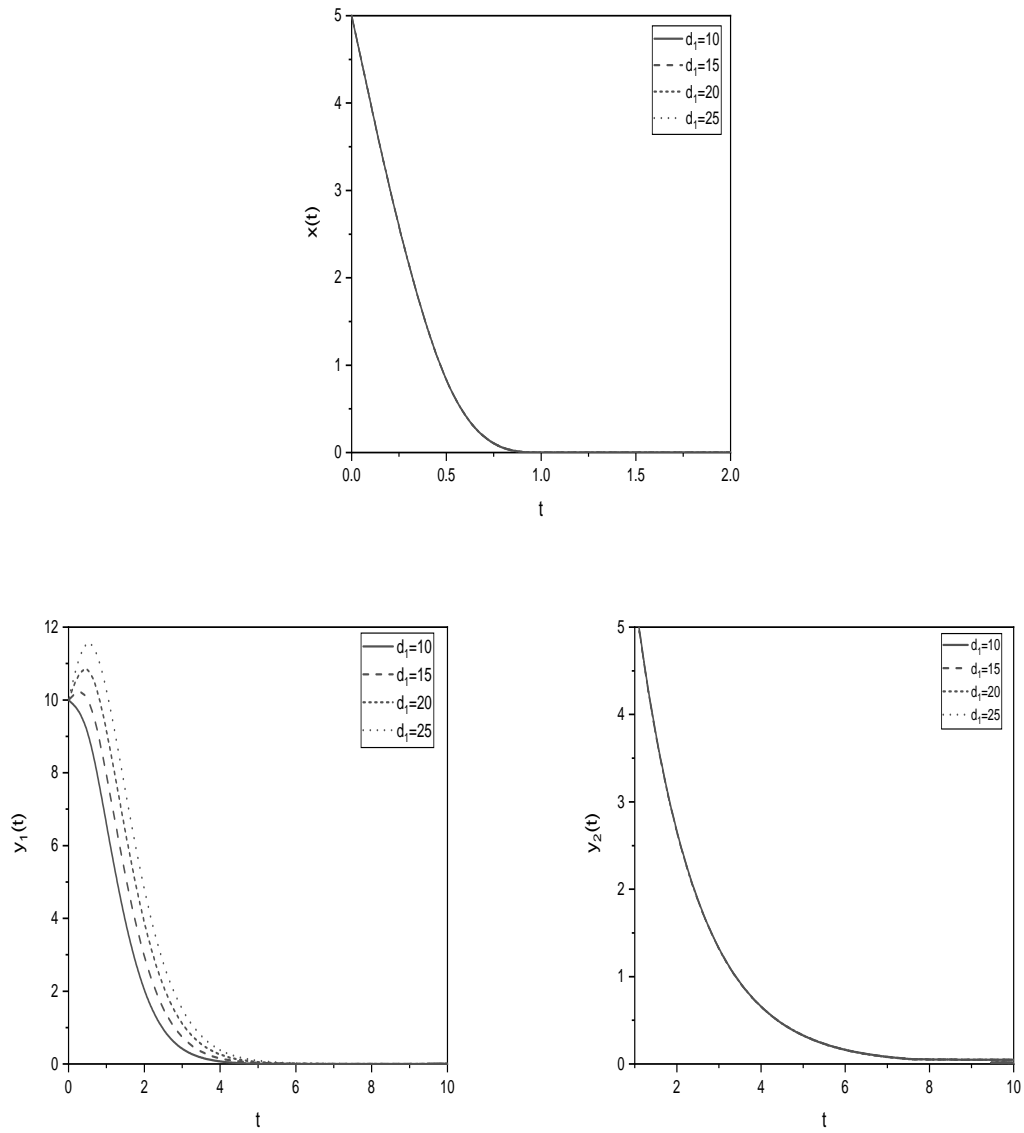


Figure 4.2: Figure depicting the prey max time (d_1) impact on the prey-predator system while impulse control is present

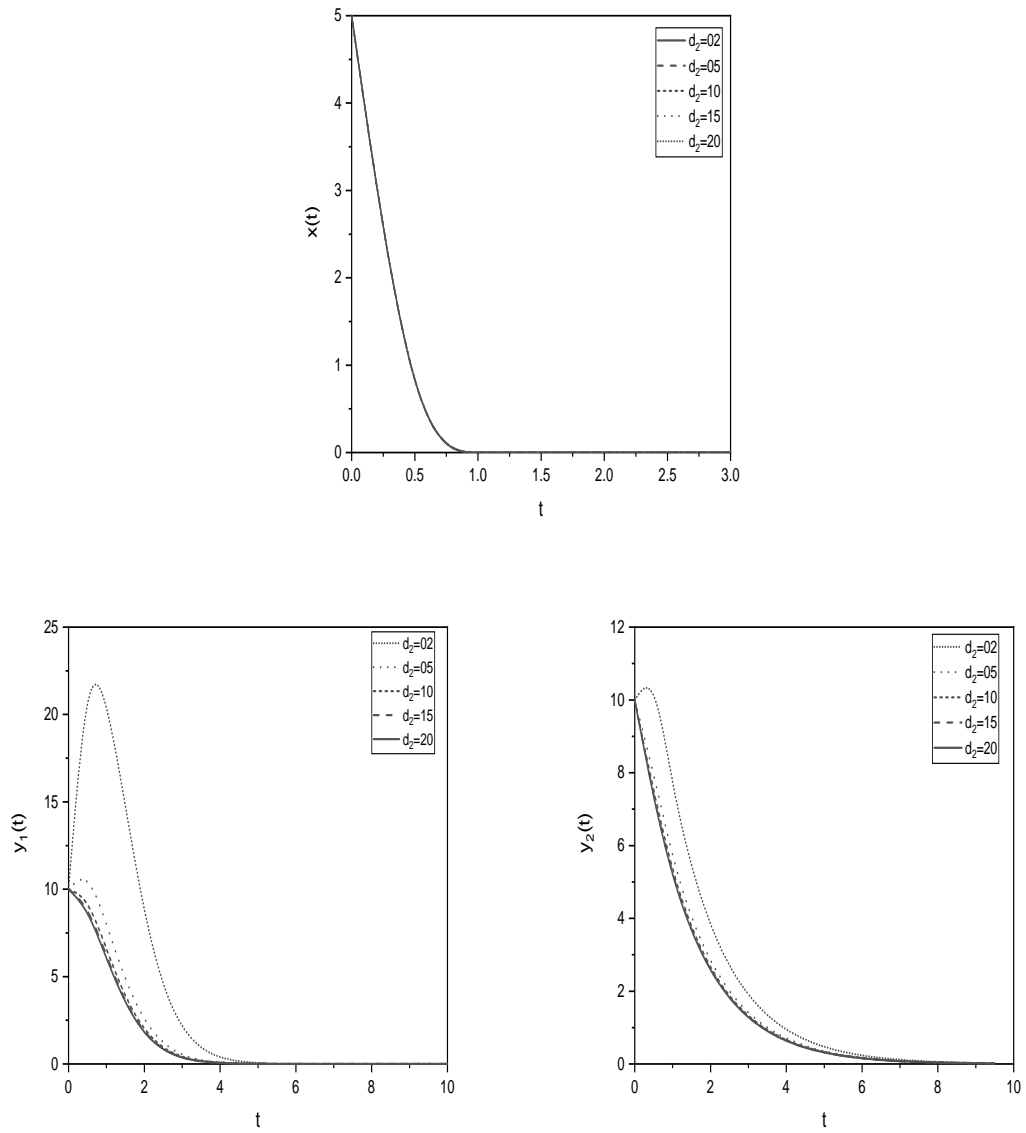


Figure 4.3: Figure depicting the impact of first predator max time (d_2) on prey-predator population while impulse control is present

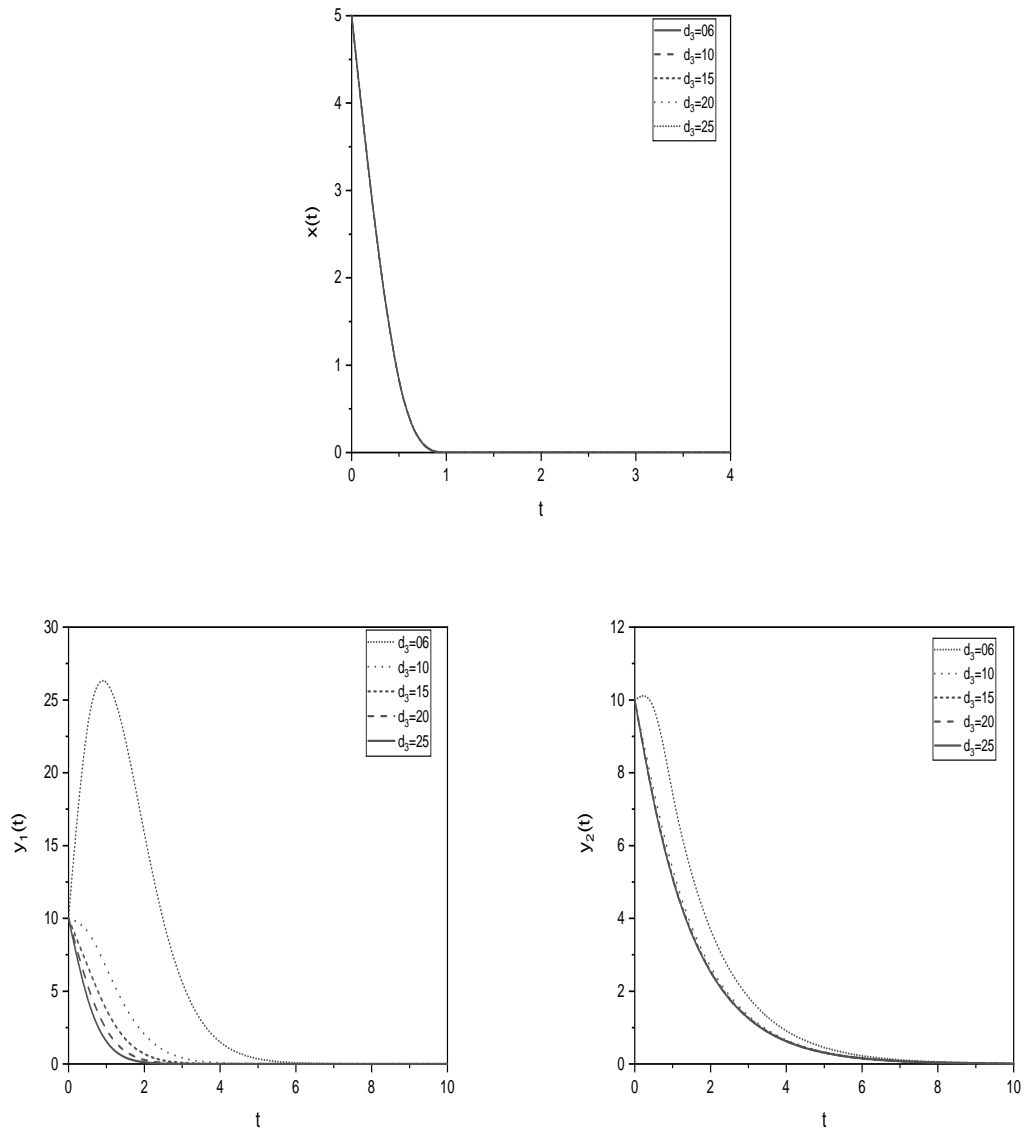


Figure 4.4: Figure depicting the impact of second predator max time (d_3) on prey-predator population while impulse control is present

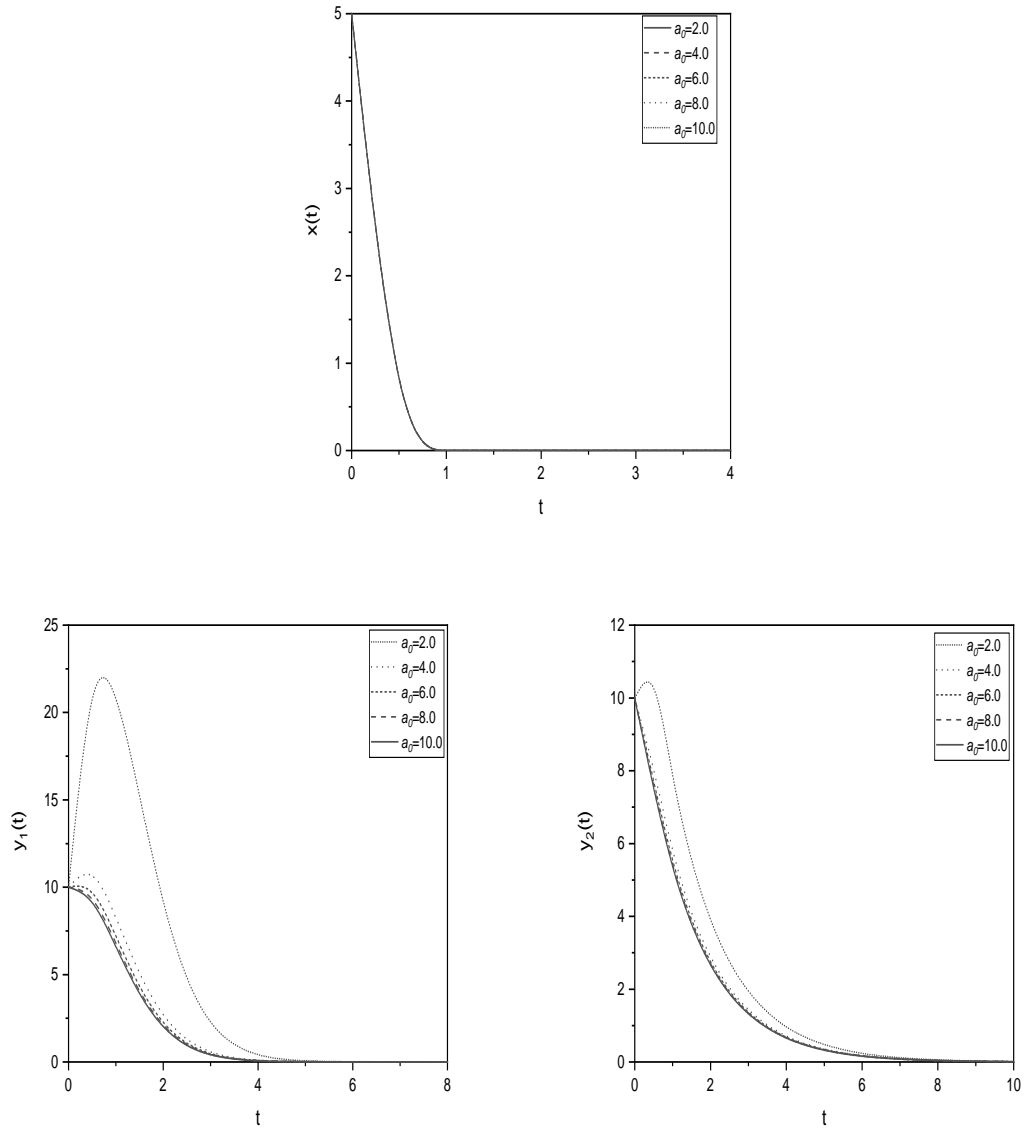


Figure 4.5: Figure depicting the effect of half saturation constant (a_o) on prey-predator system while impulse control is present

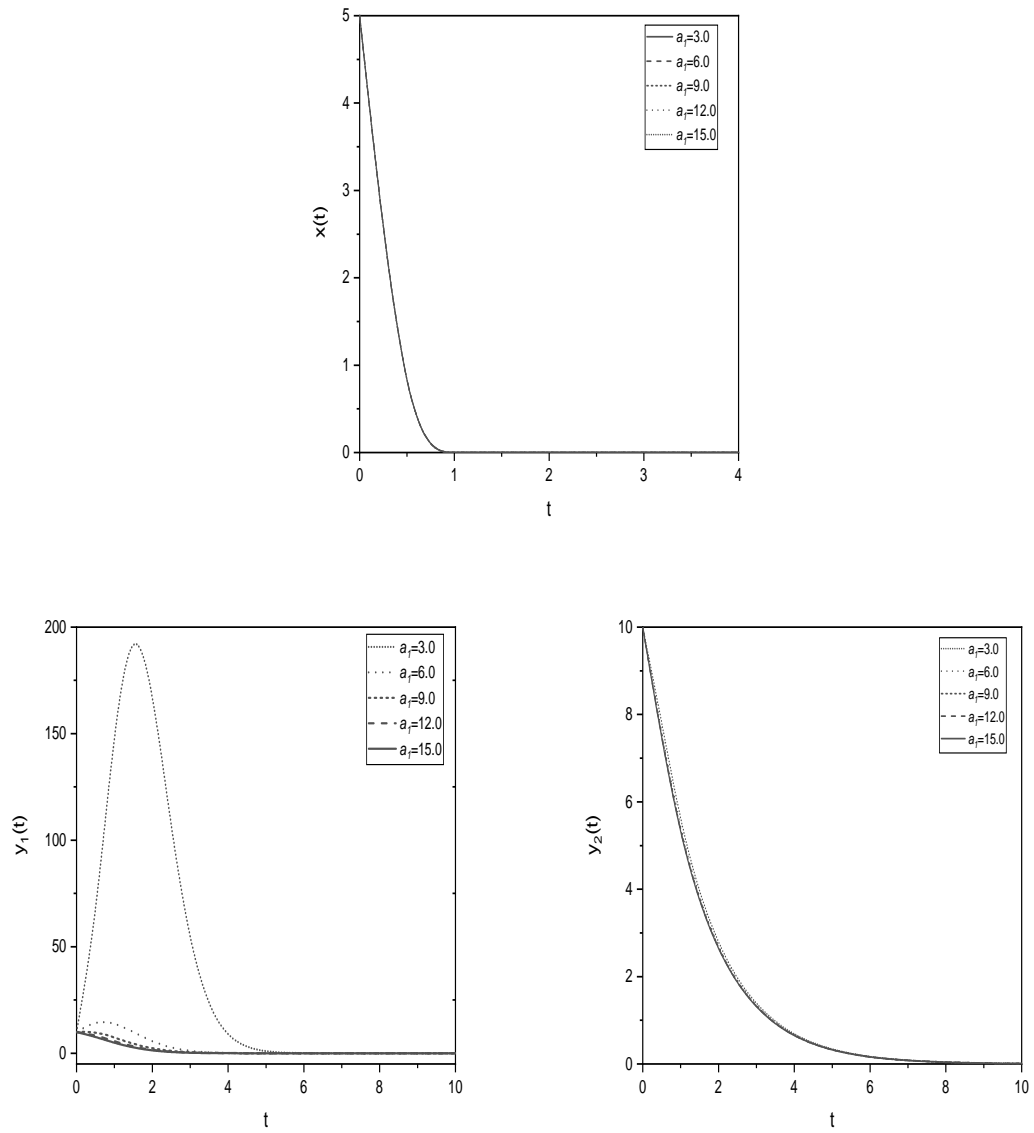


Figure 4.6: Figure depicting the effect of half saturation constant (a_1) on prey-predator system while impulse control is present

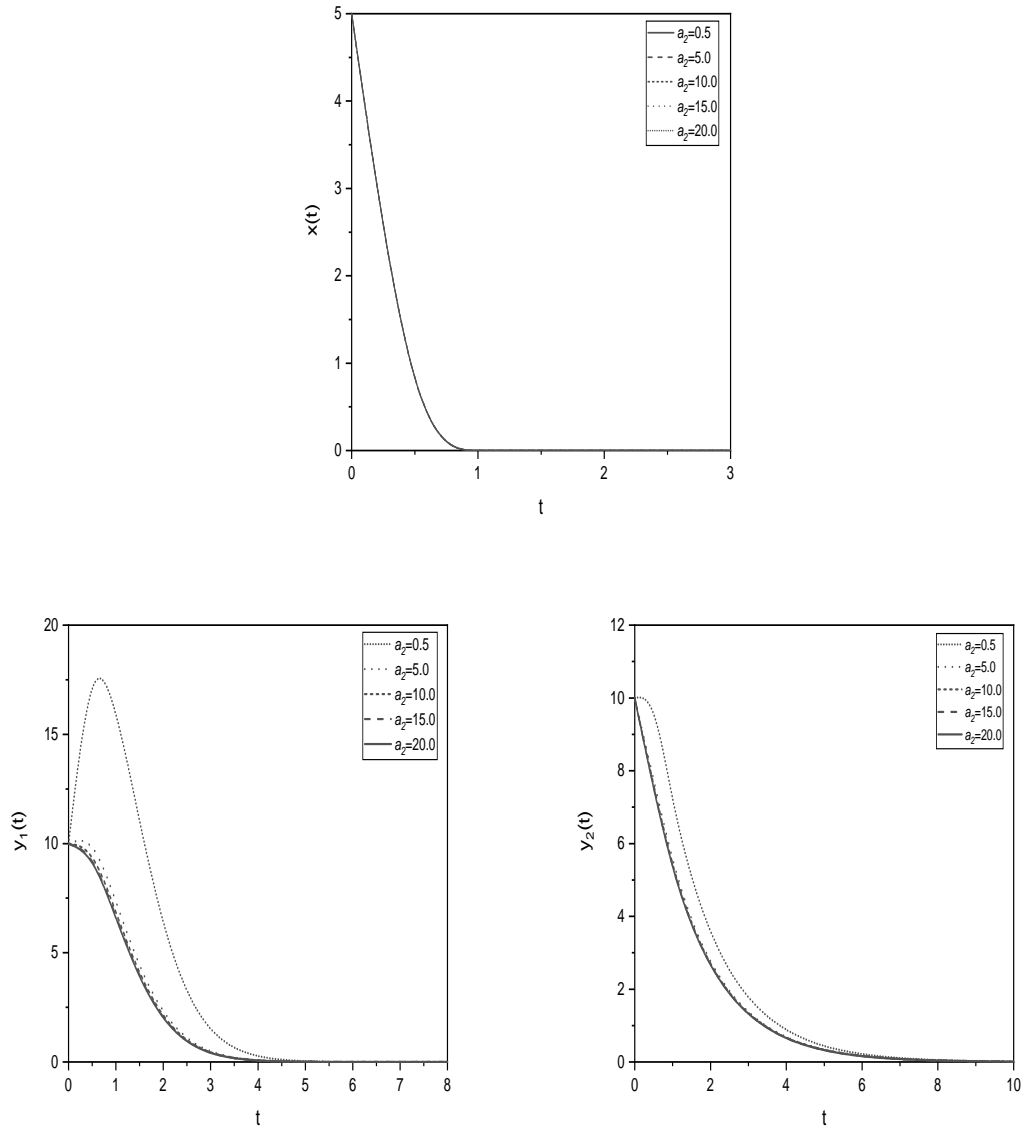


Figure 4.7: Figure depicting the effect of half saturation constant (a_2) on prey-predator system while impulse control is present

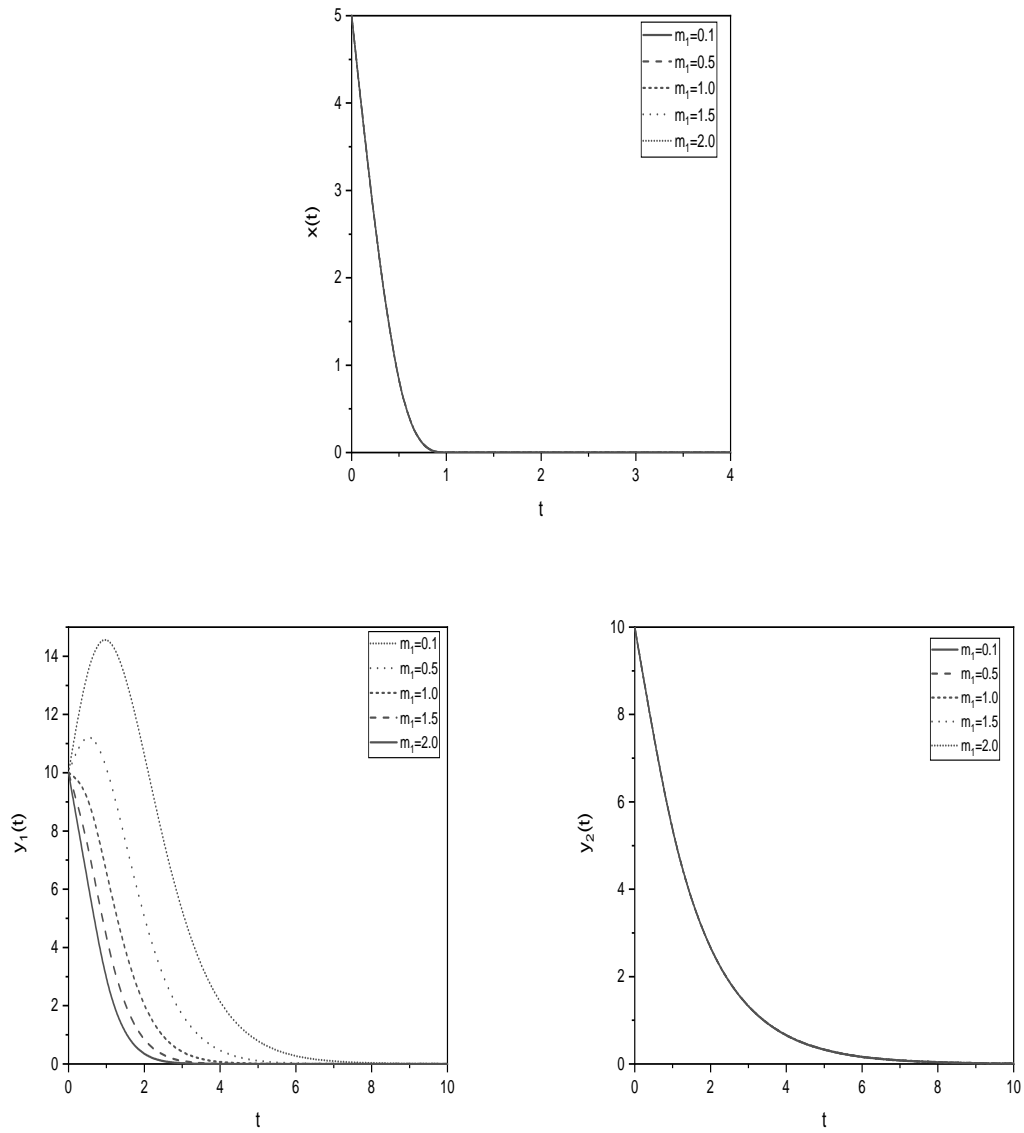


Figure 4.8: Figure depicting the impact of mortality rate of first predator (m_1) on prey-predator population while impulse control is present

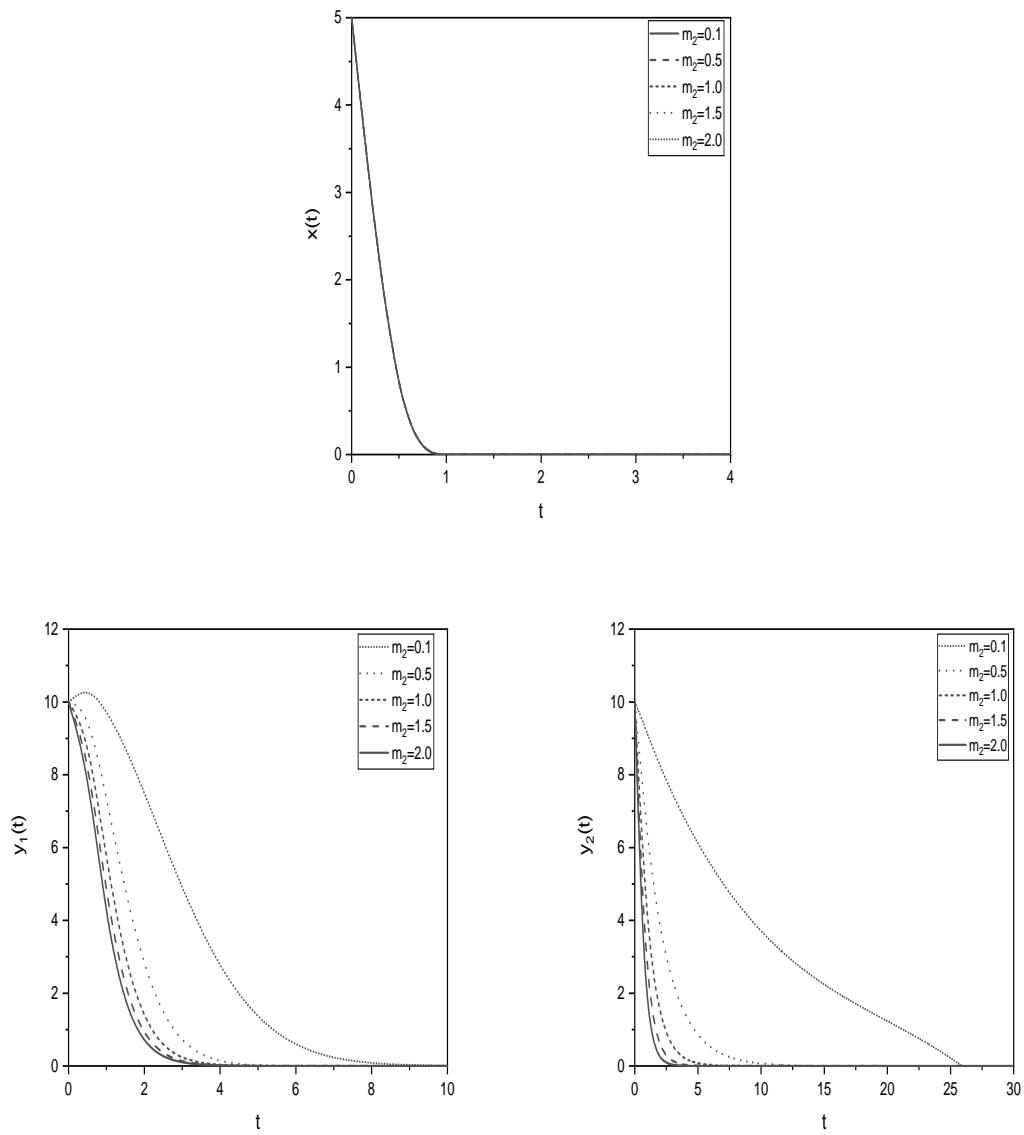


Figure 4.9: Figure depicting the impact of mortality rate of second predator (m_2) on prey-predator population while impulse control is present

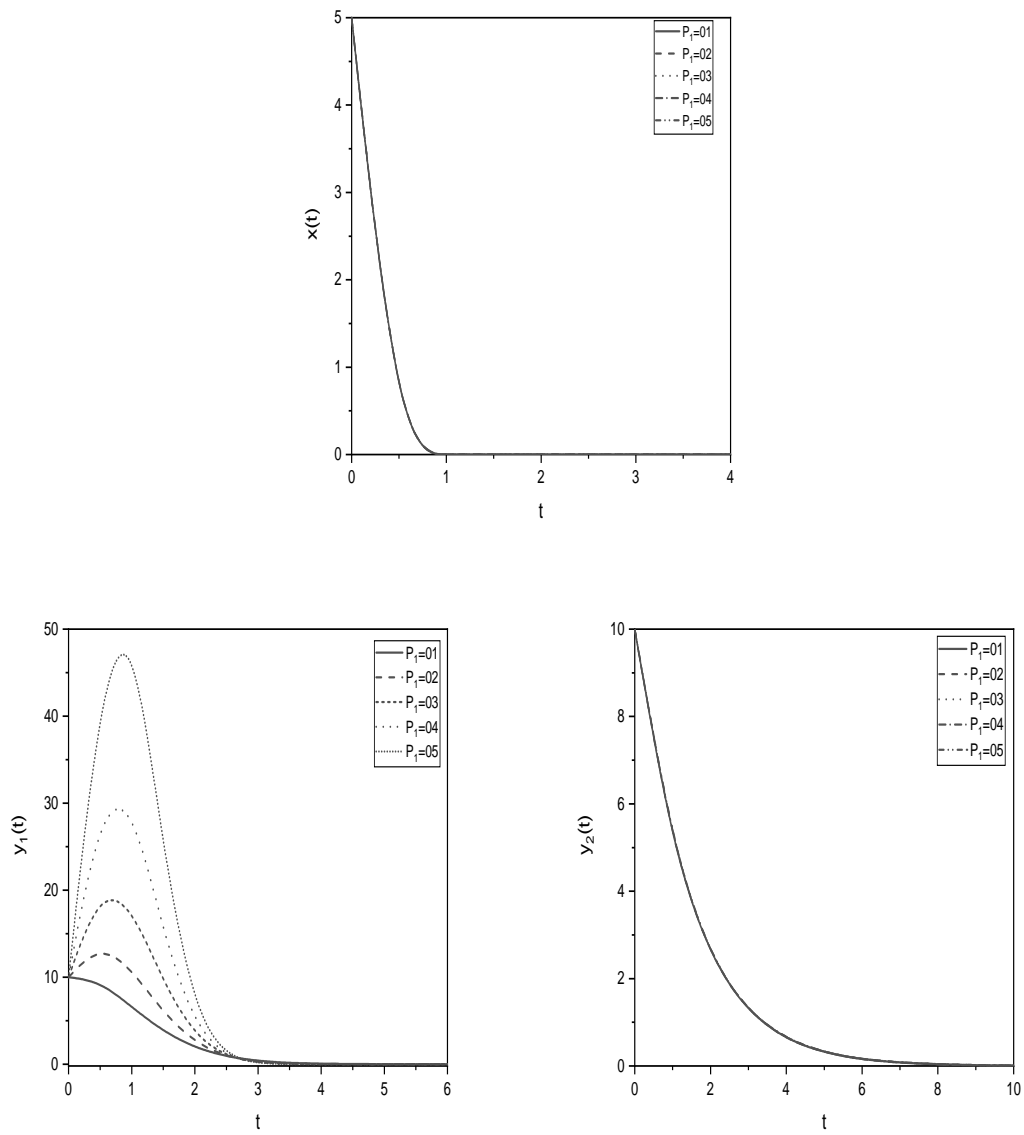


Figure 4.10: Figure depicting the effect of predation rate of first predator (P_1) on prey-predator system while impulse control is present

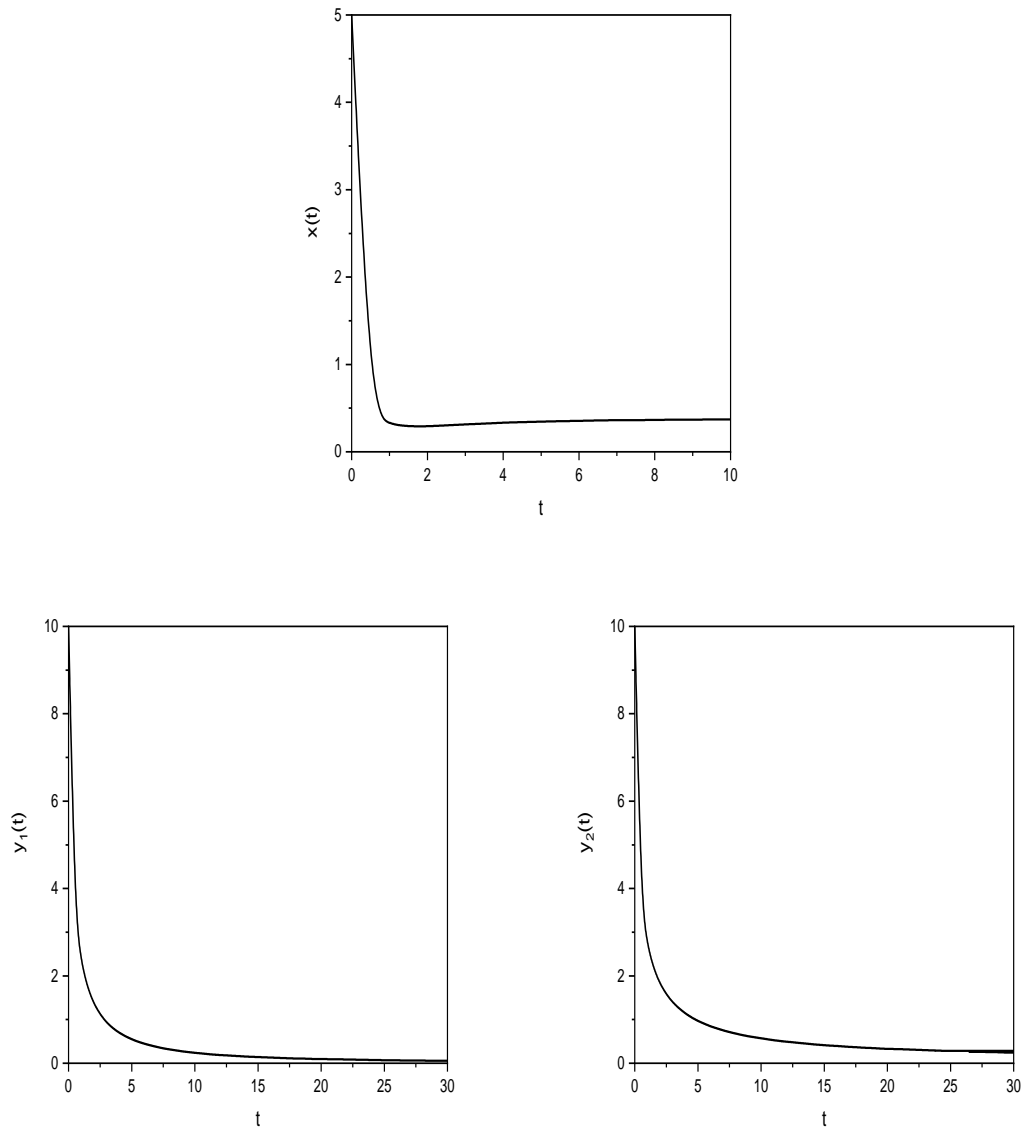


Figure 4.11: plot of a prey-predator system that does not possess impulse control

Part III

Population Dynamics of prey-predator system with infection

Chapter 5

Stability Analysis of a Predator-Prey model with infection in Predator Population ¹

5.1 Introduction

In this chapter, we considered the predator-prey model with eco-epidemiological implications among three species: prey, susceptible predator, and infected predator, in which disease solely affects the predator population [39, 59].

5.2 Model Formation

Our mathematical model is based on the following assumptions:

- When there is no predator, the prey population expands operational with a per capita constant growth rate r and a carrying capacity of the environment $c = r/e$, where e represents the prey's intra-specific competition. Thus

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$$\frac{dx}{dt} = rx(1 - \frac{x}{c}) \quad (5.1)$$

where $x(t)$ denotes the prey population at time.

- In presence of injurious infection, the whole predator population $y(t)$ is divided into two classes, among them one is the susceptible predator $y_s(t)$ and the other is the infected predator $y_i(t)$. As a result, the overall predator population density at time t is -

$$y(t) = y_s(t) + y_i(t) \quad (5.2)$$

- We believe that the disease is solely affecting the predator species, and that the prey population is unaltered. The population of diseased predators does not recover or develop immune.
- For the susceptible predator and the infected predator, the predation rate or searching efficiency constants are P_1 and P_2 , respectively, because the susceptible predator is more effective than the infected predator; therefore, we assume that the prey is eaten by the susceptible predator based on the basic mass action occurrence. According to the Holling type - II functional response, the diseased predator eats the prey. We assume that the susceptible predator has no handling time and the infected predator has a non-zero handling time, which obviously depicts a better ecological situation than assuming both predators have the same predation rate.
- We presume that the disease spreading is governed by the basic rule of mass action. It states that the rate of infection is proportional to both the number of susceptible individuals (S) and the number of infected individuals (I) in the population. i.e. Rate of infection $(\beta) \propto S \cdot I$
- Let D is the predator's natural death rate, B is the predator's birth rate, and

m is the predator's disease-related mortality rate.

We propose the following model utilizing a set of nonlinear differential equations based on the above assumptions:

$$\begin{aligned}\frac{dx}{dt} &= rx - ex^2 - P_1xy_s - \frac{P_2xy_i}{a+x} \\ \frac{dy_s}{dt} &= f_1P_1xy_s - \beta y_iy_s + (B - D)y_s \\ \frac{dy_i}{dt} &= \beta y_iy_s + \frac{f_2P_2xy_i}{a+x} - my_i\end{aligned}\tag{5.3}$$

where x_0, y_{s0}, y_{i0} are the initial populations and all the parameters r, e, P_1, P_2, m, β , are positive and $(B - D)$ can be either sign with $x = x_0 > 0, y_s = y_{s0} > 0, y_i = y_{i0} > 0, a$ is the half saturation constant, f_1, f_2 are the food conversion rates such that $0 < f_1, f_2 < 1, \beta$ is the rate of transmission of a force of infection.

A matrix differential equation is stated as follows to analyze the system's stability:

$$\dot{x} = Ax + \phi(x)\tag{5.4}$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}(t) \\ \dot{y}_s(t) \\ \dot{y}_i(t) \end{pmatrix}, A = \begin{bmatrix} r & 0 & 0 \\ 0 & (B - D) & 0 \\ 0 & 0 & -m \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex^2 - P_1xy_s - \frac{P_2xy_i}{a+x} \\ f_1P_1xy_s - \beta y_iy_s \\ \beta y_iy_s + \frac{f_2P_2xy_i}{a+x} \end{bmatrix}$$

5.3 T-S Fuzzy model with Impulsive effects

The non-linear equations can be transformed into the following linear equation as explained in earlier (chapter-2).

If $x(t)$ is M_i then,

$$\begin{aligned}
\dot{x}(t) &= A_i x(t), t \neq \tau_j \\
\Delta(x) &= K_{ij} x(t), t = \tau_j, \\
i &= 1, 2, 3 \dots r; j = 1, 2, \dots
\end{aligned} \tag{5.5}$$

where

$$A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 & 0 & 0 \\ f_1 z_2 & -z_4 + (B - D) & 0 \\ f_2 z_3 & z_4 & -m \end{bmatrix}, i = 1 \text{ to } 16, \text{ where}$$

the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k=1$ to 4 and z_1, z_2, z_3, z_4 are related to the values of $x(t) \in [0, d_1]$, $y_s(t) \in [0, d_2]$, $y_i(t) \in [0, d_3]$ (here $z_1 = ex$, $z_2 = P_1 y_s$, $z_3 = \frac{P_2 y_i}{a + x}$, $z_4 = \beta y_i$). $M_i, x(t), A_i \in R^{3 \times 3}$, r is the number of the IF-THEN rules, K_{ij} denotes the control of the j^{th} impulsive instant, $\Delta(x)|_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

5.4 Numerical Simulation

By using fuzzy impulsive T-S design model on (5.4), the membership functions obtained as

$$\begin{aligned}
M_1 &= \frac{z_1}{ed_1}, M_2 = \frac{ed_1 - z_1}{ed_1}, N_1 = \frac{z_2}{P_1 d_2}, N_2 = \frac{P_1 d_2 - z_2}{P_1 d_2}, K_1 = \frac{z_3}{\frac{P_2 d_3}{a + d_1}}, \\
K_2 &= \frac{\frac{P_2 d_3}{a + d_1} - z_3}{\frac{P_2 d_3}{a + d_1}}, P_1 = \frac{z_4}{\beta d_3}, P_2 = \frac{\beta d_3 - z_4}{\beta d_3} \text{ and the matrices } A'_i \text{s are calculated using} \\
A_i &= \begin{bmatrix} r - z_1 - z_2 - z_3 & 0 & 0 \\ f_1 z_2 & -z_4 + (B - D) & 0 \\ f_2 z_3 & z_4 & -m \end{bmatrix} \quad i = 1 \text{ to } 16, \text{ where the matrices}
\end{aligned}$$

A'_i s are generated using maximum and minimum values of z'_k s; $k=1$ to 4 and, the Defuzzification can be represented as:

$$\dot{x}(t) = \sum_{i=1}^r h_i(z(t))(A_i x(t)) \quad (5.6)$$

here h'_i s are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i=1$ to 16, $j=1$ to 4,

This Fuzzy model is a suitable representation of the non-linear system (5.4) in the region $[0,10] \times [0,10] \times [0,10]$

5.5 Results and discussion

In this section, the global stability of the the considered intra-specific competition predator-prey model (5.4) is discussed. Because of biological systems are complicated, nonlinear, and unpredictable, fuzzy logical methods with linguistic descriptions should be used to represent them. We have studied the system (5.3) numerically using MATHEMATICA software to get better insight of the proposed model.

Calculations were carried by taking the values of the parameters at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $(B-D)=0.3125$, $\beta=0.25$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$ in (5.5) to get the eigen values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ as explained in the theorems ([83]). It is found that $\max(\lambda_i) = \lambda(\alpha) = 0.87$ then we have chosen $\text{diag}[-0.84, -0.84]$ as impulsive control matrix, such that $\omega = \|I + K\| = 0.16$. It is noted that the system (5.5) is stable globally when $\epsilon=1.5$, $\delta_j=0.02$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -1.41 < 0$). Further, it is noted that the predator-prey model is unstable when $r=2.5$, $P_1=0.25$, $P_2=0.25$, $a=0.25$, $f_1=1.75$, $f_2=2.27$, $(B-D)=6$, $\beta=0.5$, $m=0.3$, $e=15$, $d_1=25$, $d_2=25$, $d_3=25$, since $\max(\lambda_i)=\lambda(\alpha) = 73.74$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.0478 > 0$.

Table 5.1 presents the stability of the system at various values of the present study.

The impact of the various parameters on prey-predator system (5.3) with T-S

r	P_1	P_2	a	f_1	f_2	β	$(B - D)$	m	e	d_1	d_2	d_3	$\max(\lambda_i) = \lambda(\alpha)$	$\ln(\epsilon\omega) + \lambda(\alpha)\delta_j$	conclusion
0.5	0.3125	0.25	0.5	0.3125	0.1875	0.25	0.3125	0.125	0.0005	10	10	10	0.87	-1.41	stable
0.1	0.5	0.5	0.5	0.2	0.2	0.5	0.45	0.2	25	30	30	30	0.95	-1.408	stable
3.8	0.5	2	0.3	1.25	1.5	0.15	2	0.3	3	20	20	20	50.34	-0.4202	stable
2.5	0.25	0.25	0.25	1.75	2.27	0.5	6	0.3	15	25	25	25	73.74	0.0478	unstable

Table 5.1: Stability of the system at various parameters

fuzzy impulsive control model is presented in figs. 5.1 - 5.10.

The effect of infection parameter (β) on prey-predator system is shown in fig. 5.1 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$. This graph makes it abundantly evident that as infection rates rise, the population of susceptible predator's decrease.

The influence of disease mortality (m) on prey-predator system is shown in fig. 5.2 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$. This figure clearly exhibits that an increase in disease mortality leads to decrease in susceptible predator population (which decreases slowly) whereas the infected predator population decreases faster.

The consequences of intra-specific competition (e) on prey-predator system is shown in fig. 5.3 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $d_1=10$, $d_2=10$, $d_3=10$. This graph demonstrates how less intra-specific competition between prey and predator results in a rise in the population of prey.

The change on prey-predator system with growth rate of prey (r) is shown in fig. 5.4 at $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$. This figure clearly exhibits that increase in growth rate of prey leads to increase in the prey population and the population of the predator becomes stable.

The outcome with varying predation rate of susceptible predator (P_1) on prey-predator system is shown in fig. 5.5 at $r=0.5$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$,

$\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$. This graph demonstrates unambiguously how an increase in a predator's predation rate causes a drop in the prey's population.

The impact of predation rate of infected predator (P_2) on prey-predator system is shown in fig. 5.6 at $r=0.5$, $P_1=0.3125$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$. This graph illustrates clearly how an infected predator's increased predation rate causes a drop in the number of preys.

The dynamical change on prey- predator population (x, y_s, y_i) by varying prey max time (d_1) parameter under fuzzy impulsive control can be noted in fig. 5.7 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e = 0.0005$, $d_2=10$, $d_3=10$. It is noticed from this figure that, the prey population decreases as d_1 decreases.

The effectiveness by varying susceptible predator max time (d_2) parameter of prey- predator population (x, y_s, y_i) under fuzzy impulsive control can be noted in fig. 5.8 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_3=10$. It is noted from this figure that, the prey population increases as d_2 decreases.

The vital pattern of prey- predator population (x, y_s, y_i) by varying infected predator max time (d_3) parameter under fuzzy impulsive control can be noted in fig. 5.9 at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2=0.1875$, $\beta = 0.25$, $(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$. It is observed from this figure that, the prey population increases and predator population decreases with an increase in d_3 .

Finally, the nature of two species (x, y_s, y_i) population (without impulsive control) is presented in fig. 5.10 by fixing all the parameters obtained from T-S fuzzy model at $r=0.5$, $P_1=0.3125$, $P_2=0.25$, $a=0.5$, $f_1=0.3125$, $f_2 =0.1875$, $\beta = 0.25$,

$(B - D)=0.3125$, $m=0.125$, $e=0.0005$, $d_1=10$, $d_2=10$, $d_3=10$ and initial conditions $x(0) = 10$, $y_s(0) = 5$, $y_i(0) = 5$, $t = 5$. The figure clearly shows how the prey- predator populations reaches to stability whereas infected predator becomes unstable.

5.6 Conclusions

In this chapter we present stability analysis of a three species competition model with fuzzy impulsive control by T-S model, in which disease infection is in predator. The main results of this study are as follows:

- We establish a predator-prey model in which predator population is infected.
- Less intra-specific competition between prey and predator results in a rise in the population of prey because of the infection in predators.
- A rise in the prey population results from an increase in the prey growth rate.
- The population of healthy predators decreases as the rate of disease transmission from diseased to susceptible predators rises because more predators will contract the disease.
- As the maximum period for prey diminishes, the prey population decreases.
- Because predators will have less time for predation as the maximum period for susceptible predators gets shorter, the number of prey rises.
- While the population of prey increases as the maximum time for an infected predator increases, the population of susceptible predators decreases because more predators will contract the infection.

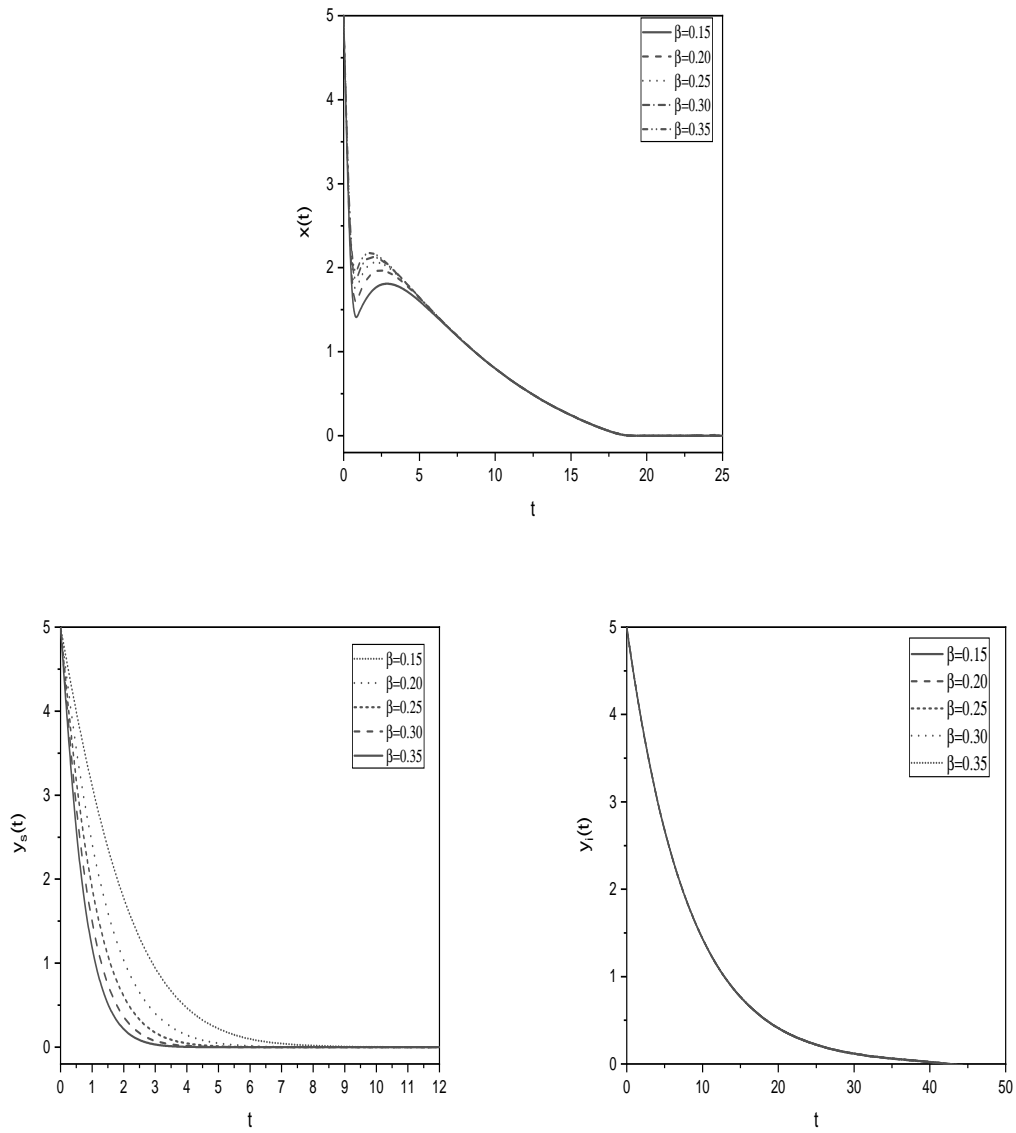


Figure 5.1: Effect of infection transmission rate (β) on prey-predator system under impulsive control

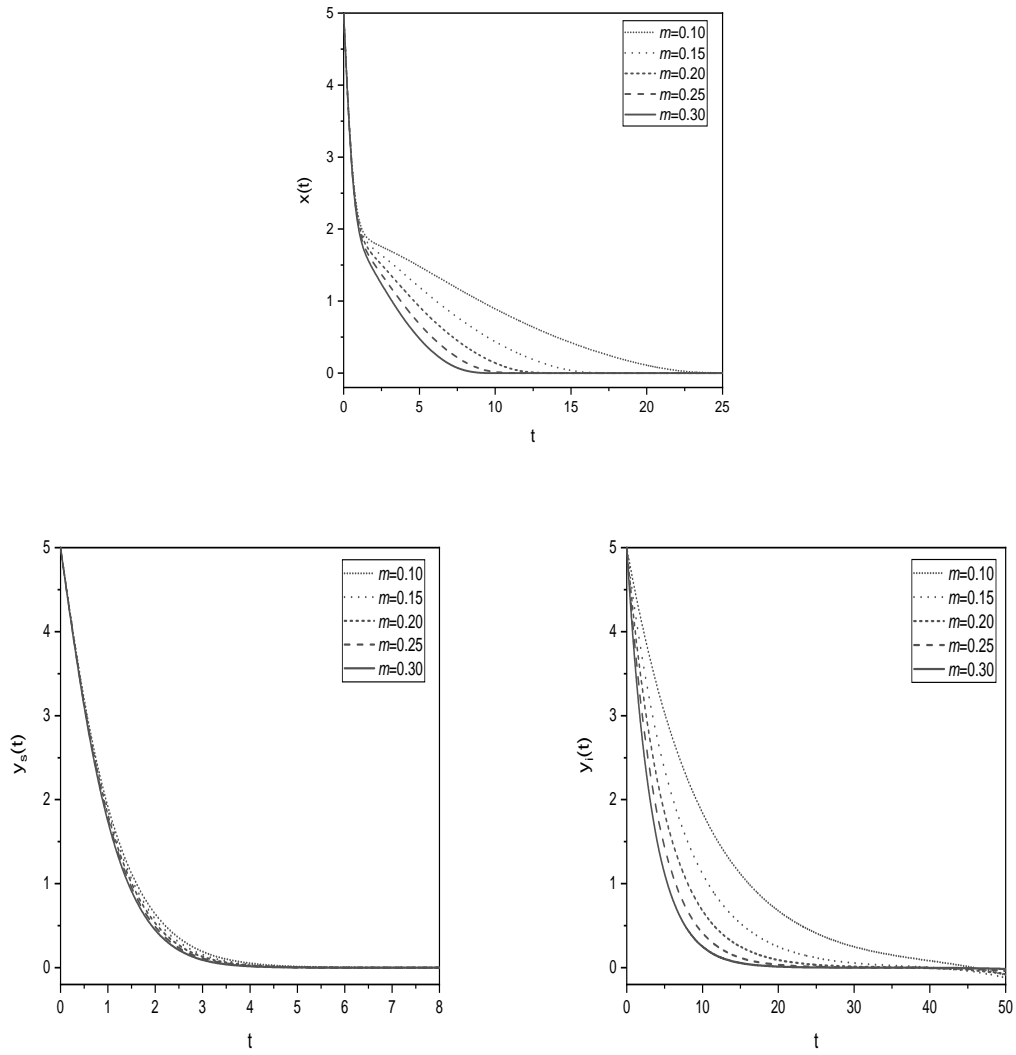


Figure 5.2: Effect of disease mortality (m) on prey-predator system under impulsive control.

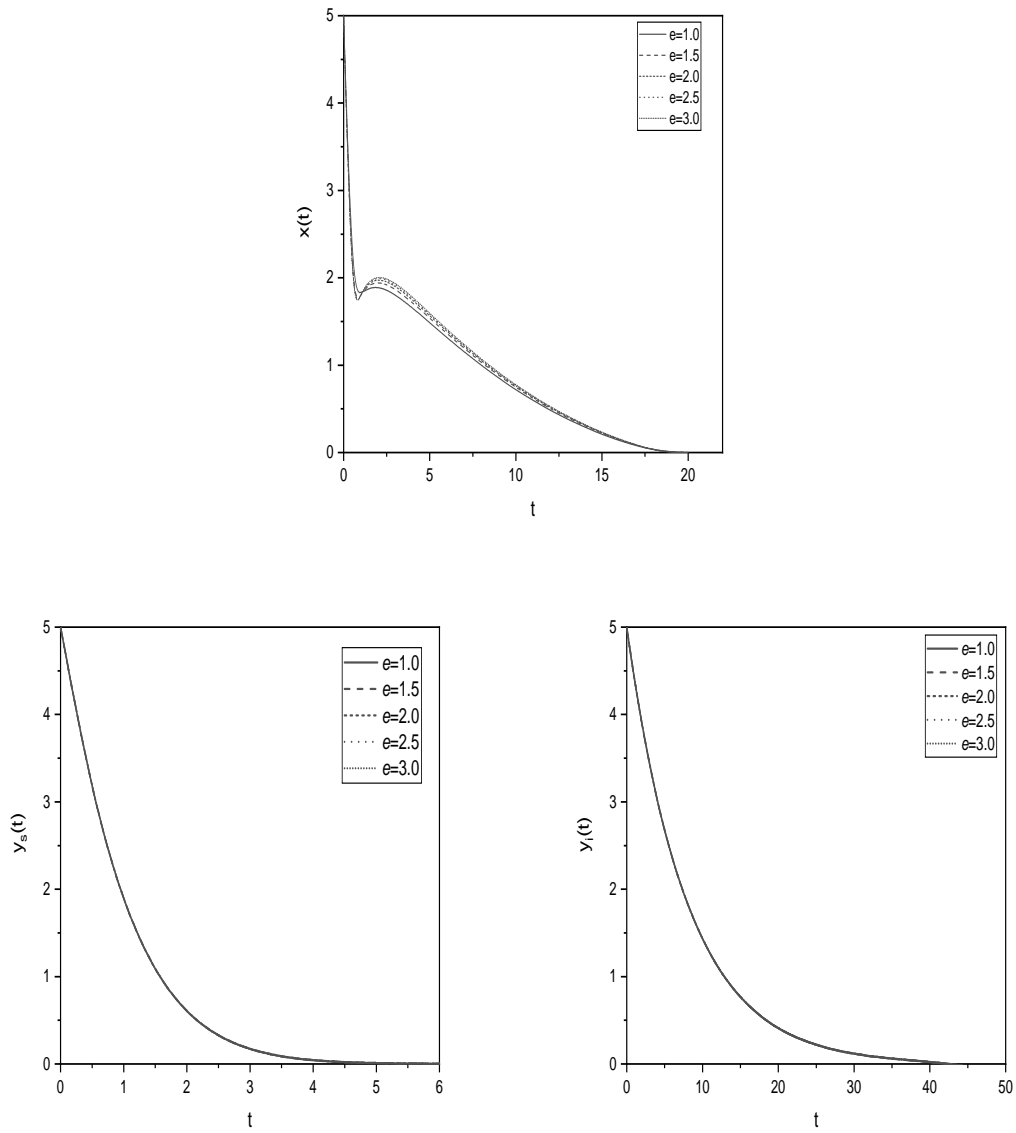


Figure 5.3: Effect of inter-specific competition (e) on prey-predator system under impulsive control.

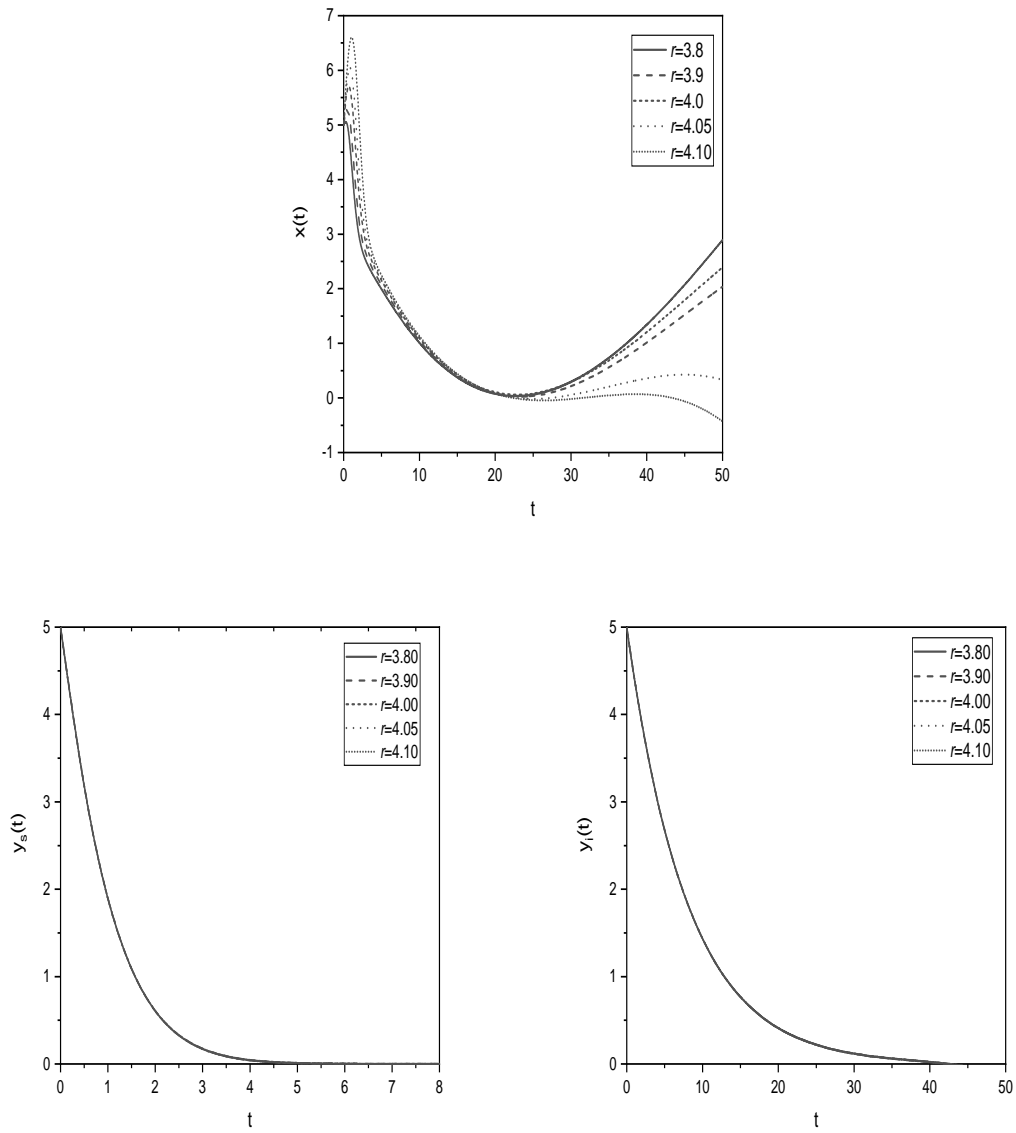


Figure 5.4: Effect of growth rate of prey (r) on prey-predator system under impulsive control.

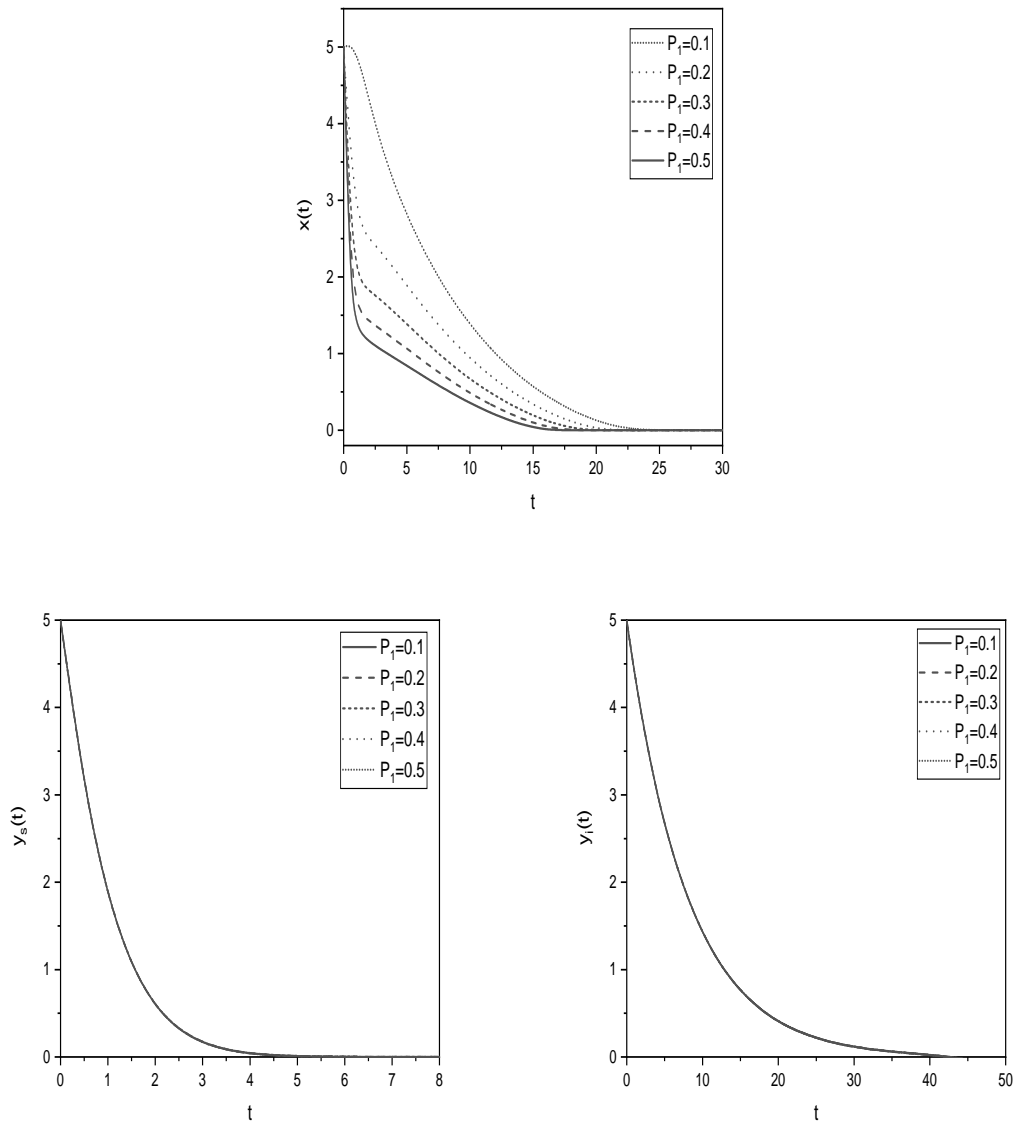


Figure 5.5: Effect of predation rate of susceptible predator (P_1) on prey-predator system under impulsive control.

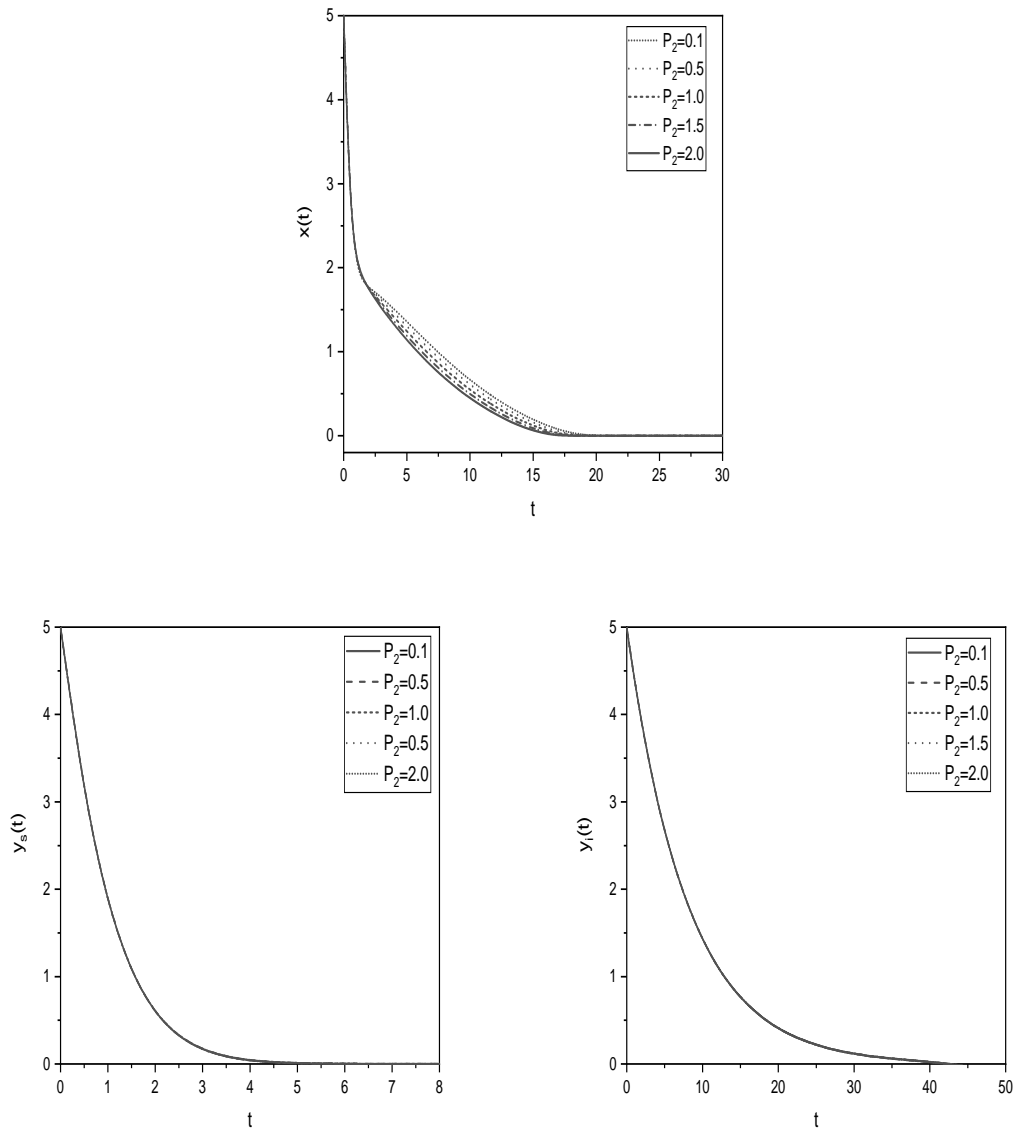
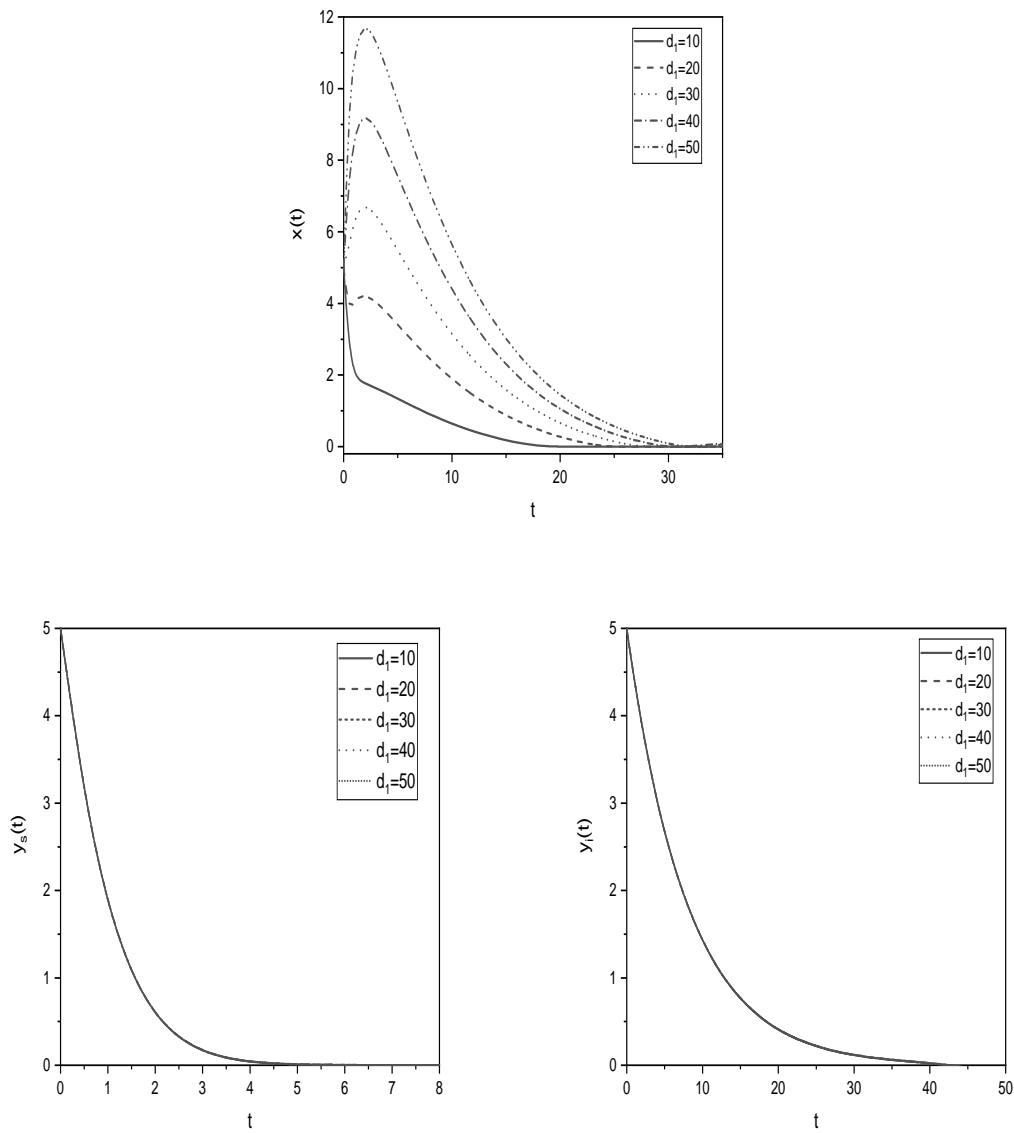
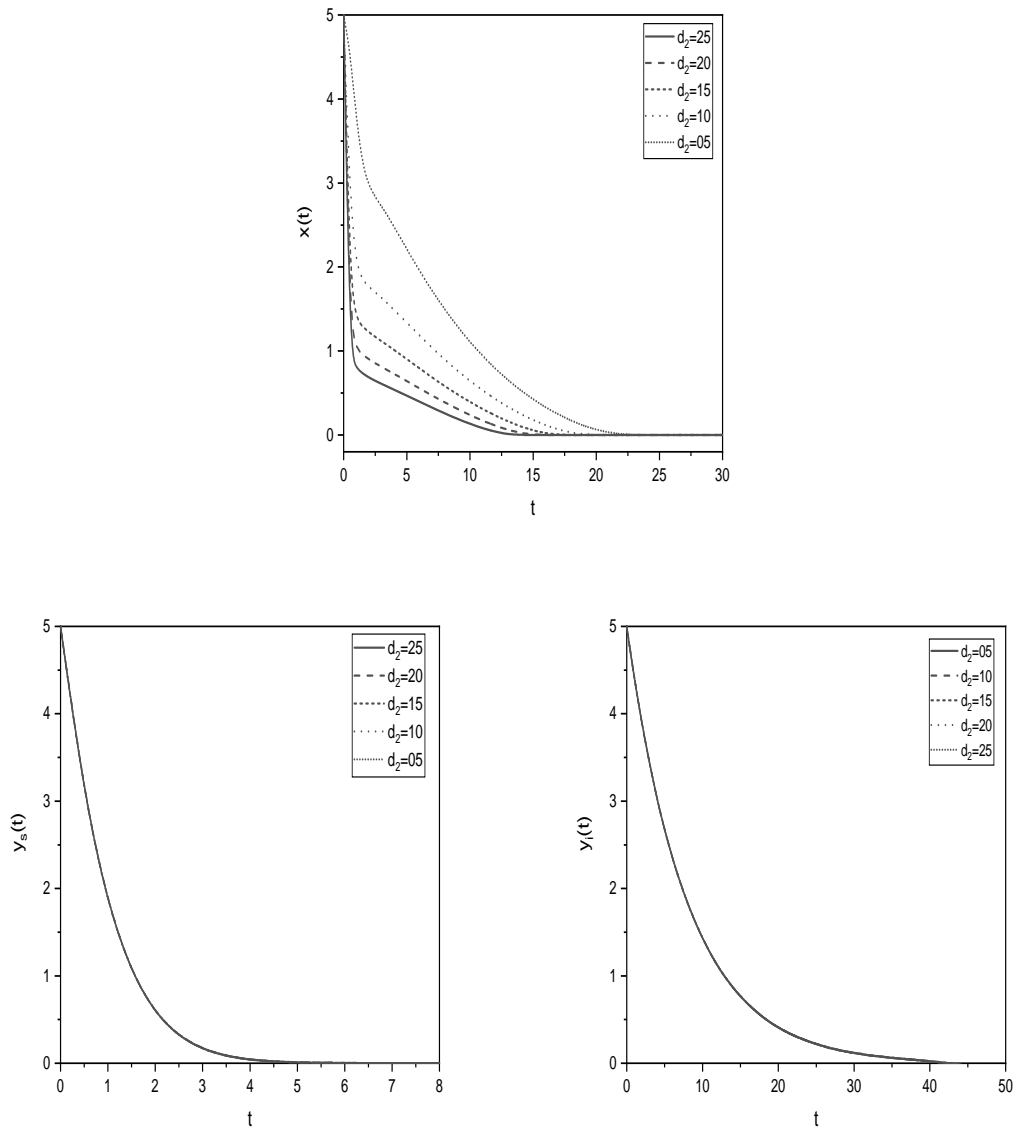


Figure 5.6: Effect of predation rate of infected predator (P_2) on prey-predator system under impulsive control.

Figure 5.7: Effect of max time (d_1) on prey-predator system under impulsive control.

Figure 5.8: Effect of max time (d_2) on prey-predator system under impulsive control.

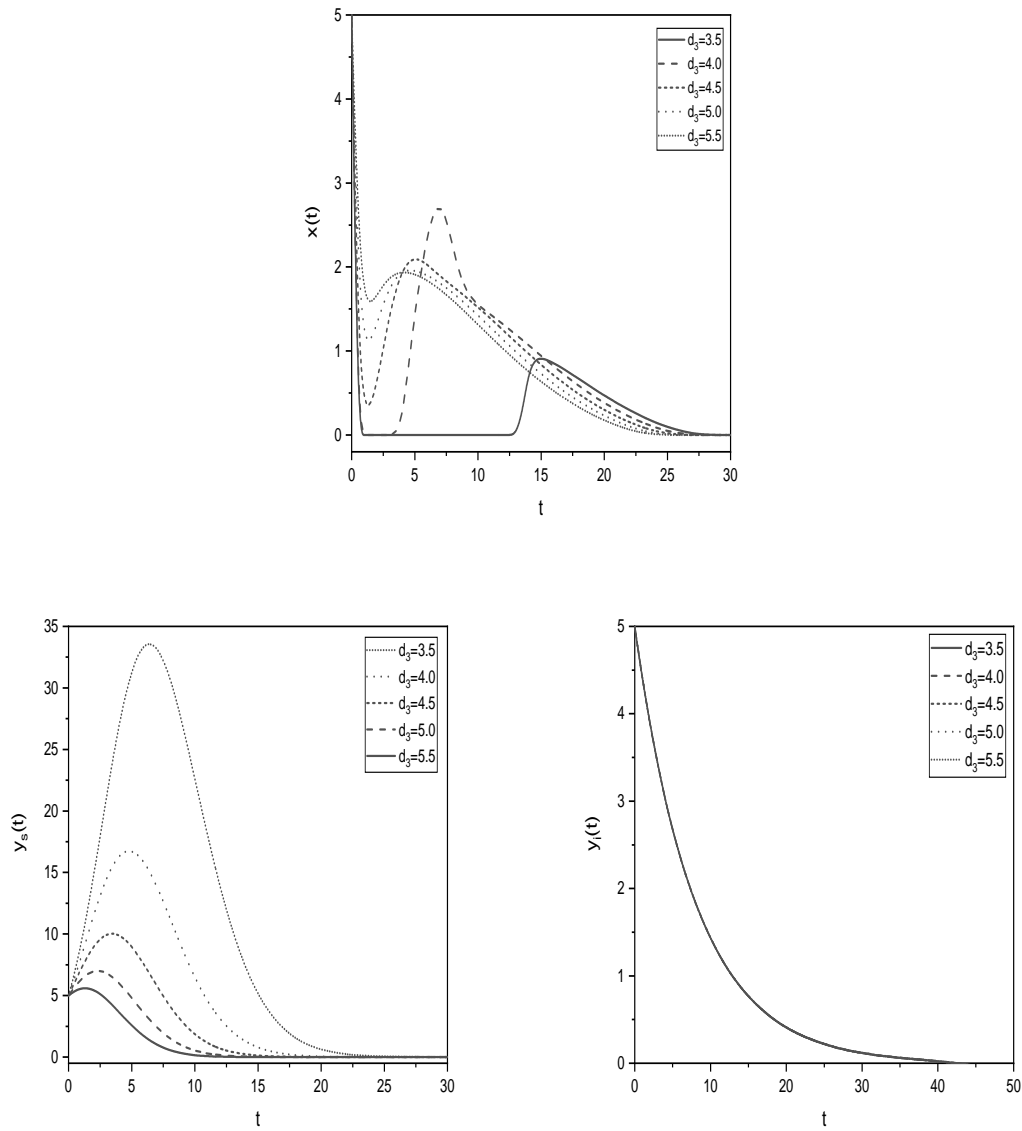


Figure 5.9: Effect of max time (d_3) on prey-predator system under impulsive control.

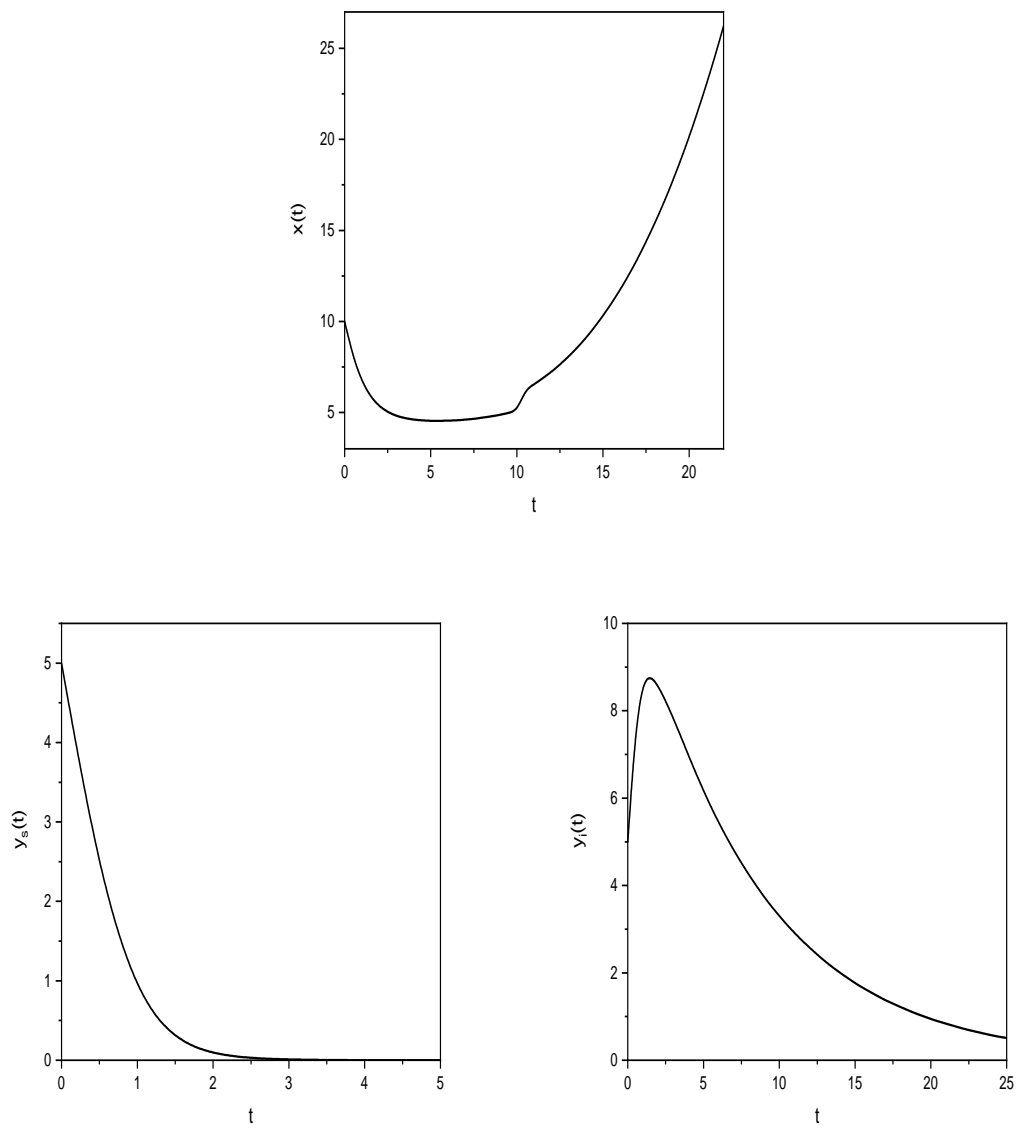


Figure 5.10: Plot of predator-prey system without impulsive control.

Chapter 6

A Takagi-Sugeno based study of population dynamics with infected prey ¹

6.1 Introduction

In this study, we describe the prey-predator model proposed by Venturino [102] to investigate the species' existence. Consider the simple example where the predator mostly consumes the ill prey for this purpose. We used an eco-epidemiological system with three species, susceptible prey, diseased prey and predator. We explore the scenario in which the predator primarily feeds ill prey [103]. Because of the illness, the prey becomes weaker and more vulnerable to predators. The T-S model and stability theorems [83, 77] are used to describe the local stability analysis.

6.2 Model Construction

The following assumptions guide our mathematical model:

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- x represents the total population density of the prey.
- y represents the total population density of predators.
- In the absence of illness, the prey population develops logistically with a carrying capacity of $c = \frac{r}{e}$ ($c > 0$) and an intrinsic growth rate of $r(> 0)$. Thus we have -

$$\frac{dx}{dt} = rx\left(1 - \frac{x}{c}\right)$$

- When an infection occurs, we assume that all preys have two classes of x ; one is the susceptible (x_s), and the other is the infected prey (x_i). Therefore, the total population density of prey at any given time (t) is $x(t) = x_s(t) + x_i(t)$.
- We believe that the disease only affects prey. Many prey species do not recover or develop immunity. Suppose the condition is simply as large as $\beta x_s x_i$, where $\beta > 0$ is called the coefficient of transmission.
- The costs of predation or continued pursuit of the injured and infected are P_1 and P_2 , respectively. Predators catch prey and get prey disease because victims are more vulnerable than prey, making it easier for them to hunt, i.e. $P_2 > P_1$.
- We hypothesized that prey animals preyed on vulnerable animals based on the simple nature of group work and injured animals based on Holling's type - II response task.

Based on the above considerations, we propose the following model using the system of non-linear differential equations.

$$\begin{aligned}
\frac{dx_s}{dt} &= rx_s - ex_s^2 - \beta x_s x_i - \frac{P_1 x_s y}{a + x_s} \\
\frac{dx_i}{dt} &= \beta x_s x_i - \frac{P_2 x_i y}{a + x_i} - m_1 x_i \\
\frac{dy}{dt} &= \frac{C_1 x_s y}{a + x_s} + \frac{C_2 x_i y}{a + x_i} - m_2 y
\end{aligned} \tag{6.1}$$

where x_{s0}, x_{i0}, y_0 are the initial populations and all parameters $(r, \beta, e, C_1, C_2, a, P_1, P_2, m_1, m_2) \geq 0$ with $x_s = x_{s0} > 0, x_i = x_{i0} > 0, y = y_0 > 0$.

Here r is the internal production growth rate of prey, β is the rate at which the disease spreads from an infected person to a susceptible person, e is the intra-species competition, P_1 is the predation rate of vulnerable prey, P_2 is the Predation level of infected prey, m_1 is the mortality rate of infected victims, m_2 is the predator mortality, C_1 is the predator conversion rate after eating vulnerable prey, C_2 is the predator conversion rate after eating infected prey, and a is the half-saturation constant.

To analyze the system's stability, the matrix differential equation is written as

$$\dot{x} = Ax + \phi(x) \tag{6.2}$$

here

$$\dot{x} = \begin{pmatrix} \dot{x}_s(t) \\ \dot{x}_i(t) \\ \dot{y}(t) \end{pmatrix}, A = \begin{bmatrix} r & 0 & 0 \\ 0 & -m_1 & 0 \\ 0 & 0 & -m_2 \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex_s^2 - \beta x_s x_i - \frac{P_1 x_s y}{a + x_s} \\ \beta x_s x_i - \frac{P_2 x_i y}{a + x_i} \\ \frac{C_1 x_s y}{a + x_s} + \frac{C_2 x_i y}{a + x_i} \end{bmatrix}$$

6.3 Fuzzy Takagi-Sugeno approach with impacts of impulse

The non-linear equations can be transformed into the following linear equation as explained in the earlier (chapter-2).

If $x(t)$ is M_i then

$$\dot{x}(t) = A_i x(t), t \neq \tau_j \quad (6.3)$$

$$\Delta(x) = k_{ij}x(t), t = \tau_j, i = 1, 2, 3 \dots r; j = 1, 2, \dots \quad (6.4)$$

$$\text{where, } A_i = \begin{bmatrix} r - z_1 - z_2 - P_1 z_3 & 0 & 0 \\ z_2 & -m_1 - P_2 z_4 & 0 \\ C_1 z_3 & C_2 z_4 & -m_2 \end{bmatrix}, i = 1 \text{ to } 16, \text{ where the}$$

matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 4 and z_1, z_2, z_3, z_4 are related to the values of $x_s(t) \in [0, d_1], x_i(t) \in [0, d_2], y(t) \in [0, d_3]$ (here $z_1 = ex_s, z_2 = rx_i, z_3 = \frac{y}{B + x_s}, z_4 = \frac{y}{B + x_i}$). $M_i, x(t), A_i \in R^{3 \times 3}$, and r is the number of the IF-THEN rules, $k_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta(x) |_{t=\tau_j} = x(\tau_j - \tau_{j-1})$.

6.4 Numerical Simulation

Because most biological systems are complicated, they should be expressed by applying a fuzzy logical framework that includes expressive reports. The suggested impulsive Takagi-Sugeno system looks at predator - prey system with functional reactions and impulsive impacts. By applying fuzzy impulsive Takagi-Sugeno model on (6.2), the membership functions [85] M'_{ij} s and the matrices A'_i s, are calculated using

$$A_i = \begin{bmatrix} r - z_1 - z_2 - P_1 z_3 & 0 & 0 \\ z_2 & -m_1 - P_2 z_4 & 0 \\ C_1 z_3 & C_2 z_4 & -m_2 \end{bmatrix}, i = 1 \text{ to } 16, \text{ where the matrices}$$

A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 4 . Therefore, Deffuzification is given as

$$\dot{x}(t) = \sum_{A_i=1}^r h_i(z(t))(A_i x(t)) \quad (6.5)$$

here h'_i s are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i = 1$ to 16 , $j = 1$ to 4 , where M'_{ij} s are membership functions.

This Fuzzy model is a perfect representation of the non-linear system in $[0,10] \times [0,10] \times [0,10]$

$$\begin{aligned} \frac{dx_s}{dt} &= rx_s - ex_s^2 - \beta x_s x_i - \frac{P_1 x_s y}{a + x_s} \\ \frac{dx_i}{dt} &= \beta x_s x_i - m_1 x_i - \frac{P_2 x_i y}{a + x_i} \\ \frac{dy}{dt} &= -m_2 y + \frac{C_1 x_s y}{a + x_s} + \frac{C_2 x_i y}{a + x_i} \end{aligned} \quad (6.6)$$

6.5 Results and discussion

This section describes the global stability of the considered intra-species predator-prey competition model (6.2). We have studied the system (6.1) numerically using MATHEMATICA software to get better insight of the proposed model.

It is calculated by taking parameter values at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$ in (6.3) to obtain the eigen values of $[A_i^T + A_i]$, ($i = 1, 2, 3 \dots r$) as discussed in ([83]). It is evident that $\max(\lambda_i) = \lambda(\alpha) = 51.4252$, then we have chosen $\text{diag}[-0.84, -0.84]$ as the matrix of impulsive control in such a way that $\omega = \|I + K\| = 0.16$. It is marked that the system (6.3) is globally-stable at $\epsilon = 1.5$, $\delta = 0.02$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -0.407 < 0$). Further, it is noted that the prey-predator

model is unstable when $r=1.5$, $\beta=0.5$, $C_1=60$, $C_2=65$, $m_1=0.5$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.015$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. Since $\max(\lambda_i)=\lambda(\alpha) = 72.18$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.0166 > 0$.

r	β	C_1	C_2	m_1	m_2	P_1	P_2	e	a	d_1	d_2	d_3	$\max(\lambda_i)$ $=\lambda(\alpha)$	$\ln(\epsilon\omega)$ $+\lambda(\alpha)\delta_j$	conclusion
0.8	0.12	50	40	0.91	2	0.03	0.5	0.0005	2	10	10	10	51.4252	-0.407	stable
1	0.1	10	20	1.5	1	0.01	0.2	0.05	2	10	10	10	16.8	-1.091	stable
1.2	0.8	30	30	1	1.5	0.1	0.1	0.15	2	10	10	10	34.3	-0.74	stable
1.5	0.5	60	65	0.5	2	0.03	0.5	0.015	2	10	10	10	72.18	0.166	unstable

Table 6.1: Stability of the system at various parameters

Table 6.1 shows the stability of the system at different rates in this study.

The effects of various parameters of the system 6.1 using the T-S fuzzy impulsive control model are shown in figs. 6.1 - 6.11.

The influence of (r) on prey - predator system is presented in fig. 6.1 at $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. This figure exhibited that an enhance in the growth rate of prey leads to enhance in the population of prey because more preys will grow.

The consequences of transmission rate of disease from infected to susceptible (β) on prey - predator system is presented in fig. 6.2 at $r=0.8$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. It is observed from this figure that healthy prey population decreases with an increase in (β) because more preys will get infected.

The change in prey-predator population (x_s, x_i, y) as a function of maximum time change of vulnerable prey (d_1) is shown in the fig. 6.3 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_2=10$, $d_3=10$. This figure clearly exhibits that, the population of prey enhances as d_1 drops.

The dynamic change of the prey-predator population (x_s, x_i, y) according to the change of the maximum time (d_2) of the infected prey in the prey-predator system is shown in fig. 6.4 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$,

$P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_3=10$. This figure shows that, the susceptible prey population decreases as d_2 increases whereas infected prey population increases because more preys will get infected.

The impact on prey - predator population (x_s, x_i, y) by ranging predator max time (d_3) on prey - predator system is demonstrated in fig. 6.5 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$. From this figure, it can be shown that, the prey population increases as d_3 decreases because predators get less time to eat prey.

Figure 6.6 depicts how intra-specific competition (e) affects the prey-predator system at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. The effect of intra-species competition is to decrease the rate of population growth as population density increases.

The changes with mortality rate of infected prey (m_1) on prey - predator system is presented in fig. 6.7 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. This graph unequivocally demonstrates that a rise in m_1 causes a fall in the population of prey.

The effect of (m_2) on prey - predator system is presented in fig. 6.8 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. This figure demonstrates that, susceptible prey population enhances with increase in (m_2) whereas infected prey and predator population decreases with an increase in (m_2).

The performance of the prey-predator population by varying (P_1) under fuzzy impulse control can be seen in figure. 6.9 at $g=0.8$, $r=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. This figure clearly displays that enhance in predation rate of susceptible prey indicate more preys are predated by predators which results to diminish in prey population.

The change with predation rate of infected prey (P_2) on prey- predator popu-

lation under fuzzy impulsive control can be found in fig. 6.10 at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$. From this figure, it can be shown that, enhance in predation rate of infected prey decreases prey population because more preys will be eaten by predator.

Finally, the characteristics of the population (x_s, x_i, y) of the three species (without impulsive control) are shown in fig. 6.11 by considering all the parameters obtained with the T-S fuzzy model at $r=0.8$, $\beta=0.12$, $C_1=50$, $C_2=40$, $m_1=0.91$, $m_2=2$, $P_1=0.03$, $P_2=0.5$, $e=0.0005$, $a=2$, $d_1=10$, $d_2=10$, $d_3=10$, and $x_s(0) = 3, x_i(0) = 5, y(0) = 5, t = 10$. The graph makes it very clear how predator and prey populations stabilize.

6.6 Conclusions

In this chapter we present stability analysis of a three species competition model with fuzzy impulsive control by T-S model. In which disease infection is in prey. The main results of this study are as follows:

- Prey populations more susceptible to predators are found where there is less intra-specific competition.
- An increase in prey growth rate results in an increase in the prey population.
- As the rate of disease transmission from diseased to vulnerable prey increases, the population of healthy prey drops.
- As the maximum time (d_2) of the infected prey rises, the number of susceptible prey declines while the number of infected prey increases.
- Prey population increases as predator max time (d_3) declines.
- Prey populations decline when sensitive predators prey more frequently.

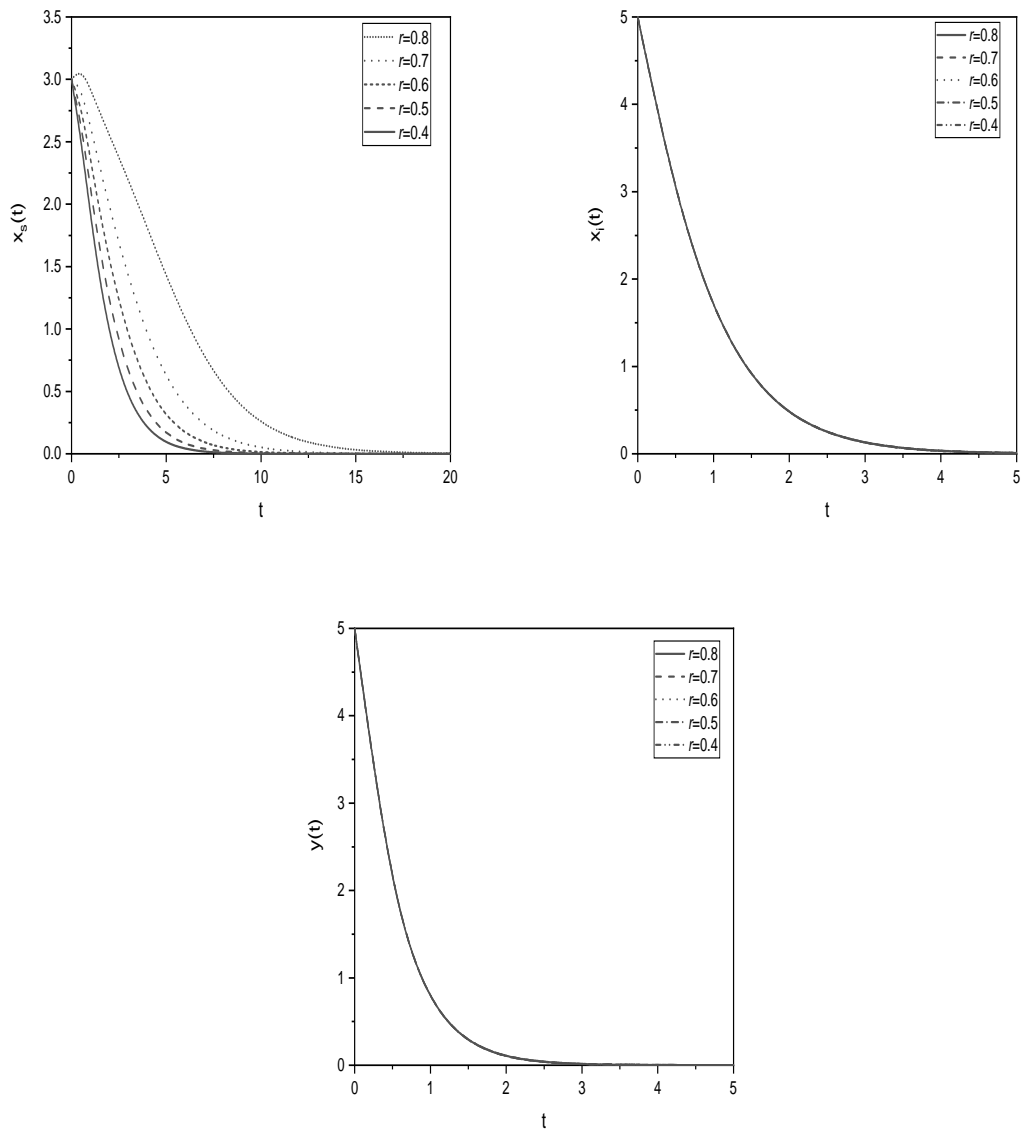


Figure 6.1: Influence of r on Prey - Predator System under impulsive Control.

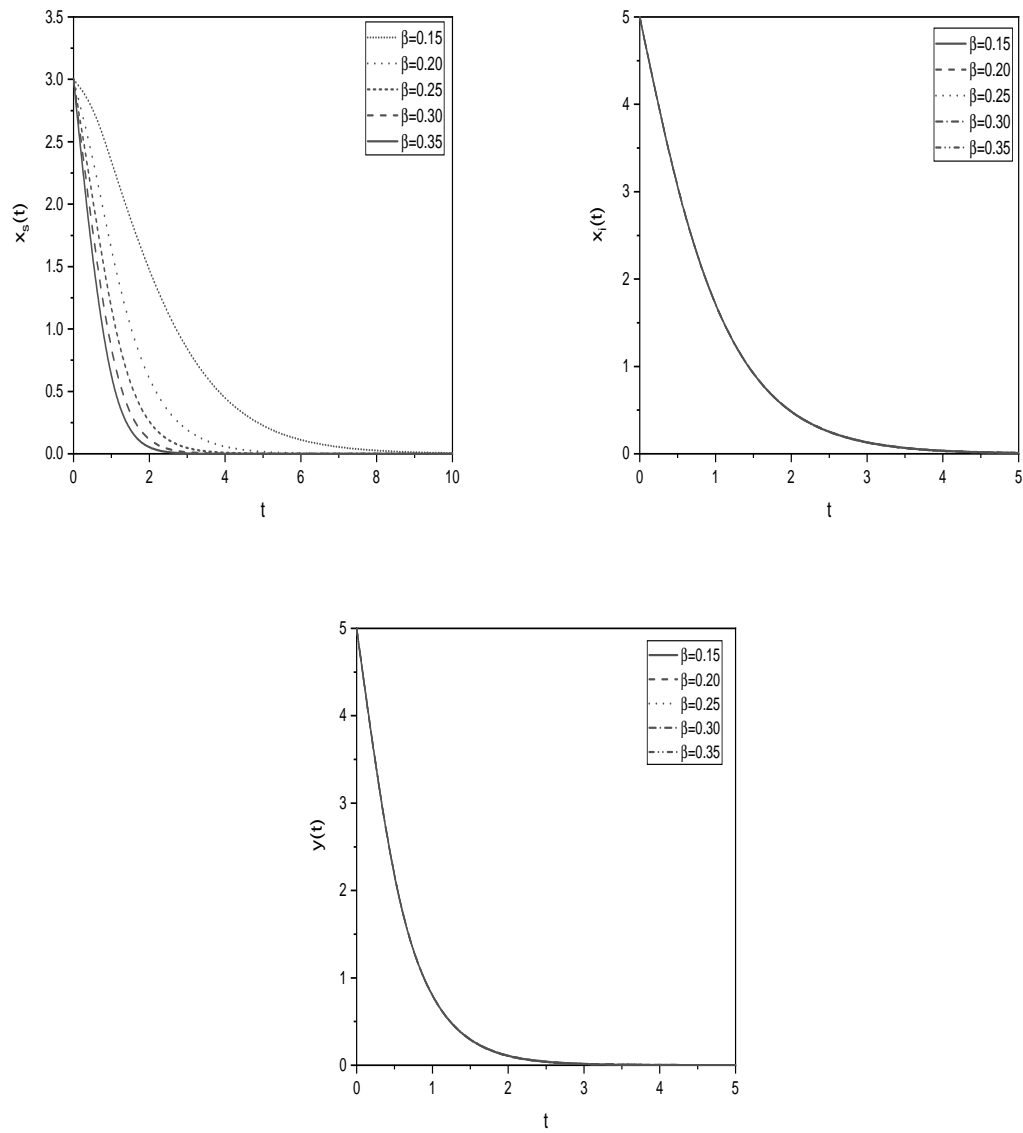


Figure 6.2: Influence of β on Prey - Predator System under impulsive Control.

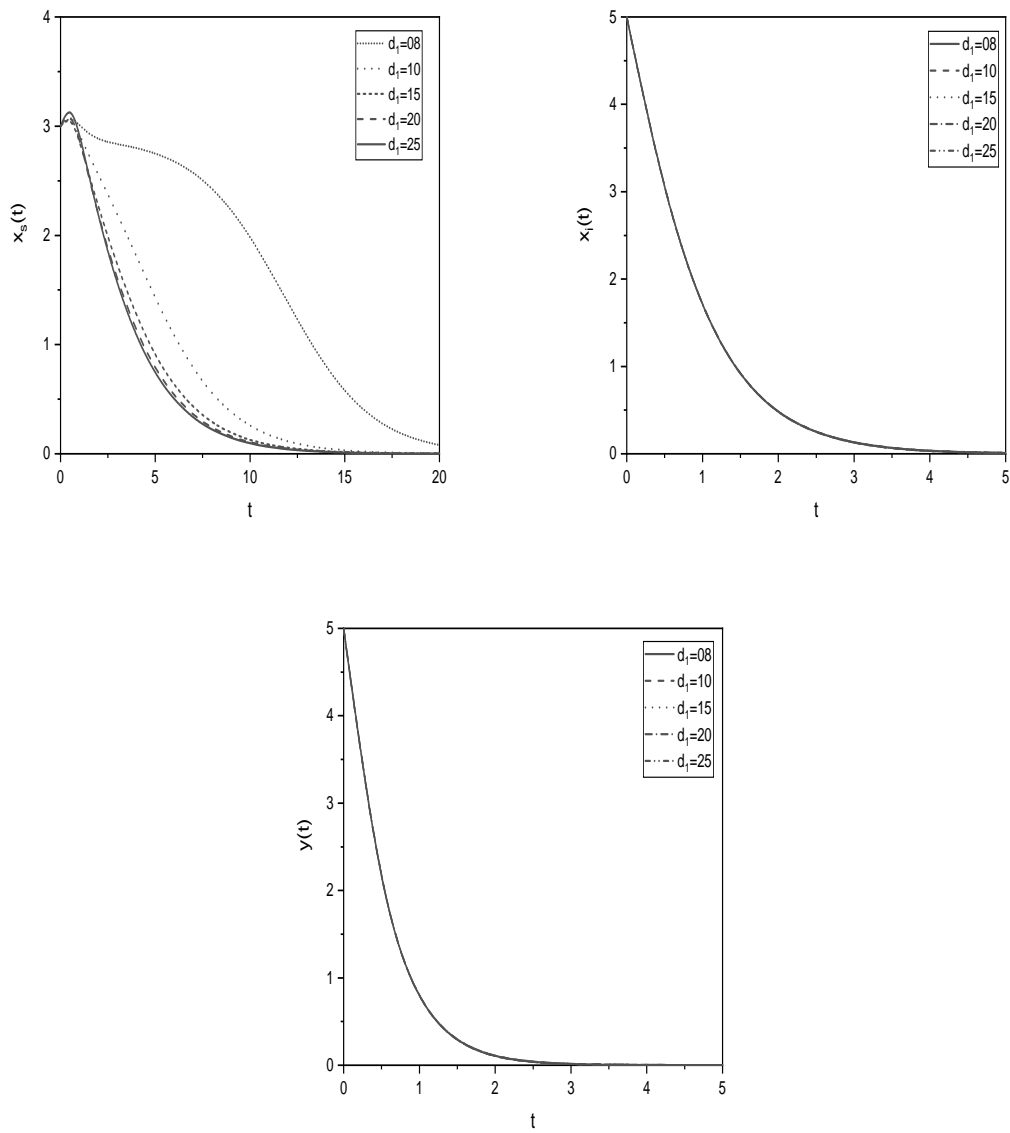


Figure 6.3: Influence of d_1 on Prey - Predator System under impulsive Control.

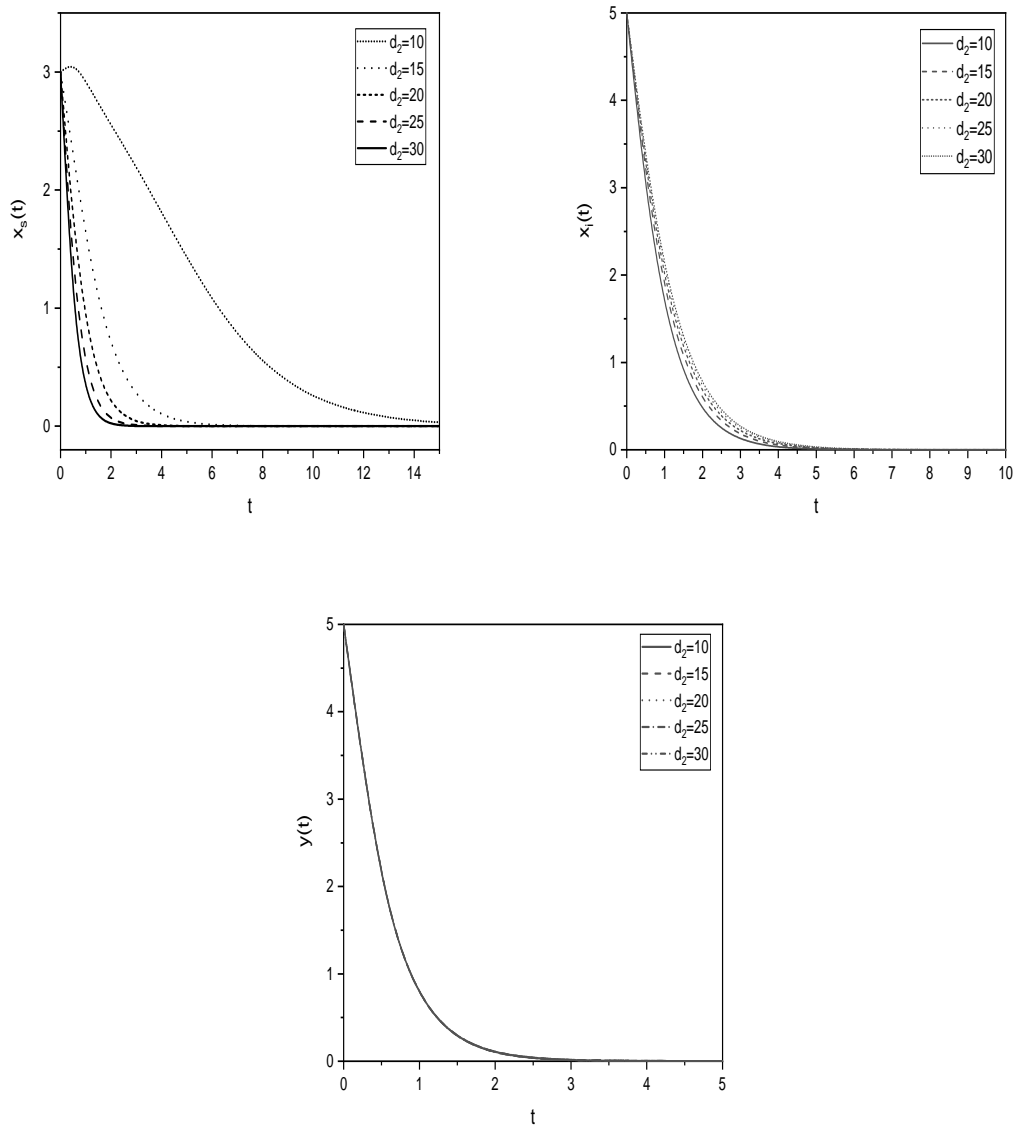


Figure 6.4: Influence of d_2 on Prey - Predator System under impulsive Control.

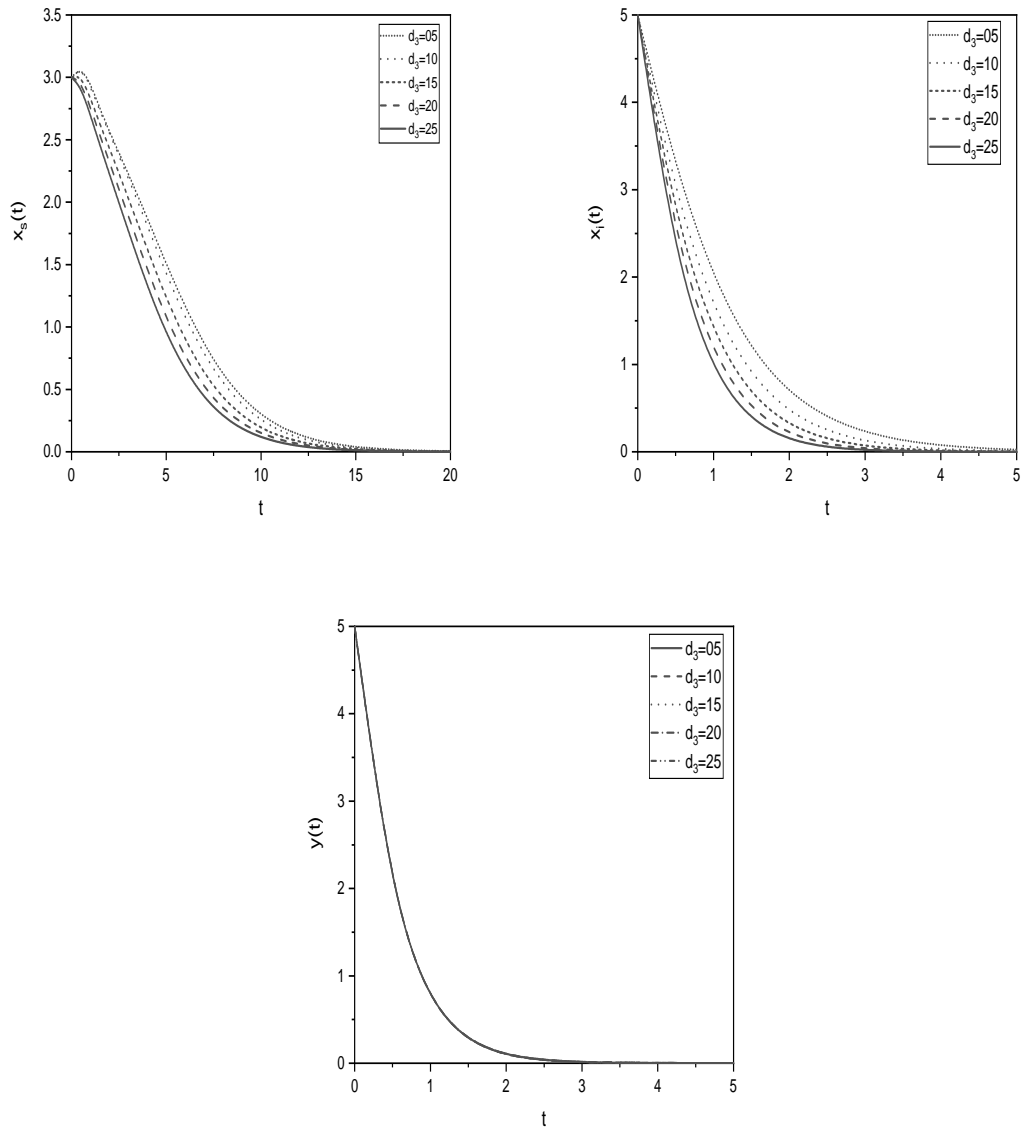


Figure 6.5: Influence of d_3 on Prey - Predator System under impulsive Control.

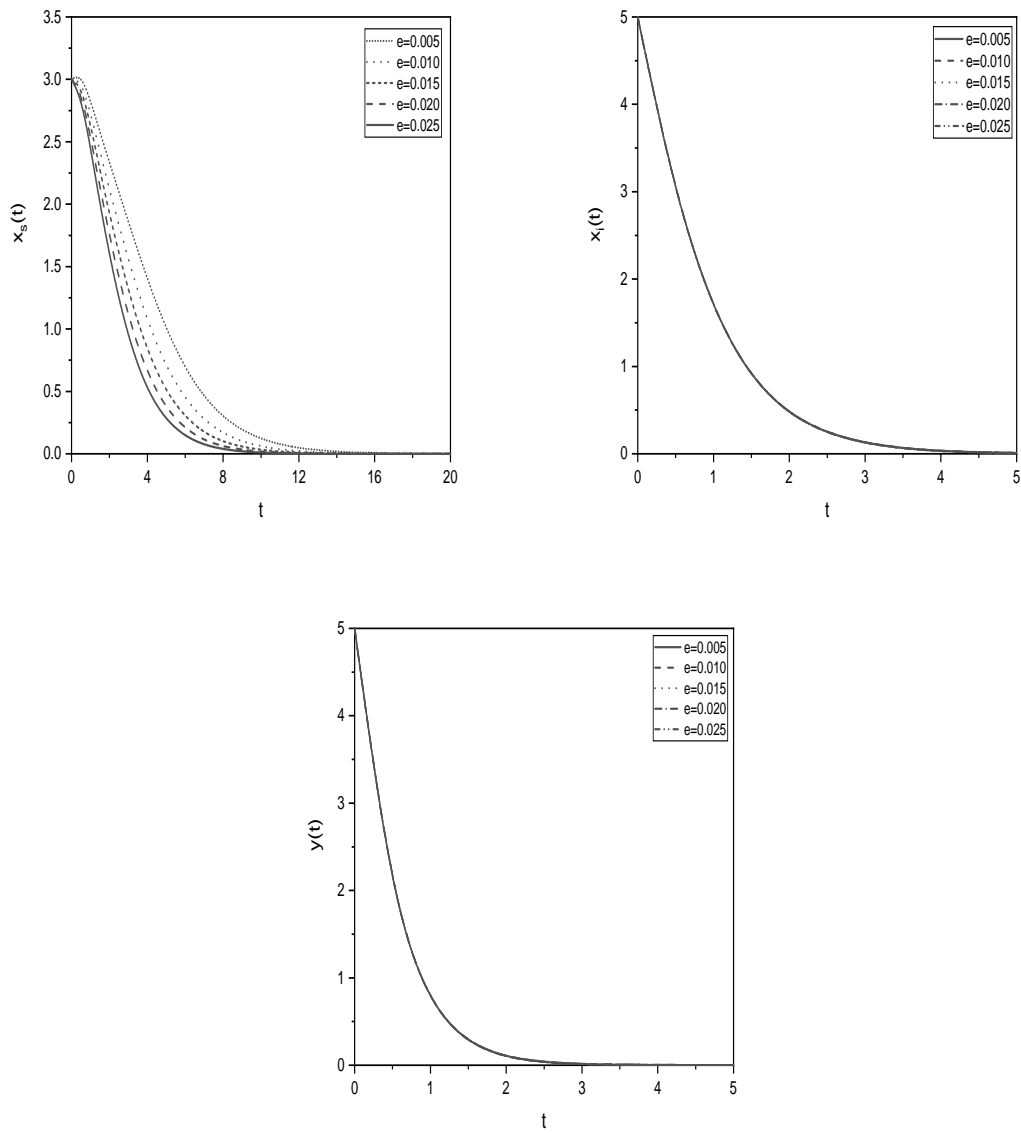


Figure 6.6: Influence of e on Prey - Predator System under impulsive Control.

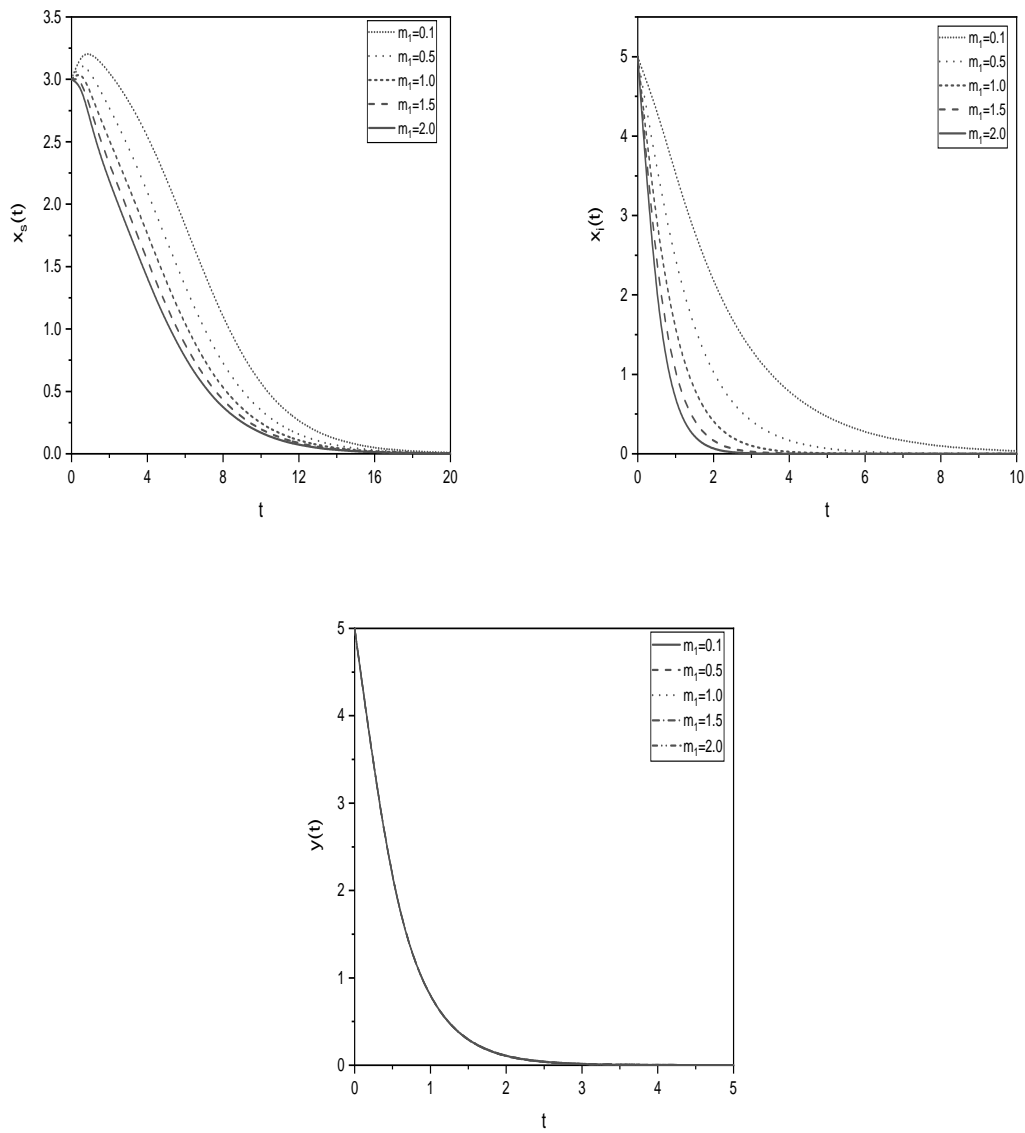
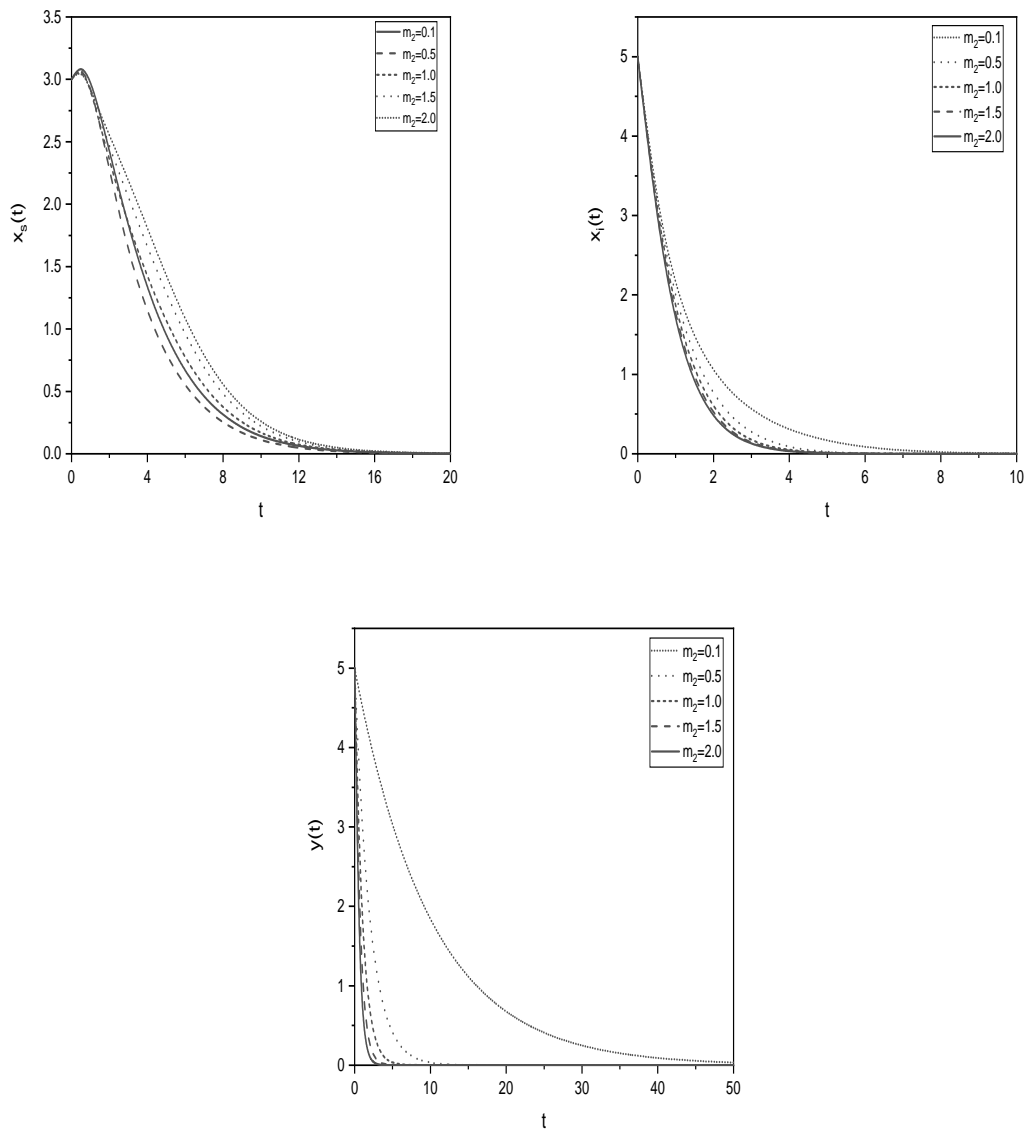


Figure 6.7: Influence of m_1 on Prey - Predator System under impulsive Control.

Figure 6.8: Influence of m_2 on Prey - Predator System under impulsive Control.

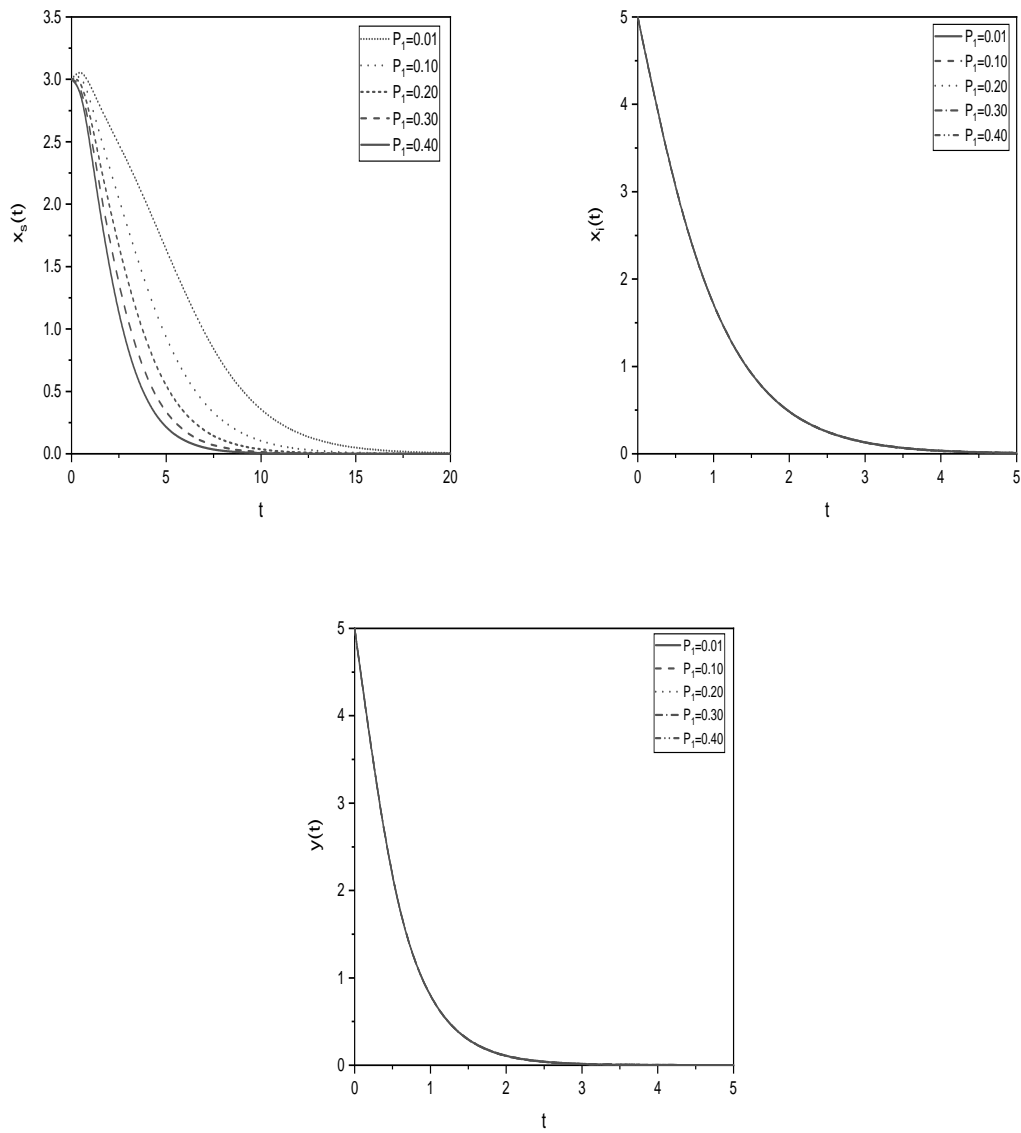
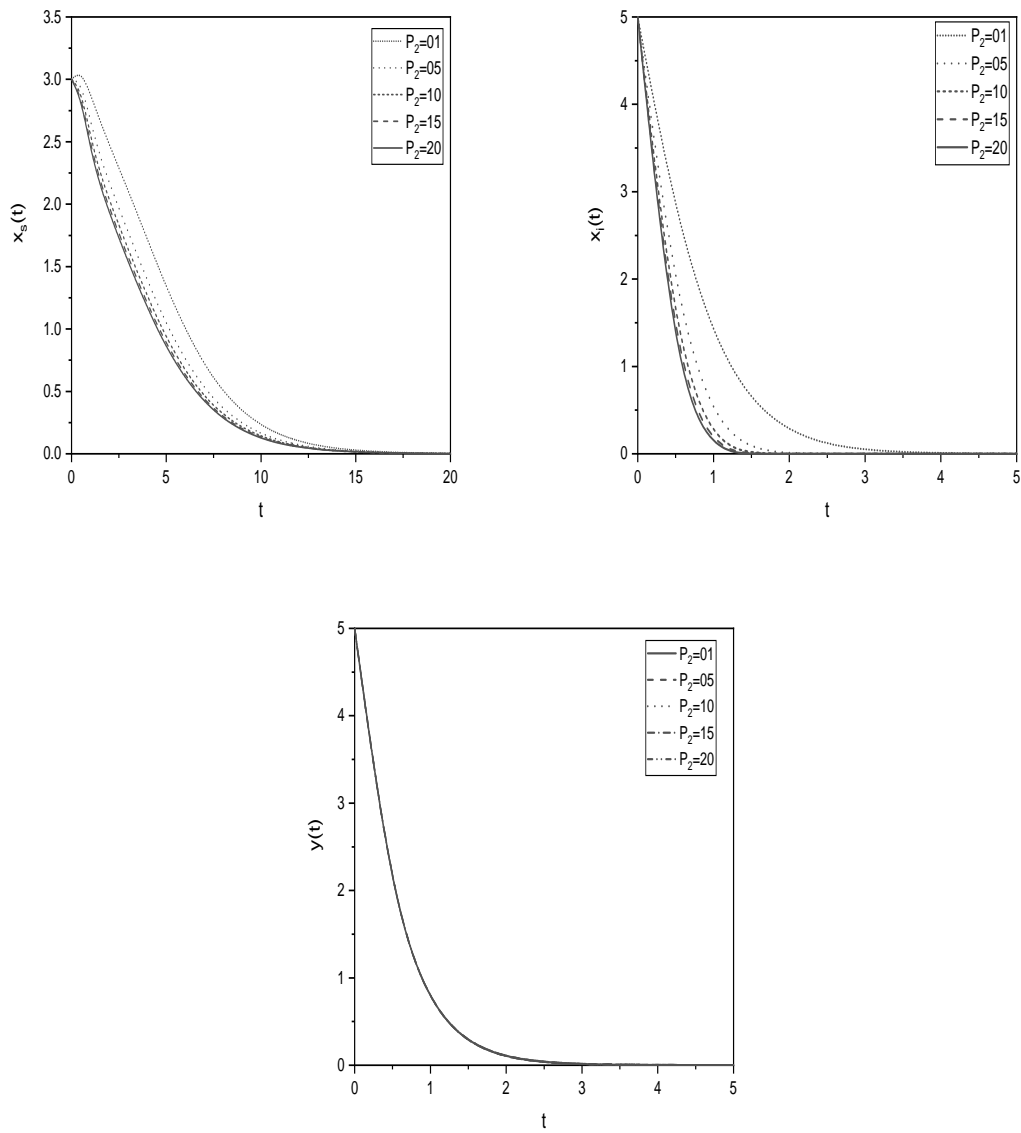


Figure 6.9: Influence of P_1 on Prey - Predator System under impulsive Control.

Figure 6.10: Influence of P_2 on Prey - Predator System under impulsive Control.

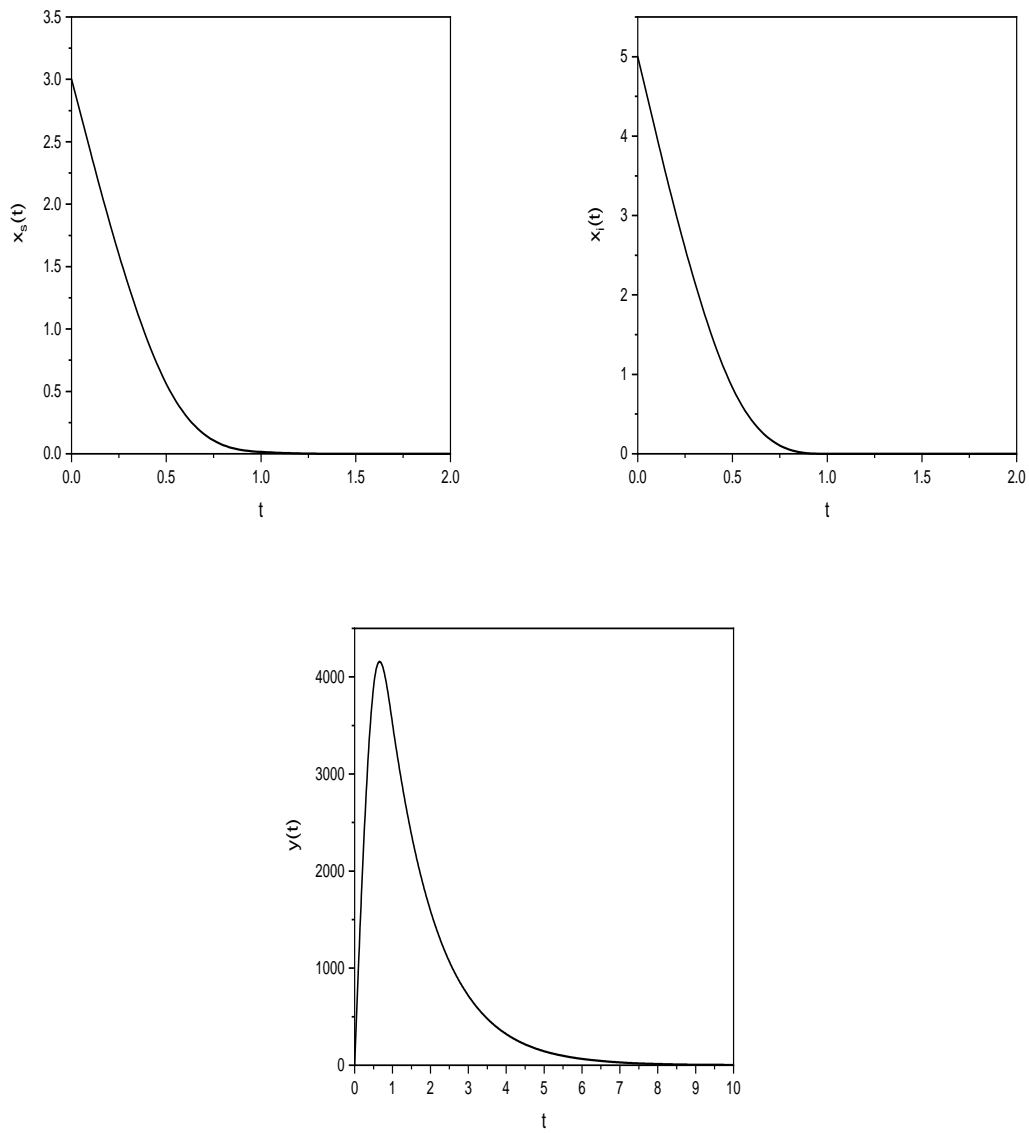


Figure 6.11: Prey- predator system with no impulsive control.

Chapter 7

Stability Analysis of a T-S

Prey-Predator model with disease in both species ¹

7.1 Introduction

We have witnessed rapidly growing interest in fuzzy control in recent years. This is largely sparked by the numerous successful applications in fuzzy control. Despite the visible success, it has been made aware that many basic issues remain to be addressed. Among them, stability analysis, systematic design, and performance analysis are crucial to the validity and applicability of any control design methodology as shown by Huang [98]. However, it should be admitted that the stability of fuzzy logic controller (FLC) is still an open problem. It is important to point out that there exist many systems, like the predator-prey system, which cannot commonly endure continuous control inputs, or they have impulsive dynamical behavior due to abrupt jumps at certain instants during the evolving processes. Hence,

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it is necessary to extend FLC and reflect these impulsive jump phenomena in the predator-prey system.

Until recently, we discussed about the stability of two and three dimensional Lotka-Volterra predator-prey system with fuzzy impulsive control. In this chapter we studied the Lotka-Volterra prey-predator model with the disease in both prey and predator populations. To improve the model's reality we analyze the global and asymptotic stability of this model with the help of the T-S model as explained in earlier chapters. Then presented the graphical solutions for the problem.

7.2 Model Formation

At time t , the densities of susceptible prey, infected prey, susceptible predator, and the infected predators are denoted by $x_s(t)$, $x_i(t)$, $y_s(t)$, and $y_i(t)$ respectively.

- The prey population increases logistically with an intrinsic growth rate (r) and environmental carrying capacity $C = r/e$ ($C > 0$) in absence of predator population and in absence of disease as in Bera et al. [104].
- Only the susceptible prey propagates.
- When a susceptible prey gets into contact with an infected one, the infection is transmitted among the susceptible ones. If infected, the prey never recovers. Either it will die or be eaten away by a predator. Disease related deaths are more common in the population of diseased prey.
- Our presumption is that diseased predators are incapable of catching a healthy prey. Therefore, only a healthy predator can catch a healthy prey. However, because they are weaker and more vulnerable, infected prey can be eaten by both susceptible and infected predators.

- Direct contact with an infected predator enables the disease to spread across the predator population. An infected predator either stays diseased or becomes extinct; it never recovers or develops immunity.
- We suppose that there are natural deaths among the predators, whereas the infected population has a disease induced excess death rate also.

On the basis of above assumptions the following model is proposed by using set of non-linear differential equations.

$$\begin{aligned}
\frac{dx_s}{dt} &= rx_s - ex_s^2 - ex_sx_i - \beta_1x_sx_i - P_1x_sy_s \\
\frac{dx_i}{dt} &= \beta_1x_sx_i - m_1x_i - P_2y_sx_i - P_3x_iy_i \\
\frac{dy_s}{dt} &= C_1x_sy_s + C_2x_iy_s - \beta_2y_iy_s - Dy_s \\
\frac{dy_i}{dt} &= \beta_2y_sy_i - (D + m_2)y_i + C_3x_iy_i
\end{aligned} \tag{7.1}$$

where x_{s0} , x_{i0} , y_{s0} , y_{i0} are the initial populations and all the parameters are positive with initial conditions are $x_s = x_{s0} > 0$, $x_i = x_{i0} > 0$, $y_s = y_{s0} > 0$, $y_i = y_{i0} > 0$. Here r is the intrinsic growth rate of prey, e is the intra-specific competition, β_1 is the infection coefficient of healthy prey, P_1 is the predation rate of healthy prey by healthy predator, m_1 is the disease induced death rate of infected prey, P_2 is the predation rate of infected prey by healthy predator, P_3 is the predation rate of infected prey by infected predator, C_1 is the conversion rate of healthy prey to healthy predator, C_2 is the conversion rate of infected prey to healthy predator, β_2 is the infection rate for predator population, D is the death rate for the predator population, m_2 is the disease induced death rate of infected predator, C_3 is the conversion rate of infected prey to infected predator.

A matrix differential equation is stated as follows to analyze the system's stability:

$$\dot{x} = Ax + \phi(x), \quad (7.2)$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}_s(t) \\ \dot{x}_i(t) \\ \dot{y}_s(t) \\ \dot{y}_i(t) \end{pmatrix}, A = \begin{bmatrix} r & 0 & 0 & 0 \\ 0 & -m_1 & 0 & 0 \\ 0 & 0 & -D & 0 \\ 0 & 0 & 0 & -(D + m_2) \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex_s^2 - ex_sx_i - \beta_1x_sx_i - P_1x_sy_s \\ -\beta_1x_sx_i - P_2y_sx_i - P_3x_iy_i \\ C_1x_sy_s + C_2x_iy_s - \beta_2y_sx_i \\ \beta_2y_iy_s + C_3x_iy_i \end{bmatrix};$$

7.3 T-S Fuzzy model with Impulsive effects

The non-linear equations can be transformed into the following linear equation as explained in earlier (chapter-2).

If $x(t)$ is M_i then

$$\begin{aligned} \dot{x}(t) &= A_i x(t), t \neq \tau_j; \\ \Delta(x) &= K_{ij} x(t), t = \tau_j; \\ i &= 1, 2, 3 \dots r; j = 1, 2, \dots \end{aligned} \quad (7.3)$$

$$\text{where, } A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 - z_4 & 0 & 0 & 0 \\ z_3 & -z_5 - z_6 - m_1 & 0 & 0 \\ z_7 & z_8 & -z_9 - D & 0 \\ 0 & z_{10} & z_9 & -(D + m_2) \end{bmatrix}, i = 1$$

to 1024, where the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 10, here z'_k s are related to the values of $x_s(t) \in [0, d_1]$, $y_s(t) \in [0, d_2]$, $x_i(t) \in [0, d_3]$, $y_i(t) \in [0, d_4]$ (here $z_1 = ex_s$, $z_2 = ex_i$, $z_3 = \beta_1 x_i$, $z_4 = P_1 y_s$, $z_5 = P_2 y_s$, $z_6 = P_3 y_i$, $z_7 = C_1 y_s$, $z_8 = C_2 y_s$, $z_9 = \beta_2 y_i$, $z_{10} = C_3 y_i$). M_i , $x(t)$, $A_i \in R^{4 \times 4}$, r is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta(x)|_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

7.4 Numerical Simulation

Since most of the biological systems are complex, they should be expressed by applying a fuzzy logical framework that includes expressive reports. The suggested impulsive T-S design model examines predator-prey system with functional response and impulsive impact.

By using fuzzy impulsive T-S design model on (7.2), the membership functions $M'_{ij}s$ and the matrices $A'_i s$, are calculated using

$$A_i = \begin{bmatrix} r - z_1 - z_2 - z_3 - z_4 & 0 & 0 & 0 \\ z_3 & -z_5 - z_6 - m_1 & 0 & 0 \\ z_7 & z_8 & -z_9 - D & 0 \\ 0 & z_{10} & z_9 & -(D + m_2) \end{bmatrix},$$

$i = 1$ to 1024, where the matrices $A'_i s$ are generated using maximum and minimum values of $z'_k s$; $k = 1$ to 10 and, the Defuzzification can be represented as:

$$\dot{x}(t) = \sum_{i=1}^r h_i(z(t))(A_i x(t)), \quad (7.4)$$

here $h'_i s$ are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$,
 $i=1$ to 1024, $j=1$ to 10

This Fuzzy model exactly represents the non-linear system (7.2) in the region $[0,10] \times [0,10] \times [0,10] \times [0,10]$

7.5 Results and discussion

In this section, the global stability of the considered intra-specific competition prey-predator model (7.1) is discussed as explained in earlier chapters. We have studied the system (7.1) numerically using MATHEMATICA software to get better insight of the proposed model. Calculations were carried by taking the values of the parameters

at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$ in 7.3 to get the eigen values of $[A_i^T + A_i]$ ($i = 1, 2, 3 \dots r$) as explained in the theorems ([83]). It is found that $\max(\lambda_i) = \lambda(\alpha) = 14.8$, then we have chosen $\text{diag}[-0.84, -0.84]$ as impulsive control matrix, such that $\omega = \|I + K\| = 0.16$. It is noted that the system 7.3 is stable globally when $\epsilon=1.5$, $\delta_j=0.02$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -1.142 < 0$). Further, it is observed that the prey-predator model is unstable when $r = 0.1$, $e = 0.5$, $\beta_1 = 0.2$, $P_1 = 4$, $P_2 = 0.5$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1$, $D = 0.5$, $m_2 = 0.5$, $C_3 = 8$, $d_1 = 10$, $C_2 = 0.5$, $\beta_2 = 0.4$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, since $\max(\lambda_i) = \lambda(\alpha) = 79.5$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.17 > 0$ for $\omega = 0.16$, $\epsilon=1.5$, $\delta_j=0.02$.

The stability of the system at various values of this study is presented in 7.1.

r	e	β_1	P_1	P_2	m_1	C_1	P_3	D	m_2	C_3	d_1	C_2	β_2	d_2	d_3	d_4	$\max(\lambda_i)$ $=\lambda(\alpha)$	$\ln(\epsilon\omega)$ $+\lambda(\alpha)\delta_j$	conclusion
0.8	0.05	0.4	1	0.1	0.5	0.1	1.5	0.2	0.5	1.4	10	0.15	0.2	10	10	10	14.8	-1.142	stable
0.5	0.25	0.1	2	0.2	1	0.5	0.5	0.1	0.2	1	10	0.2	0.1	10	10	10	10.5	-1.21	stable
1.0	0.4	0.1	0.5	1.0	0.1	0.2	2.0	1.5	0.5	0.1	10	1.5	0.5	10	10	10	13.5	-1.15	stable
0.1	0.5	0.2	4	0.5	0.5	0.1	1	0.5	0.5	8	10	0.5	0.4	10	10	10	79.5	0.17	unstable

Table 7.1: Stability of the system at various parameters

The impact of various emerging parameters on prey-predator system (7.1) with T-S fuzzy impulsive control model is presented in figs. 7.1 - 7.12.

The effect of intrinsic growth rate of prey (r) on prey-predator system is shown in fig. 7.1 at $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly displays that the growth of healthy prey increases with increase in r and it has no impact on infected prey, and predators.

The dynamical behavior of the two species population (x_s, x_i, y_s, y_i) under the influence of intra-specific competition parameter (e) on prey-predator system is shown in fig. 7.2 at $r = 0.8$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$,

$D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly exhibits that the population of healthy prey decreases with an increase in e whereas the infected prey, healthy predator, infected predator population were not affected.

The effect of infection coefficient of healthy prey (β_1) on prey-predator system is shown in fig. 7.3 at $r = 0.8$, $e = 0.05$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly displays that an increase in the infection rate decreases healthy prey population.

The nature of prey-predator (x_s, x_i, y_s, y_i) population with the effect of (P_1) on prey-predator system is shown in fig. 7.4 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. It is observed from this figure that healthy prey population decreases with an increase in P_1 .

The influence of predation rate of infected prey by healthy predator (P_2) on prey-predator system is shown in fig. 7.5 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. From this figure it is clear that the population of healthy and infected prey decreases with an increase in P_2 and predator population remains unaffected.

The effectiveness of infection of predator population (β_2) on prey-predator system is shown in fig. 7.6 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. It shows that healthy predator population decreases with increasing infection in predator. Healthy prey and infected prey populations decreases slightly with an increase in β_2 .

The influence of predation rate of infected prey by infected predator (P_3) on

prey-predator system is shown in fig. 7.7 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure displays that prey population (healthy and infected both) decreases with an increase in P_3 .

The effect of conversion of infected prey to infected predator (C_3) is shown in fig. 7.8 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. In this figure clearly exhibits that the population of both prey and predators (healthy and infected) decreases with an increase in C_3 .

The dynamical pattern of prey - predator population (x_s, x_i, y_s, y_i) by varying prey max time (d_1) is shown in fig. 7.9 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly shows that as we increase prey's (healthy prey) max time d_1 , the population of all species (i.e healthy prey and predator, infected prey and predator) decreases.

The trend of four species (x_s, x_i, y_s, y_i) population with respect to infected prey max time (d_2) is shown in fig. 7.10 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_3 = 10$, $d_4 = 10$. It can be seen from this figure that as increase in infected prey's max time d_2 , the population of all the species (i.e healthy prey and predator, infected prey and predator) decreases.

The change on prey- predator population (x_s, x_i, y_s, y_i) by varying susceptible predator max time (d_3) is shown in fig. 7.11 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_4 = 10$. This figure clearly shows that as an increase in predator's (healthy predator) max time d_3 , the population of all the species (i.e. healthy prey and predator, infected prey and predator) decreases.

The impact of max time (d_4) on prey-predator system is shown in fig. 7.12 at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$. It can be noted from this figure that, increase in predator's (infected predator) max time d_4 leads to decrease in the population for all species (i.e healthy prey and predator, infected prey and predator).

Finally, the nature of four species (x_s, x_i, y_s, y_i) population (without impulsive control) is presented in fig. 7.13 by fixing all the parameters obtained from T-S fuzzy model at $r = 0.8$, $e = 0.05$, $\beta_1 = 0.4$, $P_1 = 1$, $P_2 = 0.1$, $m_1 = 0.5$, $C_1 = 0.1$, $P_3 = 1.5$, $D = 0.2$, $m_2 = 0.5$, $C_3 = 1.4$, $d_1 = 10$, $C_2 = 0.15$, $\beta_2 = 0.2$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $t = 10$. The figure clearly shows how the prey and predator populations reach to stability.

7.6 Conclusions

In this chapter, we studied the stability analysis of a prey-predator system with fuzzy impulsive control by T-S fuzzification. In which disease infection is on both prey and predators. The main results of this study are as follows:

- The increase in intra-specific competition leads to a decrease in the susceptible prey population.
- The prey population rises in response to an increase in the prey growth rate.
- A higher infection transmission of prey results in a smaller prey population and a higher infection transmission of predator results in a smaller predator population.
- Effect of susceptible prey max time is to decrease all the four populations.

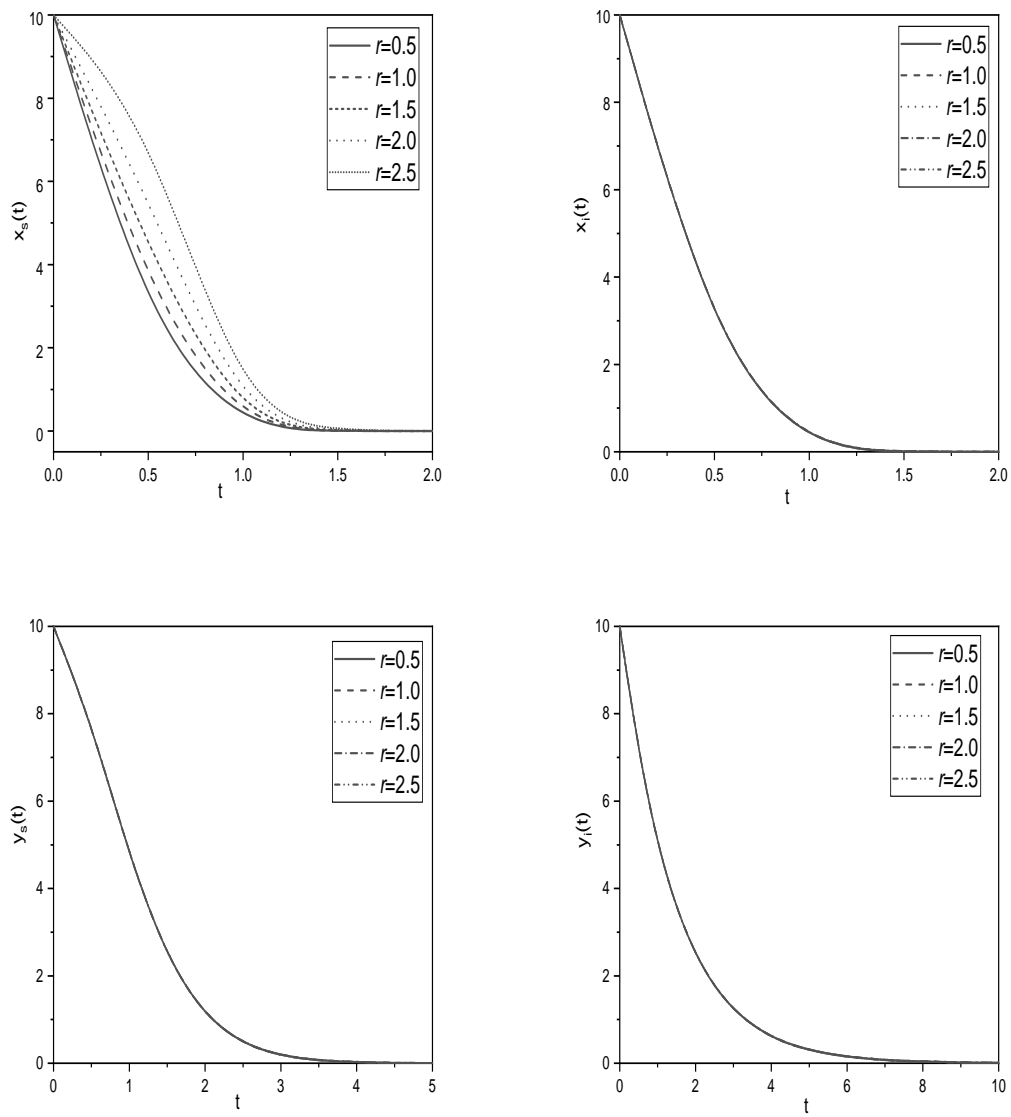


Figure 7.1: Effect of intrinsic growth rate of prey (r) on prey-predator system under impulsive control.

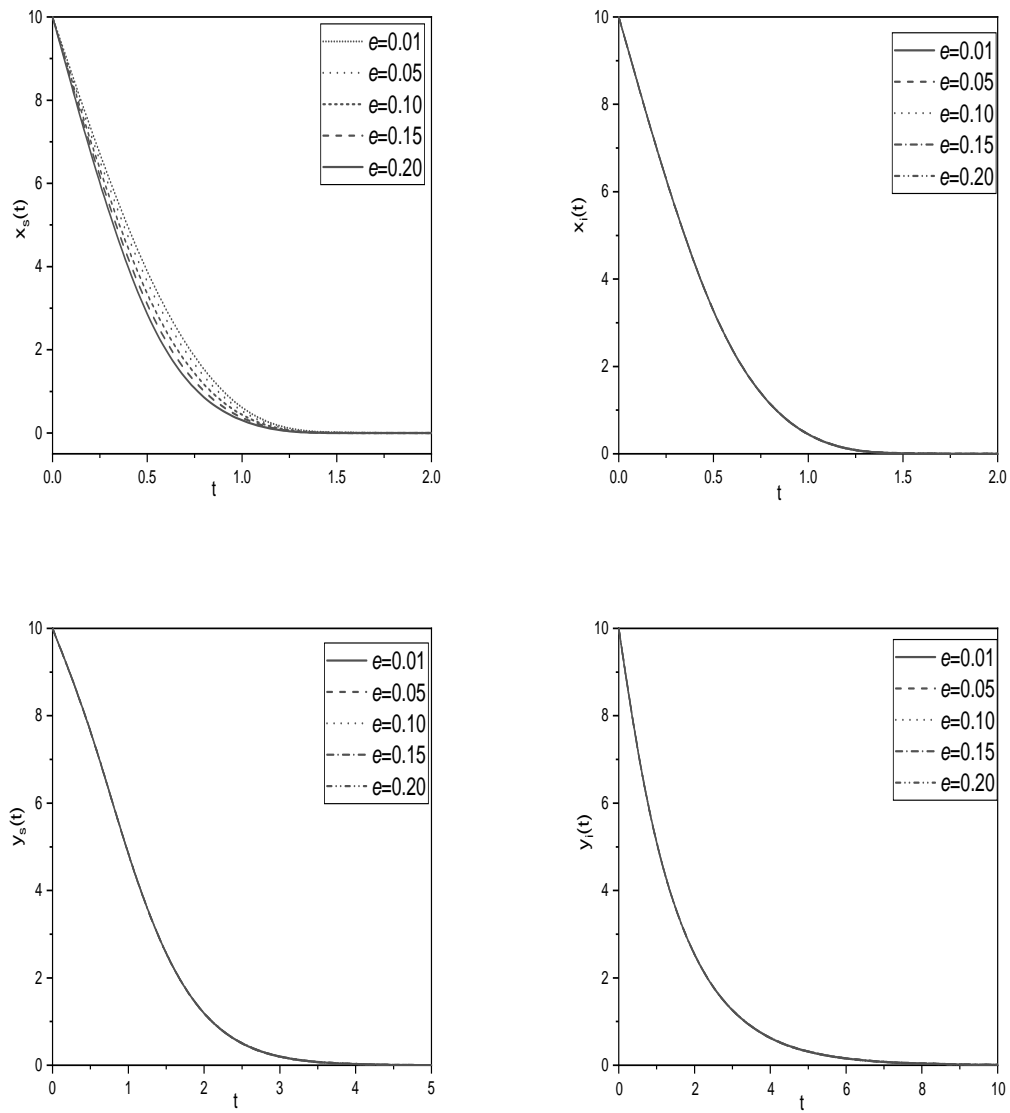


Figure 7.2: Effect of intra-specific competition (e) on prey-predator system under impulsive control

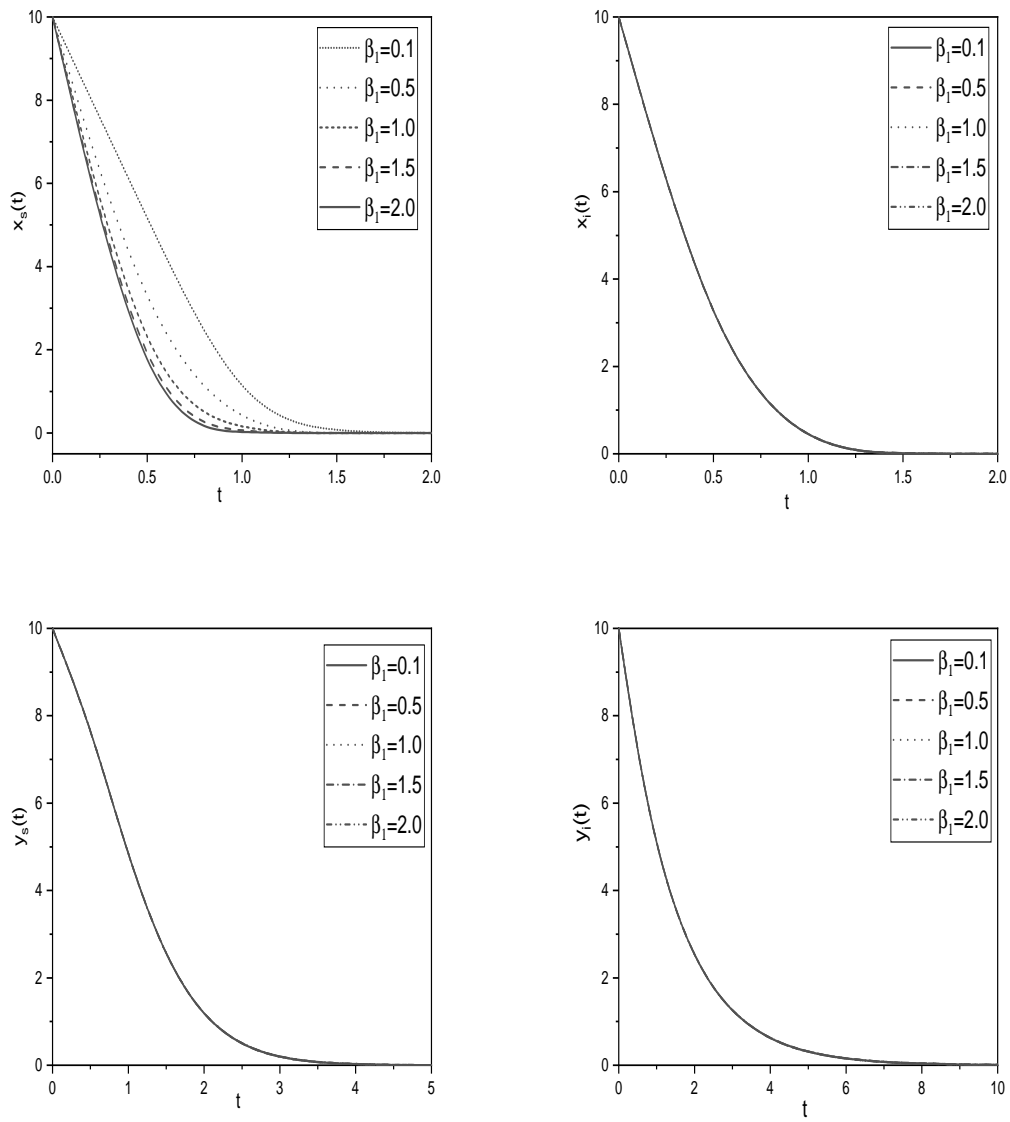


Figure 7.3: Effect of Infection coefficient of healthy prey (β_1) on prey-predator system under impulsive control

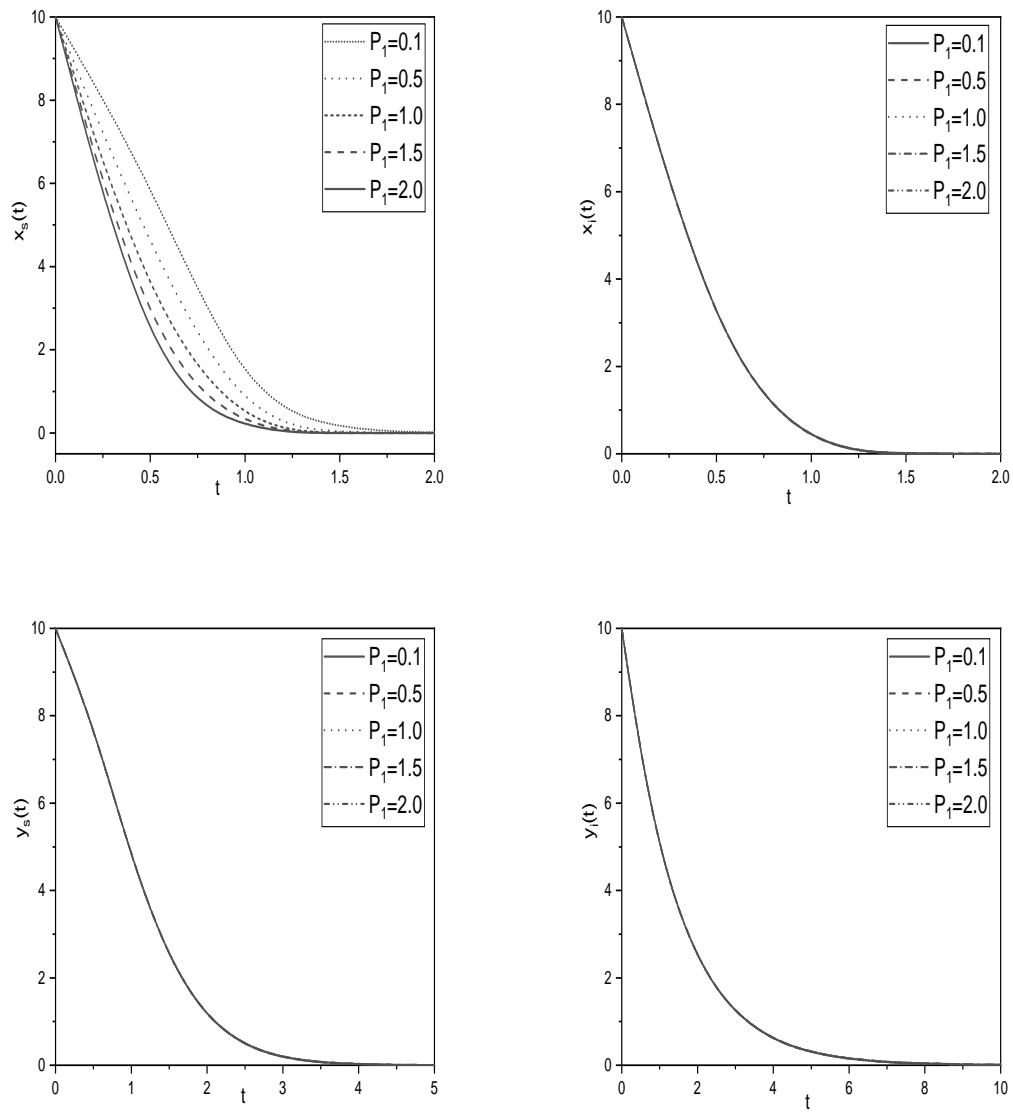


Figure 7.4: Effect of Predation rate of healthy prey by healthy predator (P_1) on prey-predator system under impulsive control

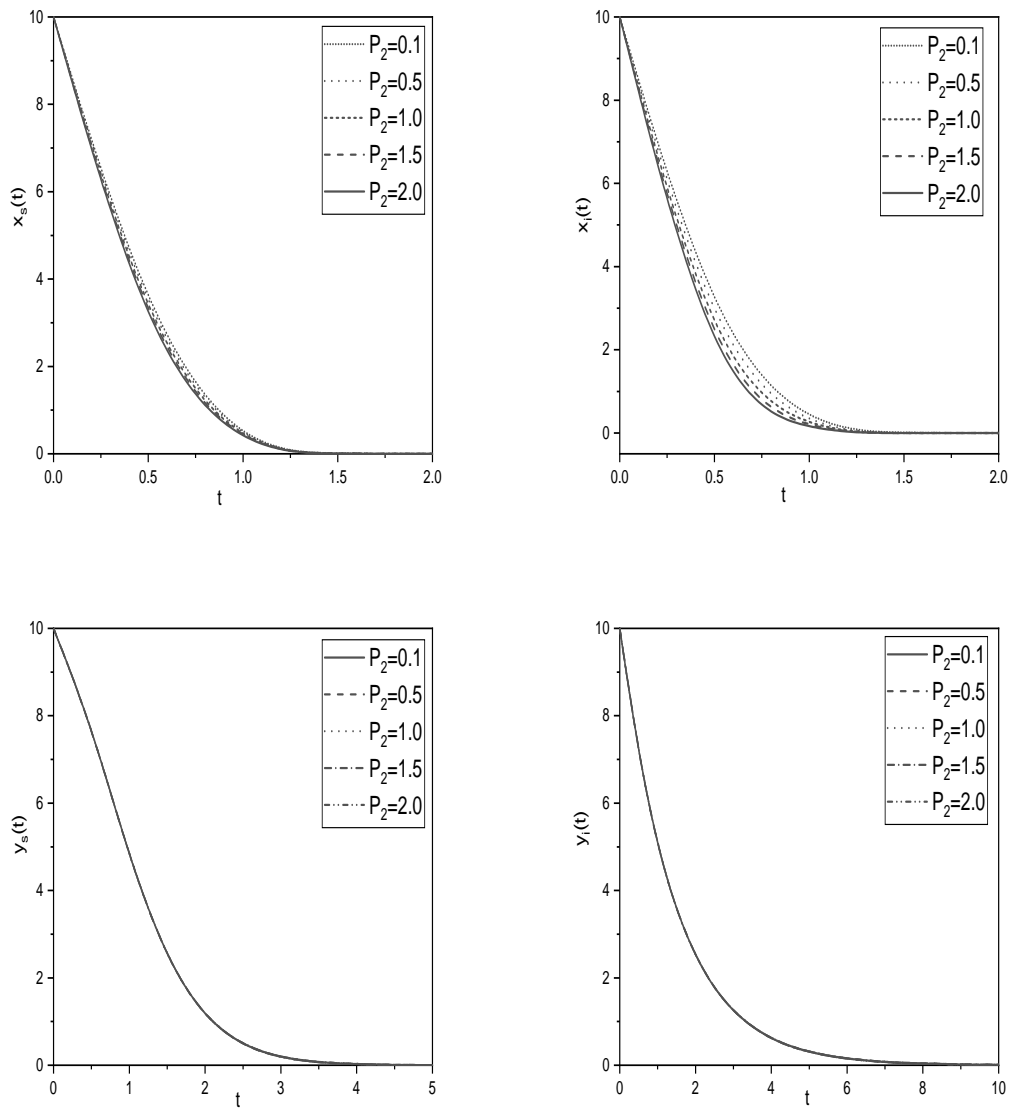


Figure 7.5: Effect of Predation rate of infected prey by healthy predator (P_2) on prey-predator system under impulsive control

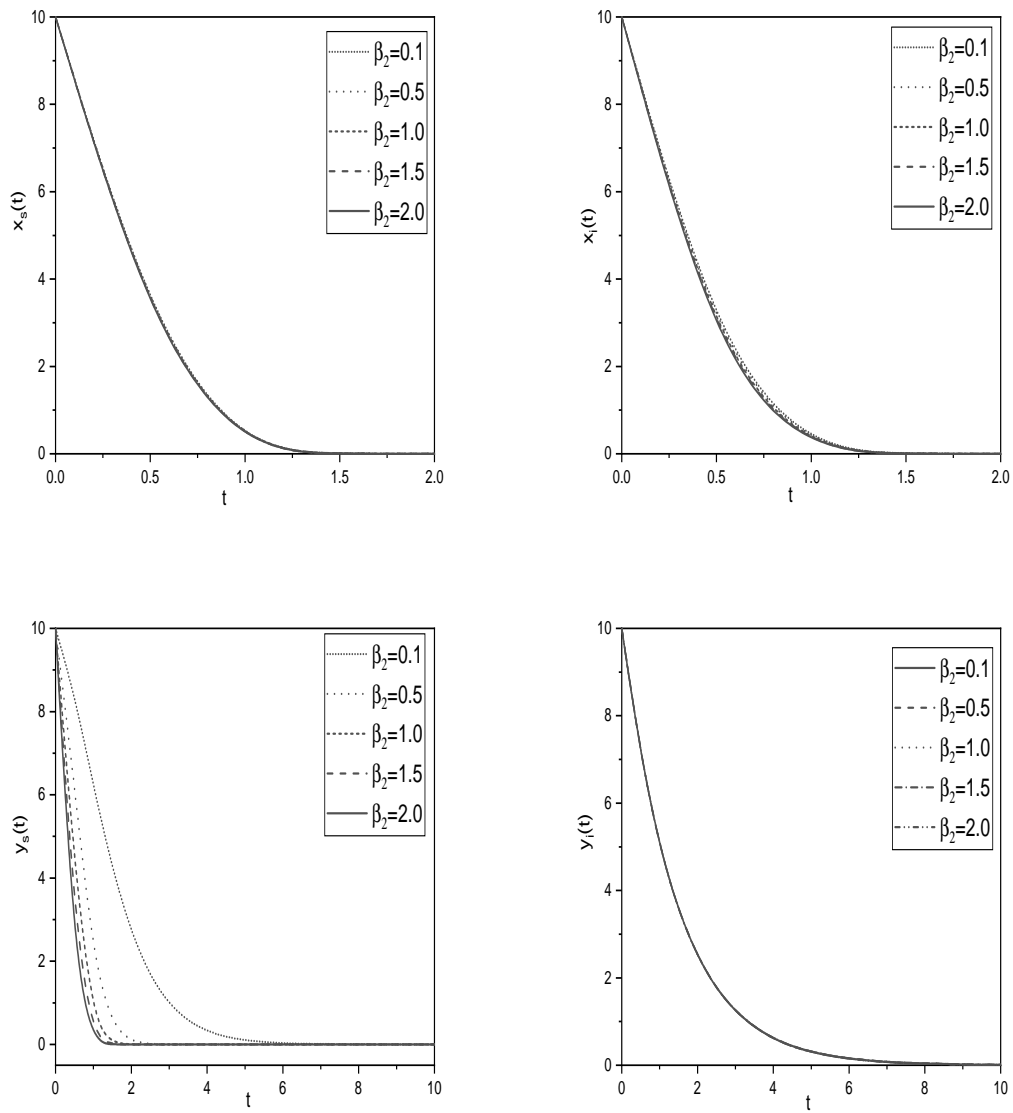


Figure 7.6: Effect of Infection for predator population (β_2) on prey-predator system under impulsive control

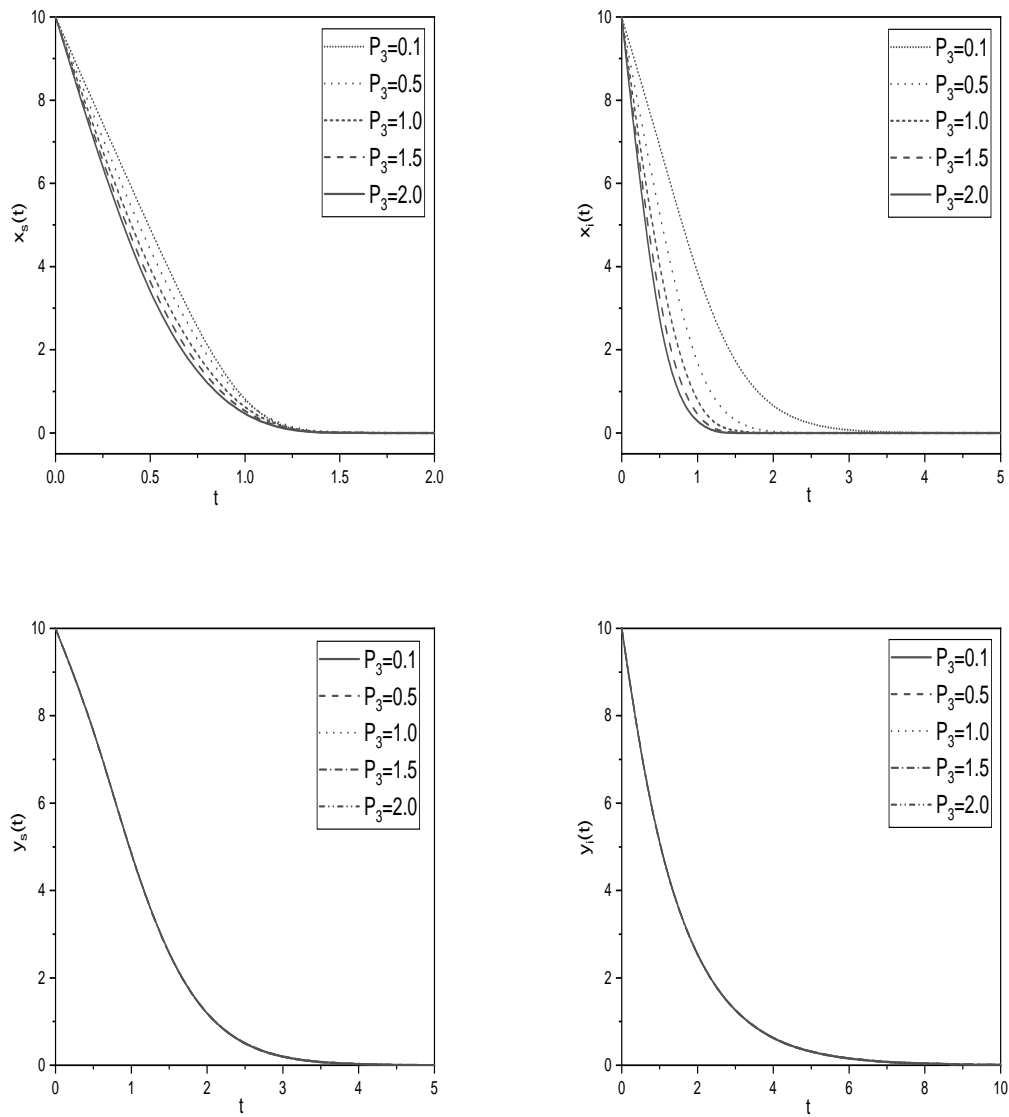


Figure 7.7: Effect of Predation rate of infected prey by infected predator (P_3) on prey-predator system under impulsive control

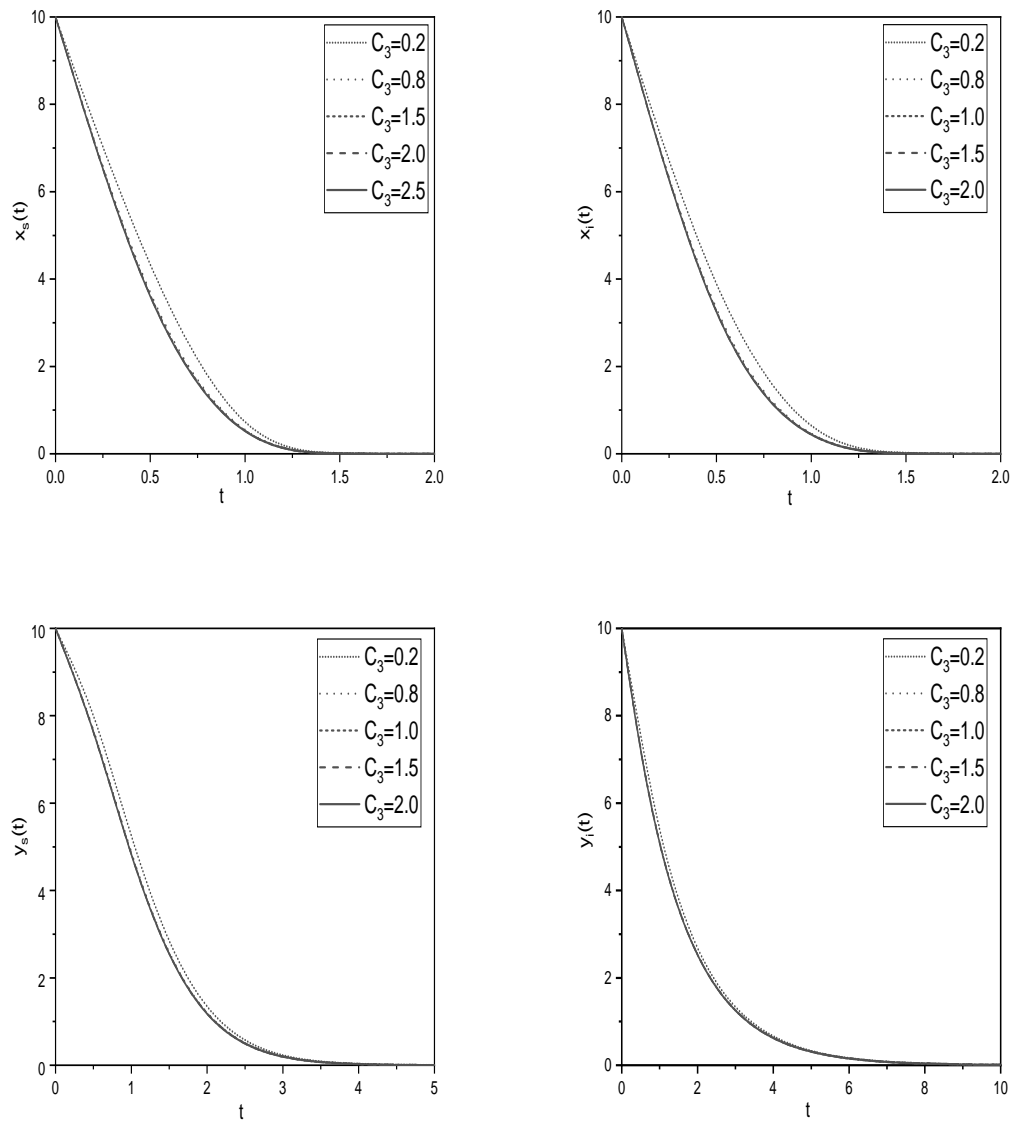
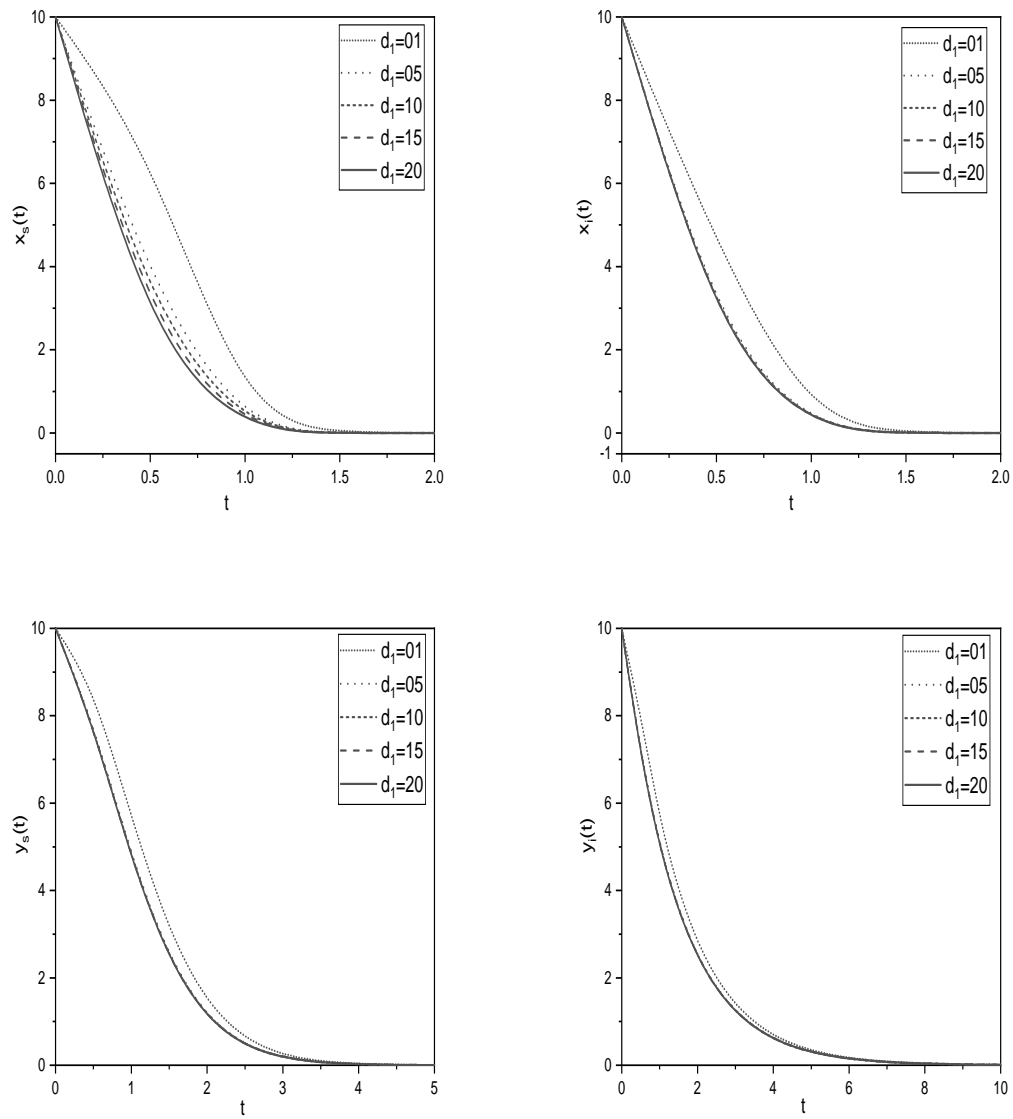
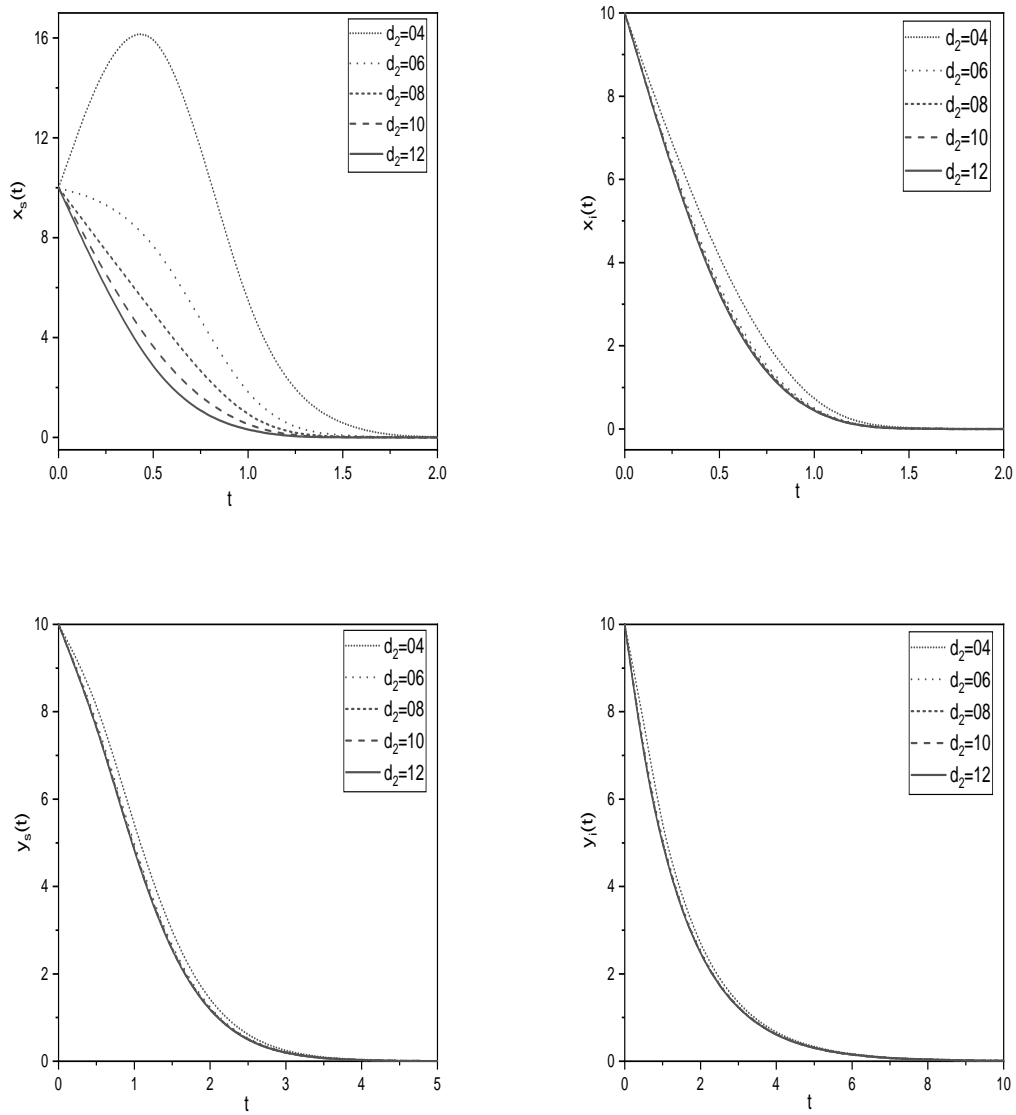
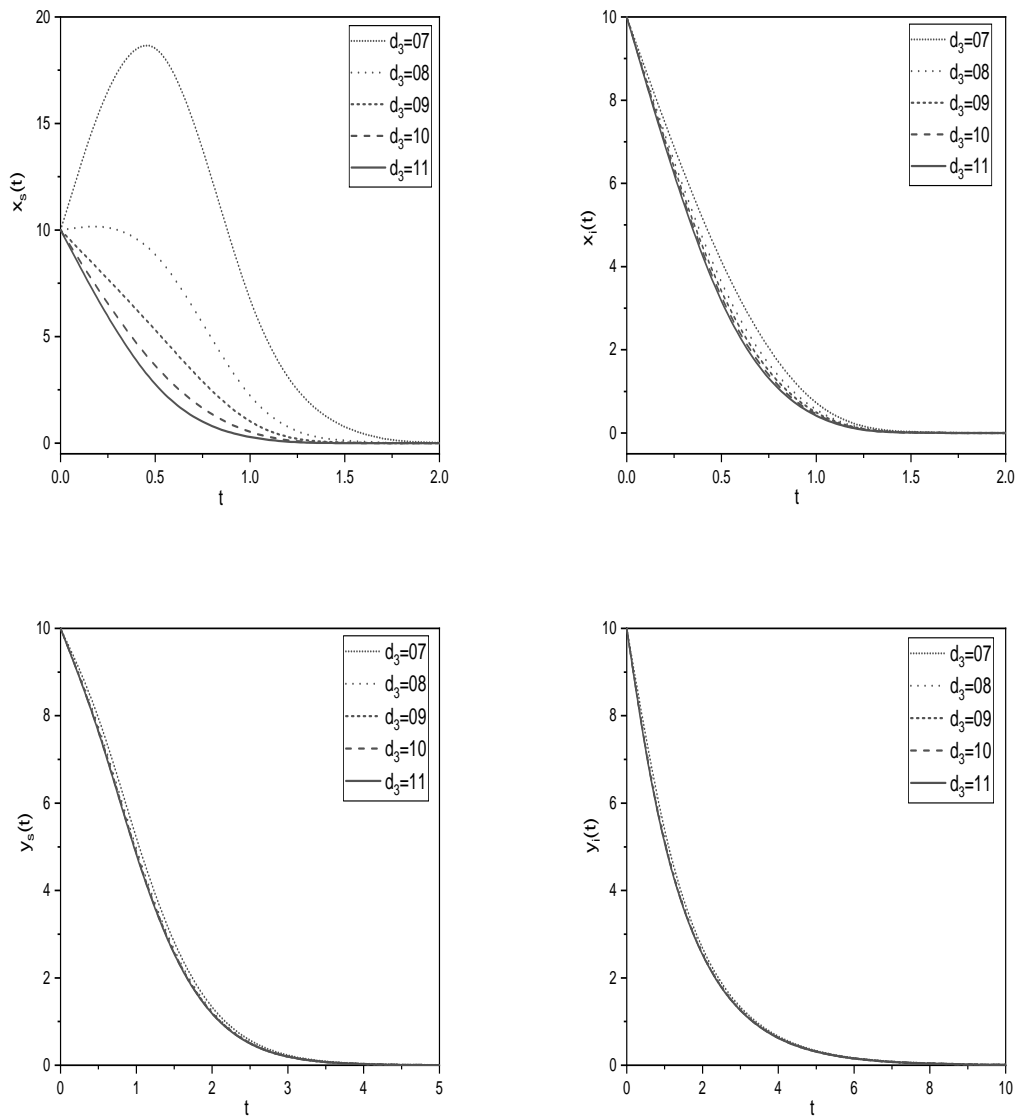
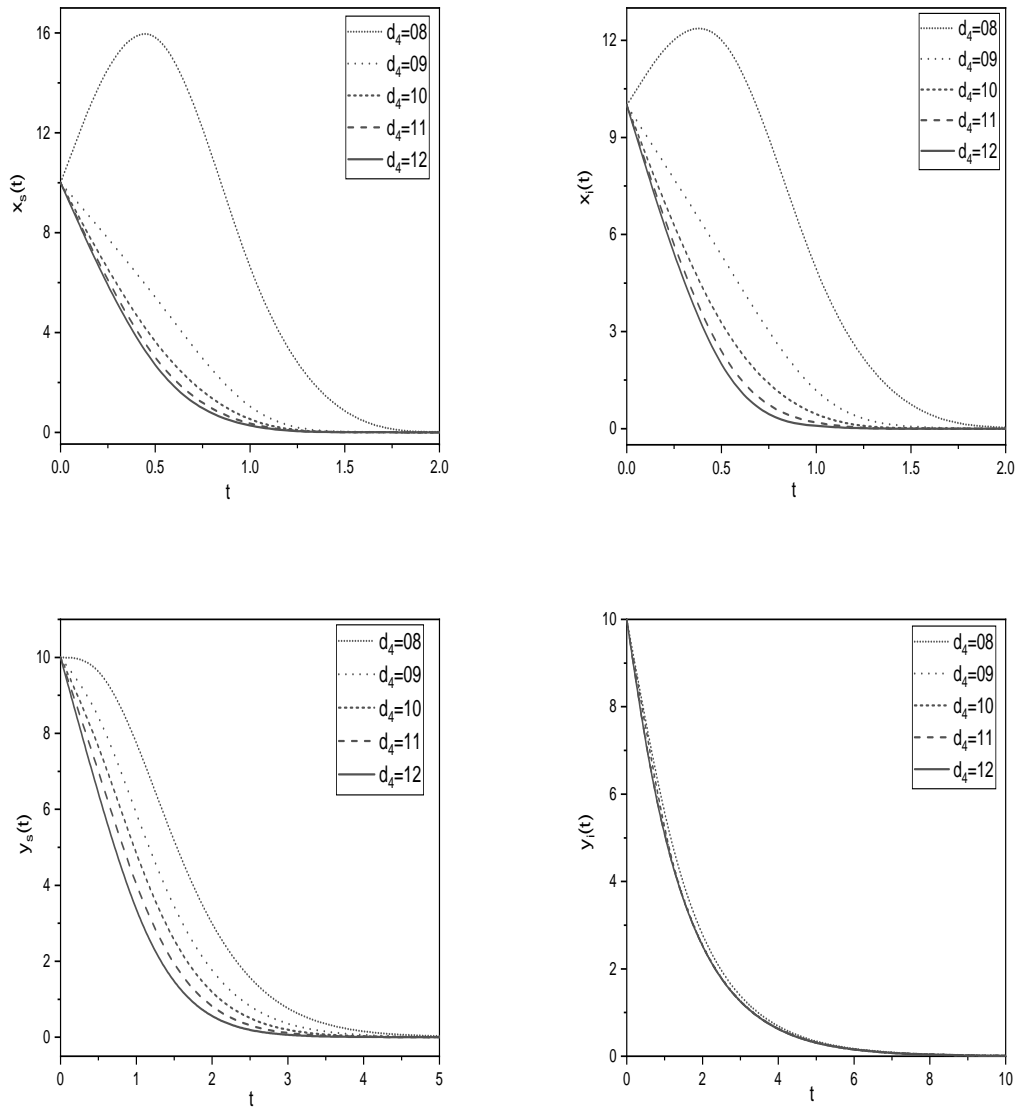


Figure 7.8: Effect of Conversion of infected prey to infected predator (C_3) on prey-predator system under impulsive control

Figure 7.9: Effect of max time (d_1) on prey-predator system under impulsive control

Figure 7.10: Effect of max time (d_2) on prey-predator system under impulsive control

Figure 7.11: Effect of max time (d_3) on prey-predator system under impulsive control

Figure 7.12: Effect of max time (d_4) on prey-predator system under impulsive control

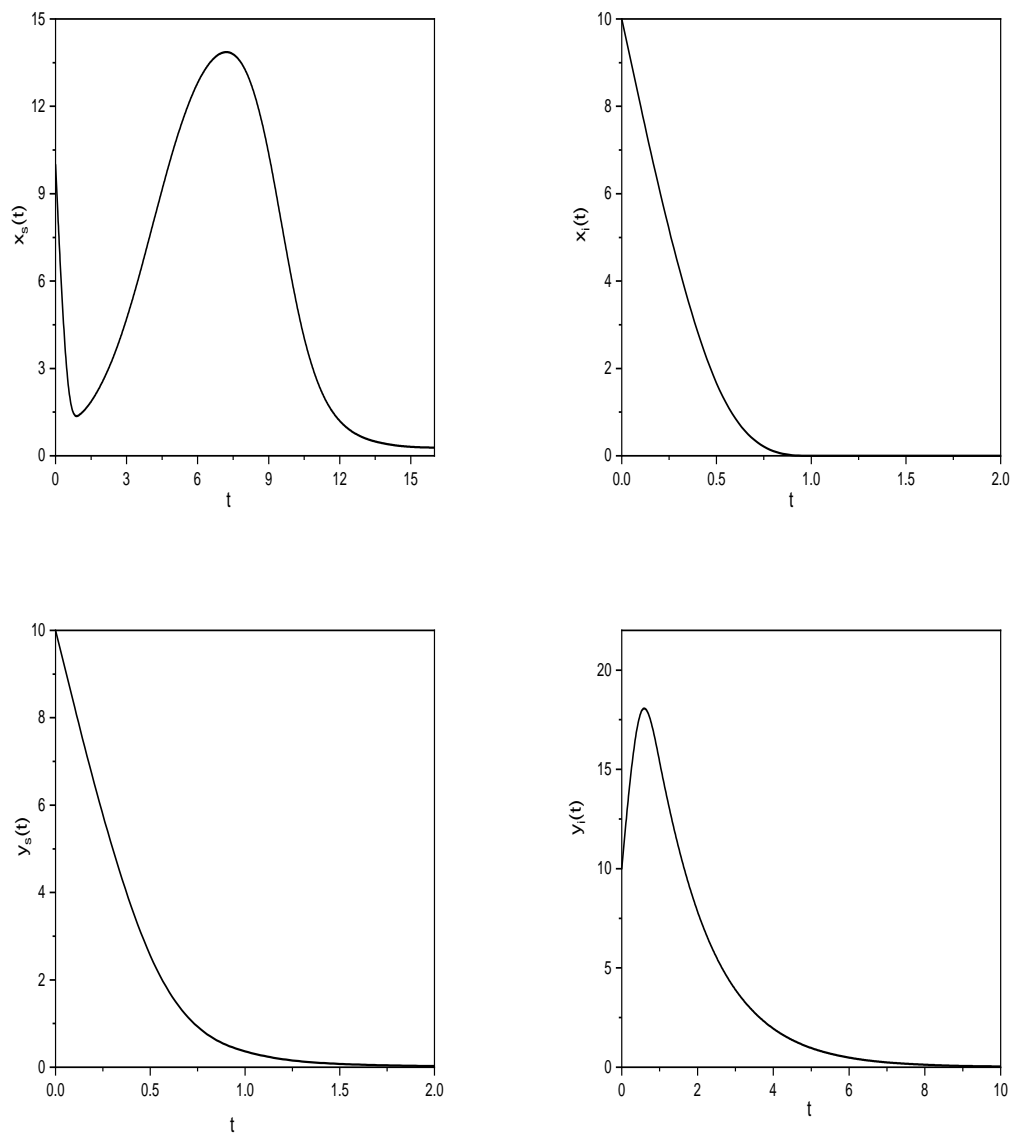


Figure 7.13: Plot of predator-prey system without impulsive control

Chapter 8

Population Dynamic Study of two prey one predator system with disease in first prey ¹

8.1 Introduction

Many scholars have focused significantly on epidemiological models. The incidence rate, a function that describes how a disease spreads from an infected person to a susceptible person, plays a crucial role in epidemiological models. Many scholars have carefully explored a number of epidemic models with such a wide range of incidence rates [105, 106, 107]. To make epidemic simulations more realistic, some researchers added with time delays [108].

Ecological literature has extensively investigated models of two and three-species populations having such functional responses. There are numerous studies on two-species systems, such as those involving predators and prey and on three-species systems, such as two prey and one predator [109, 110, 111] have long been important

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in theoretical ecology.

Researchers have developed a greater interest in the merging of ecology and epidemiology because the effects of infectious diseases on the ecological system regulate population size. There are numerous prey-predator models that include infectious diseases. In eco-epidemic model, the predator populations become infected by eating prey was investigated by Anderson and Robert [33]. The changing of the prey-predator system in relation to disease in the prey and the predator population have been hypothesized and studied by some researchers, including Hudson [64], Haque and Venturino [112], Amar and Joydev [40] etc.

In this work, we considered the Lotka-Volterra predator-prey model with two preys and one predator. We also take into account the fact that only the first prey, who are affected by an infectious disease, consists of two sub-classes: susceptible and infected. We examine the global and asymptotic stability to strengthen the reality of the model as explained in earlier chapter [82, 77].

8.2 Development of the model

These presumptions serve as the foundation for our mathematical model-

- Let x be the first prey's overall population density.
- The first group of prey is the only one to develop a transmissible disease.
- When a disease is present, the whole population of first prey are divided into two categories: (i) the susceptible prey population (x_s) and (ii) the infected prey population (x_i).
- The disease in the first prey population is disseminated horizontally from the susceptible to the infected in first prey population at a constant rate of infection β , in accordance with the rule of mass action.

- Diseased members of the first prey population do not recover. Disease does not spread from the ill first victim to the predator by feeding or any other means.
- There is an abundant supply of second prey in the population in the absence of a predator, and there is no need to search for the second prey population because it has no intra-specific interactions and is expanding exponentially.
- Let x_2 be the second prey's overall population density.
- y stands for the total population density of predators.

Based on the above considerations, we propose the following model using the system of non-linear differential equations.

$$\begin{aligned}
\frac{dx_s}{dt} &= r_1 x_s - e x_s^2 - (e + \beta) x_s x_i + h_1 x_s x_2 y - \frac{P_1 x_s y}{a + \lambda \alpha x_2 + x_s} \\
\frac{dx_i}{dt} &= \beta x_s x_i - m_1 x_i - \frac{P_2 x_i y}{a + \lambda \alpha x_2 + x_i} \\
\frac{dx_2}{dt} &= r_2 x_2 - P_3 x_2 y + h_2 x_i x_2 y \\
\frac{dy}{dt} &= \frac{C_1 x_s y}{a + \lambda \alpha x_2 + x_s} + \frac{C_2 x_i y}{a + \lambda \alpha x_2 + x_i} + C_3 x_2 y - m_2 y
\end{aligned} \tag{8.1}$$

where x_{s0} , x_{i0} , x_{20} , y_0 are the initial populations and all parameters $(r_1, r_2, e, C_1, C_2, a, P_1, P_2, m_1, m_2) \geq 0$ with $x_s = x_{s0} > 0$, $x_i = x_{i0} > 0$, $x_2 = x_{20} > 0$, $y = y_0 > 0$.

Here r_1 is the internal production growth rate of first prey, r_2 is the internal production growth rate of second prey, e is intra-species competition, P_1 is Predation level of susceptible first prey, P_2 is Predation level of diseased first prey, P_3 is Predation level of second prey, C_1 is rate at which susceptible prey become predators, C_2 is the rate at which diseased prey become predators, β is transmission rate on first prey vulnerable to diseased population, a is half-saturation constant, λ is ratio

of the predator's handling time to that of its first prey item and to its second prey item, α is ratio between the second prey's and the first prey's capture rates, m_1 is mortality rate of the infected first prey, m_2 is rate of the predator's death, h_1, h_2 are coefficients of help between the preys.

To analyze the system's stability, the matrix differential equation is written as

$$\dot{x} = Ax + \phi(x) \quad (8.2)$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}_s(t) \\ \dot{x}_i(t) \\ \dot{x}_2(t) \\ \dot{y}(t) \end{pmatrix}, A = \begin{bmatrix} r_1 & 0 & 0 & 0 \\ 0 & -m_1 & 0 & 0 \\ 0 & 0 & r_2 & 0 \\ 0 & 0 & 0 & -m_2 \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex_s^2 - (e + \beta)x_sx_i + h_1x_sx_2y - \frac{P_1x_sy}{a+\lambda\alpha x_2+x_s} \\ \beta x_sx_i - \frac{P_2x_iy}{a+\lambda\alpha x_2+x_i} \\ -P_3x_2y + h_2x_ix_2y \\ \frac{C_1x_sy}{a+\lambda\alpha x_2+x_s} + \frac{C_2x_iy}{a+\lambda\alpha x_2+x_i} + C_3x_2y; \end{bmatrix}$$

8.3 Takagi-Sugeno model

The non-linear equations can be transformed into the following linear equation as explained in earlier (chapter-2).

If $x(t)$ is M_i then

$$\dot{x}(t) = A_i x(t), t \neq \tau_j \quad (8.3)$$

$$\Delta(x) = k_{ij}x(t), t = \tau_j, i = 1, 2, 3 \dots r; j = 1, 2, \dots \quad (8.4)$$

$$\text{where, } A_i = \begin{bmatrix} r_1 - z_1 - z_2 - z_3 + h_1z_4 - P_1z_7 & 0 & 0 & 0 \\ z_3 & -P_2z_5 - m_1 & 0 & 0 \\ 0 & h_2z_4 & r_2 - z_6 & 0 \\ C_1z_7 & C_2z_5 & z_8 & -m_2 \end{bmatrix},$$

$i = 1$ to 255, where the matrices A'_i 's are generated using maximum and mini-

mum values of z'_k s; $k = 1$ to 8 , here z'_k s are related to the values of $x_s(t) \in [0, d_1]$, $x_i(t) \in [0, d_2]$, $x_2(t) \in [0, d_3]$, $y(t) \in [0, d_4]$ (here $z_1 = ex_i$, $z_2 = ex_s$, $z_3 = \beta x_i$, $z_4 = x_2 y$, $z_5 = \frac{y}{a + \lambda \alpha x_2 + x_i}$, $z_6 = P_3 y$, $z_7 = \frac{y}{a + \lambda \alpha x_2 + x_s}$, $z_8 = C_3 y$) $M_i, x(t), A_i \in R^{4 \times 4}$, r is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta(x)|_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

8.4 Numerical Simulation

Analytical investigations can never be finished without the results numerical validation. Computer simulations of the system's solutions are presented in this section (8.2). These numerical solutions are crucial from a practical standpoint in addition to serving as confirmation of our analytical conclusions. Because most biological systems are intricate, they ought to be modeled using a descriptive, fuzzy logical approach. In order to analyze predator-prey systems with functional responses and impulsive effects, the recommended impulsive Takagi-Sugeno design model is used. In this part, intra-species competition predator-prey model (8.2) is analyzed. Due to the complexity, non-linearity, and uncertainty of biological systems, they should be express using a fuzzy logical approach and language description.

The membership functions [96] were produced as follows using the fuzzy impulsive Takagi-Sugeno design model on the (8.2):

$$\begin{aligned} M_1 &= \frac{z_1}{ed_2}, M_2 = \frac{ed_2 - z_1}{ed_2}, N_1 = \frac{z_2}{ed_1}, N_2 = \frac{ed_1 - z_2}{ed_1}, K_1 = \frac{z_3}{\beta d_2}, K_2 = \frac{\beta d_2 - z_3}{\beta d_2}, \\ L_1 &= \frac{z_4}{d_3 d_4}, L_2 = \frac{d_3 d_4 - z_4}{d_3 d_4}, O_1 = \frac{z_5}{\frac{d_4}{a + \lambda \alpha y + d_2}}, O_2 = \frac{\frac{d_4}{a + \lambda \alpha y + d_2} - z_5}{\frac{d_4}{a + \lambda \alpha y + d_2}}, R_1 = \frac{z_6}{P_3 d_4}, \\ R_2 &= \frac{P_3 d_4 - z_6}{P_3 d_4}, S_1 = \frac{z_7}{\frac{d_4}{a + \lambda \alpha y + d_1}}, S_2 = \frac{\frac{d_4}{a + \lambda \alpha y + d_1} - z_7}{\frac{d_4}{a + \lambda \alpha y + d_1}}, T_1 = \frac{z_8}{C_3 d_4}, T_2 = \frac{C_3 d_4 - z_8}{C_3 d_4} \end{aligned}$$

and the matrices A'_i s are calculated using

$$A_i = \begin{bmatrix} r_1 - z_1 - z_2 - z_3 + h_1 z_4 - P_1 z_7 & 0 & 0 & 0 \\ z_3 & -P_2 z_5 - m_1 & 0 & 0 \\ 0 & h_2 z_4 & r_2 - z_6 & 0 \\ C_1 z_7 & C_2 z_5 & z_8 & -m_2 \end{bmatrix},$$

$i = 1$ to 255, where the matrices A'_i 's are generated using maximum and minimum values of z'_k 's; $k = 1$ to 8 and, the Defuzzification can be shown as:

$$\dot{x}(t) = \sum_{i=1}^r h_i(z(t))(A_i x(t)) \quad (8.5)$$

here h'_i 's are given as, $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i=1$ to 255, $j=1$ to 8.

This Fuzzy model exactly represents the non-linear system (8.2) in the region $[0,10] \times [0,10] \times [0,10] \times [0,10]$

$$\begin{aligned} \frac{dx_s}{dt} &= r_1 x_s - e x_s^2 - (e + \beta) x_s x_i + h_1 x_s x_2 y - \frac{P_1 x_s y}{a + \lambda \alpha x_2 + x_s} \\ \frac{dx_i}{dt} &= \beta x_s x_i - m_1 x_i - \frac{P_2 x_i y}{a + \lambda \alpha x_2 + x_i} \\ \frac{dx_2}{dt} &= r_2 x_2 - P_3 x_2 y + h_2 x_i x_2 y \\ \frac{dy}{dt} &= \frac{C_1 x_s y}{a + \lambda \alpha x_2 + x_s} + \frac{C_2 x_i y}{a + \lambda \alpha x_2 + x_i} + C_3 x_2 y - m_2 y \end{aligned} \quad (8.6)$$

8.5 Results and discussion

This section describes the global stability of the considered intra-species predator - prey competition model (8.2). We have studied the system (8.1) numerically using MATHEMATICA software to get better insight of the proposed model.

It is calculated by taking parameter values at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $P_1 = 0.7$, $C_1 = 0.5$, $P_2 = 0.06$, $C_2 = 0.43$, $C_3 = 0.36$, $d_4 = 10$, $h_1 = 0.4$, $h_2 = 0.1$,

$m_1 = 0.001$, $m_2 = 0.5$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $P_3 = 0.8$, $a = 1$, $\beta = 0.7$, $\lambda = 10$, $\alpha = 2$, in 8.3 to obtain the eigen-values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ described in ([83]). It is evident that $\max(\lambda_i) = \lambda(\alpha) = 83$ then, we've decided diagonal matrix $\text{diag}[-0.99, -0.99]$ as impulsive control matrix in such a way that $\omega = \|I + K\| = 0.01$. The 8.3 system's existence is noted that it is globally-stable at $\epsilon=1.5$, $\delta_j=0.02$ (at the aforementioned values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -2.539 < 0$). Let's assume that the system's parameters are $r_1 = 1.5$, $r_2 = 0.7$, $e = 0.5$, $P_1 = 0.2$, $P_2 = 13$, $P_3 = 0.1$, $C_1 = 1.5$, $C_2 = 1.5$, $C_3 = 0.5$, $h_1 = 1.5$, $h_2 = 0.3$, $m_1 = 0.4$, $m_2 = 0.2$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $a = 1$, $\beta = 0.7$, $\lambda = 10$, $\alpha = 2$, since $\max(\lambda_i) = \lambda(\alpha) = 303$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 1.861 > 0$ for $\omega = 0.01$, $\epsilon=1.5$, $\delta_j=0.02$. The prey-predator model is seen to be unstable for the aforementioned parameter values.

Table. 8.1 shows the stability of the system at different rates in this study.

r_1	r_2	e	P_1	P_2	P_3	C_1	C_2	C_3	h_1	h_2	m_1	m_2	d_1	d_2	d_3	d_4	a	β	λ	α	$\max(\lambda_i)$ $=\lambda(\alpha)$	$\ln(\epsilon\omega)$ $+\lambda(\alpha)\delta_j$	conclusion
1.5	0.07	0.2	0.7	0.6	0.8	0.5	0.4	0.36	0.4	0.1	0.001	0.5	10	10	10	10	1	0.7	10	2	83	-2.539	stable
1.0	0.07	0.2	0.7	0.6	0.8	1.0	0.5	1.3	0.4	0.1	0.001	0.5	10	10	10	10	1	0.7	10	2	82	-2.559	stable
1.2	0.7	0.1	0.2	0.3	0.1	1.5	0.5	1.0	0.5	0.01	0.01	0.5	10	10	10	10	1	0.7	10	2	102.4	-2.159	stable
1.5	0.7	0.5	0.2	1.3	0.1	1.5	1.5	0.5	1.5	0.3	0.4	0.2	10	10	10	10	1	0.7	10	2	303	1.861	unstable

Table 8.1: Stability of the system at various parameters

The effects of various parameters of the system 8.1 with Takagi-Sugeno fuzzy impulsive control model is presented in figs. 8.1 - 8.12 by fixing few parameters $P_1 = 0.7$, $P_2 = 0.06$, $P_3 = 0.8$, $C_1 = 0.5$, $C_2 = 0.43$, $C_3 = 0.36$, $h_2 = 0.1$, $a = 1$, $\lambda = 10$.

The consequence of transmission coefficient (β) on prey-predator interaction is given in figure. 8.1 at $r_1 = 1.5$, $r_2 = 0.07$, $d_1 = 10$, $e = 0.2$, $h_1 = 0.4$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $m_1 = 0.001$, $m_2 = 0.5$. This graph illustrates how an increase in β increases the population of susceptible and diseased first prey but decreases population of second prey and predator as more preys will become diseased.

Figure 8.2 depicts the impact that changing the maximum time for susceptible

first prey (d_1) on the prey-predator population at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $h_1 = 0.4$, $m_1 = 0.001$, $m_2 = 0.5$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $a = 1$, $\beta = 0.7$, $\alpha = 2$. This figure clearly shows that, as d_1 increases the population of first prey increases but second prey and predator population decreases as prey will get more time to live.

Figure 8.3 illustrates that the dynamic shift in the prey-predator population caused by adjusting the infected first prey max time (d_2) in the prey-predator system at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $h_1 = 0.4$, $m_1 = 0.001$, $d_1 = 10$, $m_2 = 0.5$, $d_3 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. This graph shows that when d_2 rises, first prey (susceptible and diseased) population rises while second prey and predator population decreases because more preys and predators will become diseased.

The change on prey - predator population (x_s, x_i, x_2, y) by varying second prey max time (d_3) is shown in fig. 8.4 at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $h_1 = 0.4$, $m_1 = 0.001$, $d_1 = 10$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. This figure clearly shows how decrease in second prey max time increases first (diseased and susceptible both) prey population but second prey and predator population decreases.

The impact of max time of predator (d_4) on prey-predator system is expressed in fig. 8.5 at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $h_1 = 0.4$, $m_1 = 0.001$, $d_1 = 10$, $m_2 = 0.5$, $d_2 = 10$, $d_3 = 10$, $\beta = 0.7$, $\alpha = 2$. It is noticed from this figure that, decrease in predator max time increases first (diseased and susceptible both) prey population but second prey and predator population decreases.

The dynamical change on prey - predator interaction by changing ratio between capture rate of first and second prey (α) can be seen in fig. 8.6 at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $d_1 = 10$, $h_1 = 0.4$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $m_1 = 0.001$, $m_2 = 0.5$, $\beta = 0.7$. This graph shows that the number of diseased first prey increases as α increases.

The changes with mortality rate of diseased first prey (m_1) parameter of prey-predator interaction under fuzzy impulsive control exhibited in fig. 8.7 at $r_1 = 1.5$,

$r_2 = 0.07$, $e = 0.2$, $h_1 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $m_2 = 0.5$, $\beta = 0.7$, $\alpha = 2$. This graph unequivocally demonstrates how a rise in m_1 reduces the number of infected first prey since more diseased prey will pass away.

The performance of prey - predator interaction by varying death rate of predator (m_2) parameter exhibited in fig. 8.8 at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $d_1 = 10$, $h_1 = 0.4$, $D_1 = 0.001$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. This image demonstrates how the population of diseased first prey, second prey, and predator reduces as m_2 grows.

The intra-species competition (e) consequence on prey-predator system is shown in fig. 8.9 at $r_1 = 1.5$, $r_2 = 0.07$, $h_1 = 0.4$, $d_1 = 10$, $m_1 = 0.001$, $d_3 = 10$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. It is clear from this figure that as the intra-species competition raises, the population of first prey increases whereas second prey and predators diminishes.

The influence of susceptible prey's intrinsic growth rate (r_1) on prey-predator system is presented in fig. 8.10 at $r_2 = 0.07$, $d_1 = 10$, $e = 0.2$, $h_1 = 0.4$, $m_1 = 0.001$, $d_3 = 10$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. This graph demonstrates unambiguously how a decrease in intrinsic growth rate of susceptible prey causes a rise in the population of diseased first prey whereas there is a drop in second prey and predator population.

The impact of a second prey's intrinsic growth rate (r_2) on prey-predator system can be seen in fig. 8.11 at $r_1 = 1.5$, $d_3 = 10$, $e = 0.2$, $h_1 = 0.4$, $d_1 = 10$, $m_1 = 0.001$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. It is evident from this figure that an increase in the second prey's intrinsic growth rate decreases its population.

The change with coefficient of help between preys (h_1) on prey-predator system is presented in fig. 8.12 at $r_1 = 1.5$, $r_2 = 0.07$, $d_1 = 10$, $e = 0.2$, $m_1 = 0.001$, $d_3 = 10$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $\beta = 0.7$, $\alpha = 2$. It is noticed from this figure that, the first prey population (diseased and susceptible) increases as h_1 decreases

and second prey and predator population increases as h_1 increases.

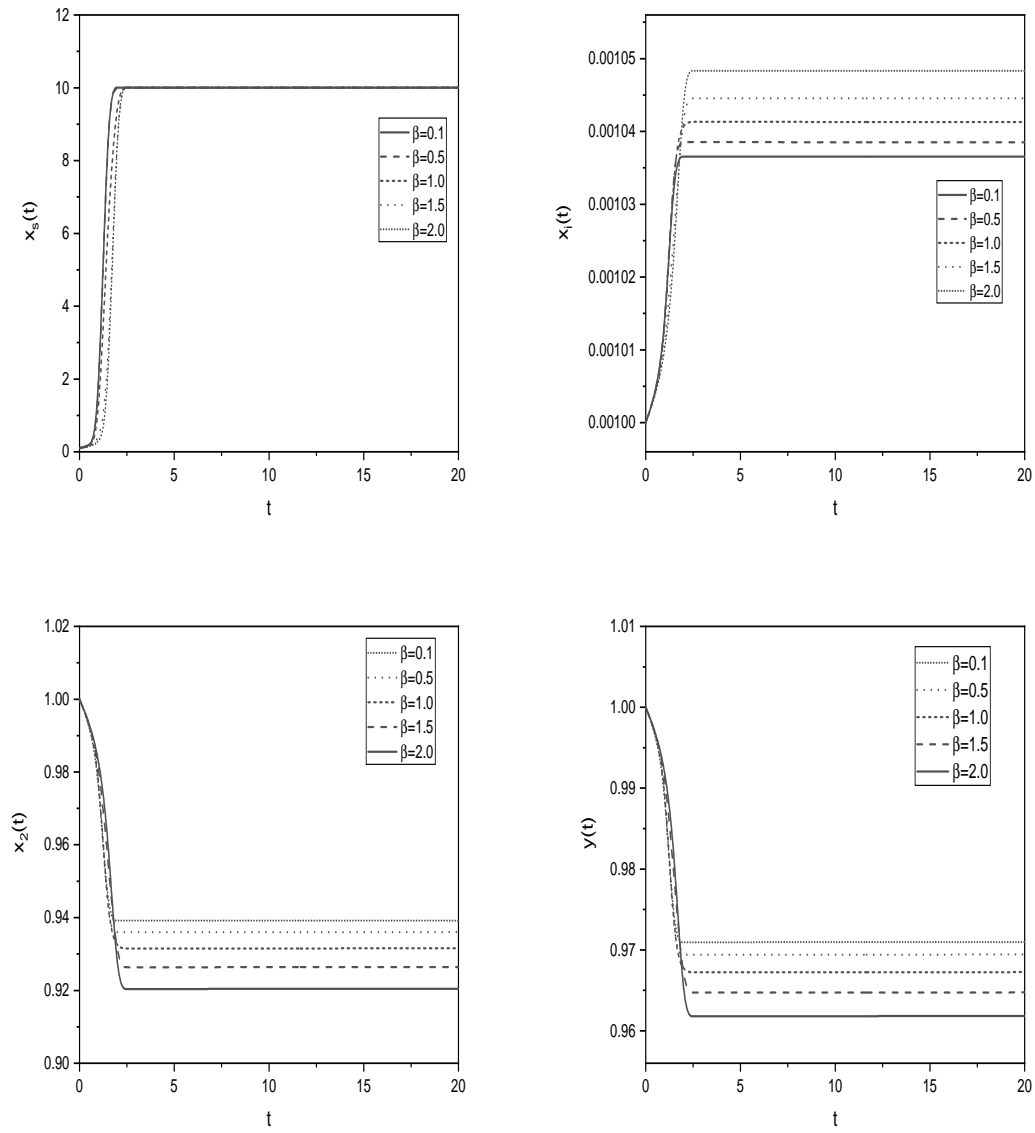
The physiology of the populations of four-species (x_s, x_i, x_2, y) without impulse control is finally shown in figure. 8.13 by maintaining each of the Takagi-Sugeno fuzzy model's output parameters at $r_1 = 1.5$, $r_2 = 0.07$, $e = 0.2$, $P_1 = 0.7$, $P_2 = 0.06$, $P_3 = 0.8$, $C_1 = 0.5$, $C_2 = 0.43$, $C_3 = 0.36$, $h_1 = 0.4$, $h_2 = 0.1$, $d_1 = 10$, $m_1 = 0.001$, $m_2 = 0.5$, $d_2 = 10$, $d_4 = 10$, $a = 1$, $d_3 = 10$, $\beta = 0.7$, $\lambda = 10$, $\alpha = 2$, with $x_s(0) > 0$, $x_i(0) > 0$, $x_2(0) > 0$, $y(0) > 0$, $t = 10$. The graph makes it obvious how predator and prey populations are stabilizes.

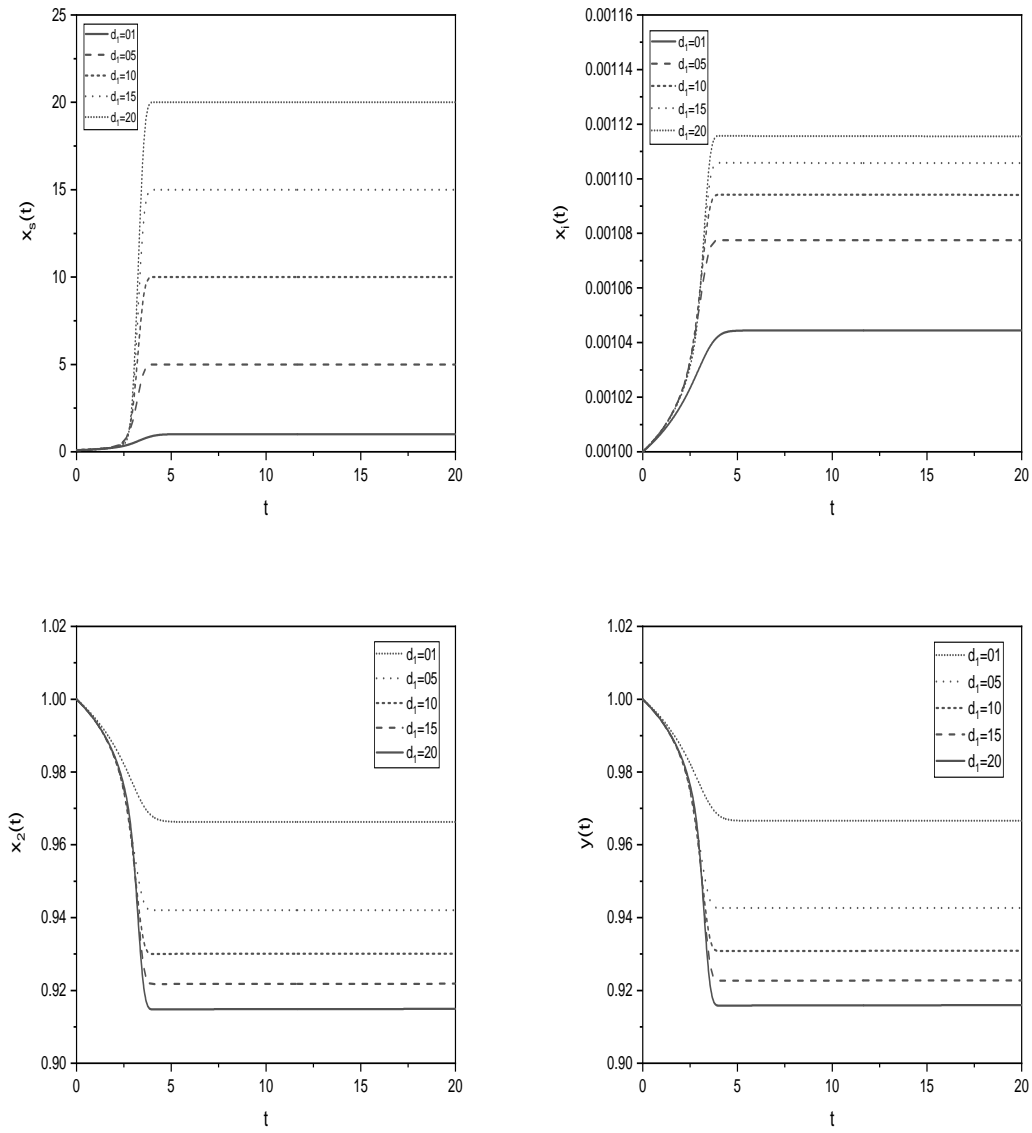
8.6 Conclusions

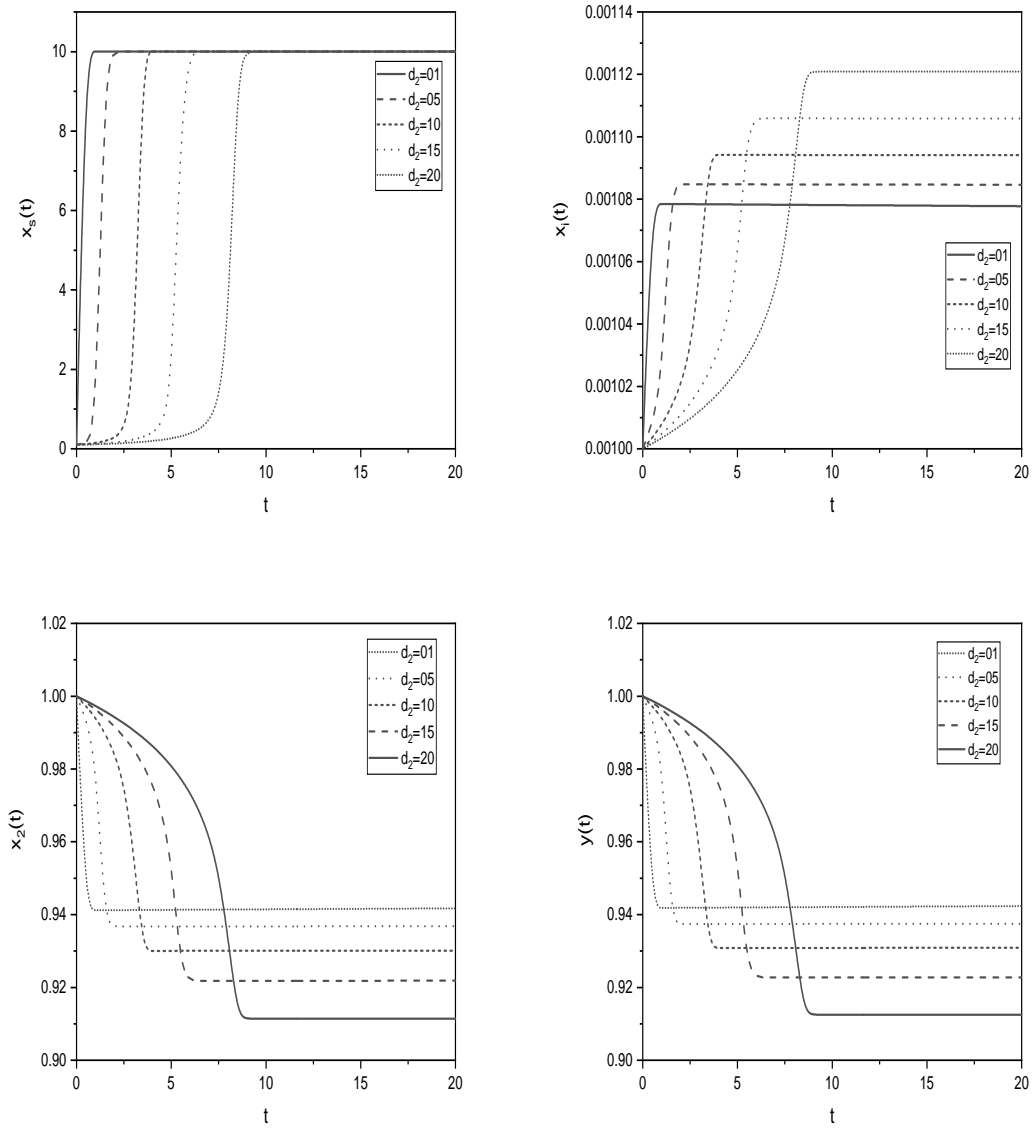
In this study, we created a two prey, one predator eco-epidemiological model in which an infectious disease exclusively affects the first prey group. The first prey population has been split into two sub-classes: susceptible and diseased. The study is further extend to the concept to four dimensional system of the Lotka-volterra predator-prey model where fuzzy impulsive control technique was employed to evaluate the stability of the relationship connecting the species. In addition, the stability analysis of the system was also observed by a numerical example of predator-prey system with impulsive effects. The main results of this study are as follows:

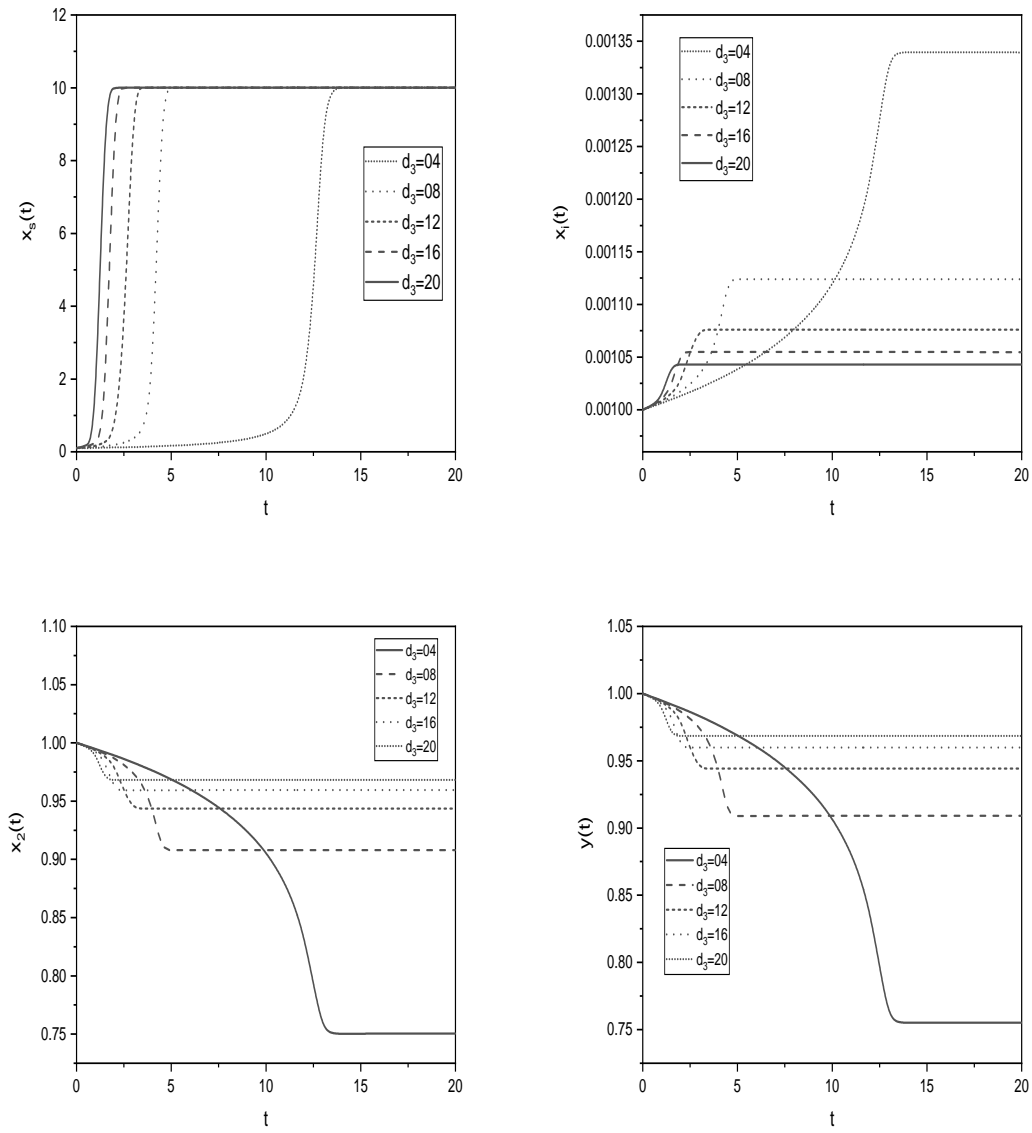
- The number of diseased prey grows with increased infection transmission.
- The number of infected prey increases when the capture rate ratio between first and second prey rises.
- Intra-species competition effects all the four populations. As population density rises, the effect of intra-species competition is a reduction in population growth rates.

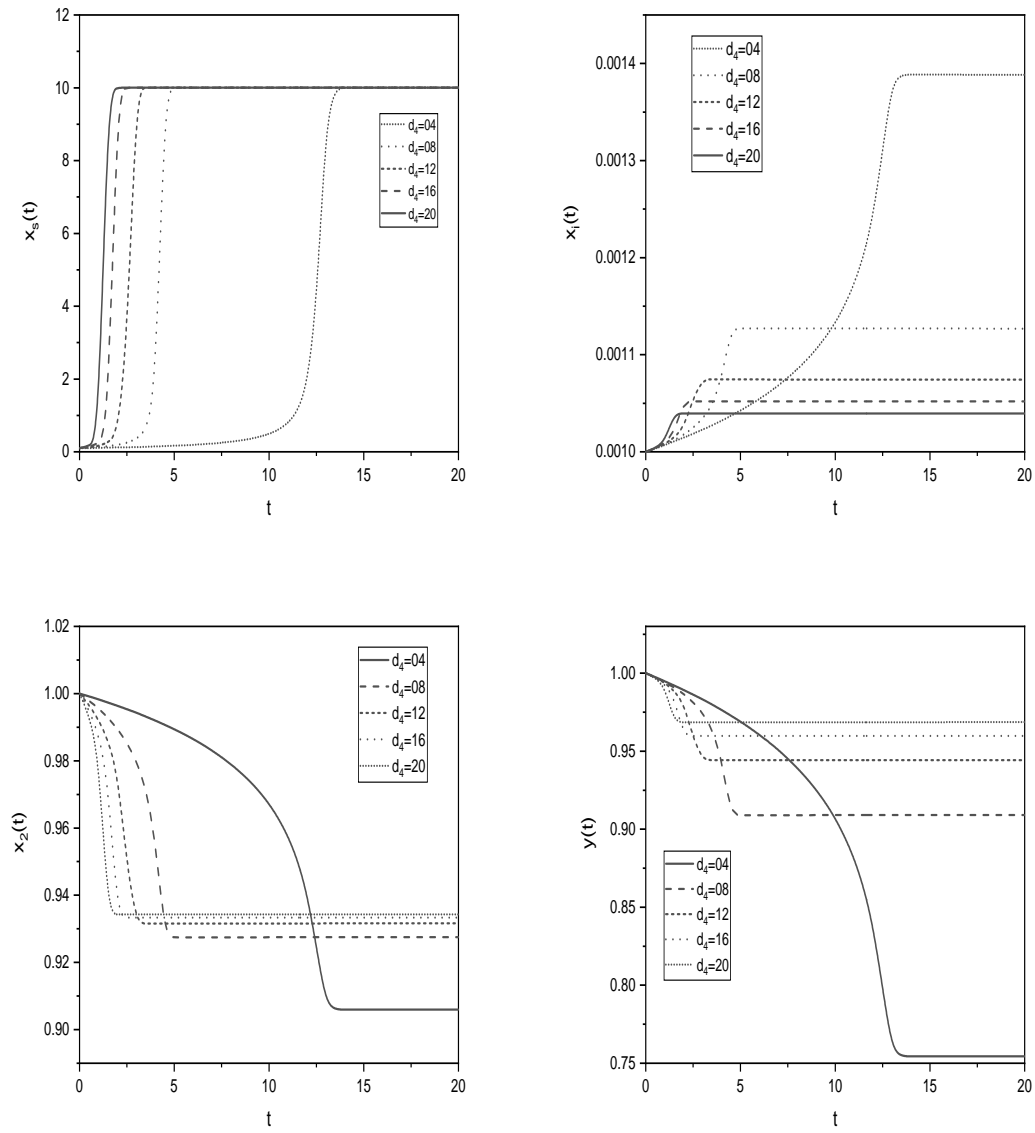
-
- The population of diseased first prey is reduced by an increase in their death rate.

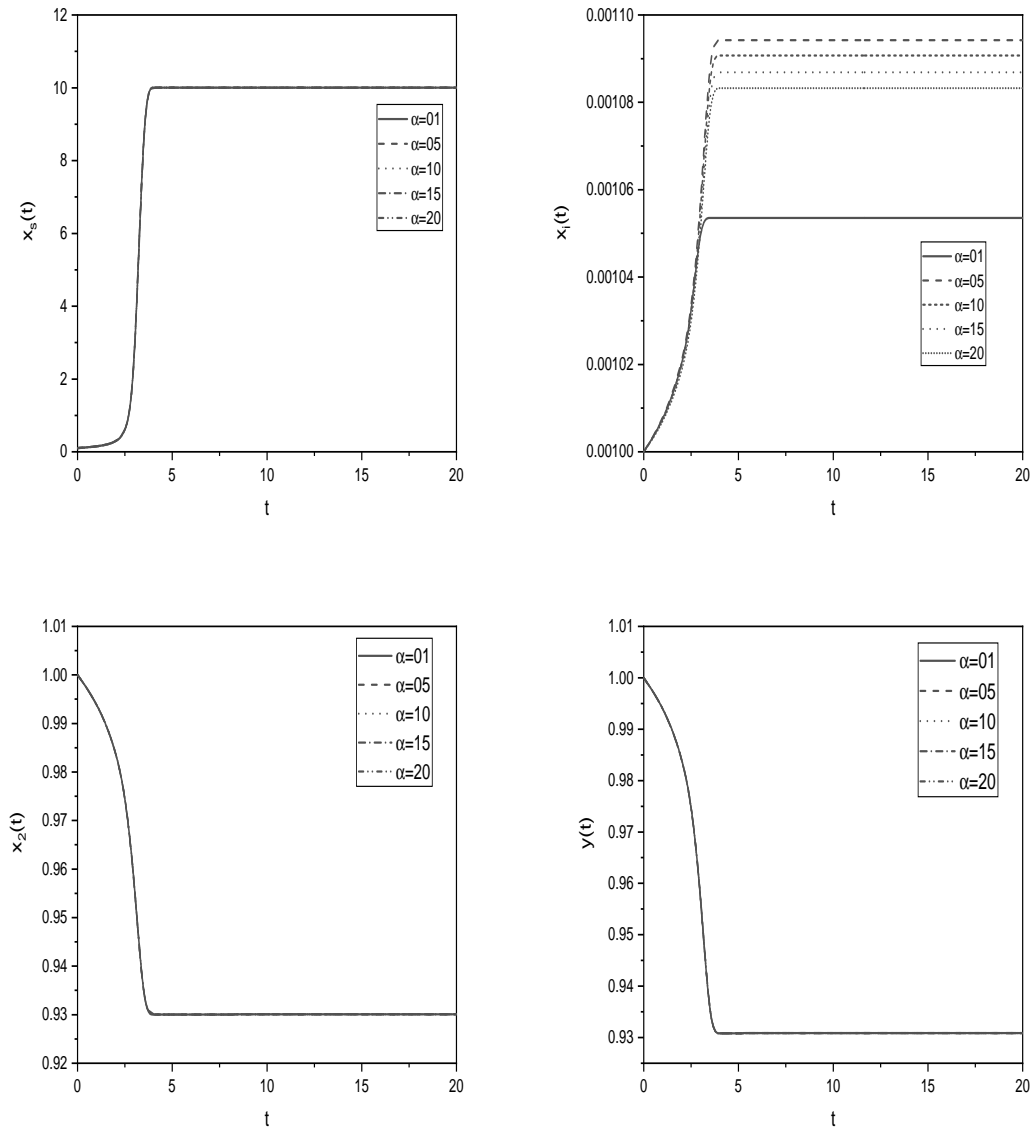
Figure 8.1: Impact of (β) on prey-predator system

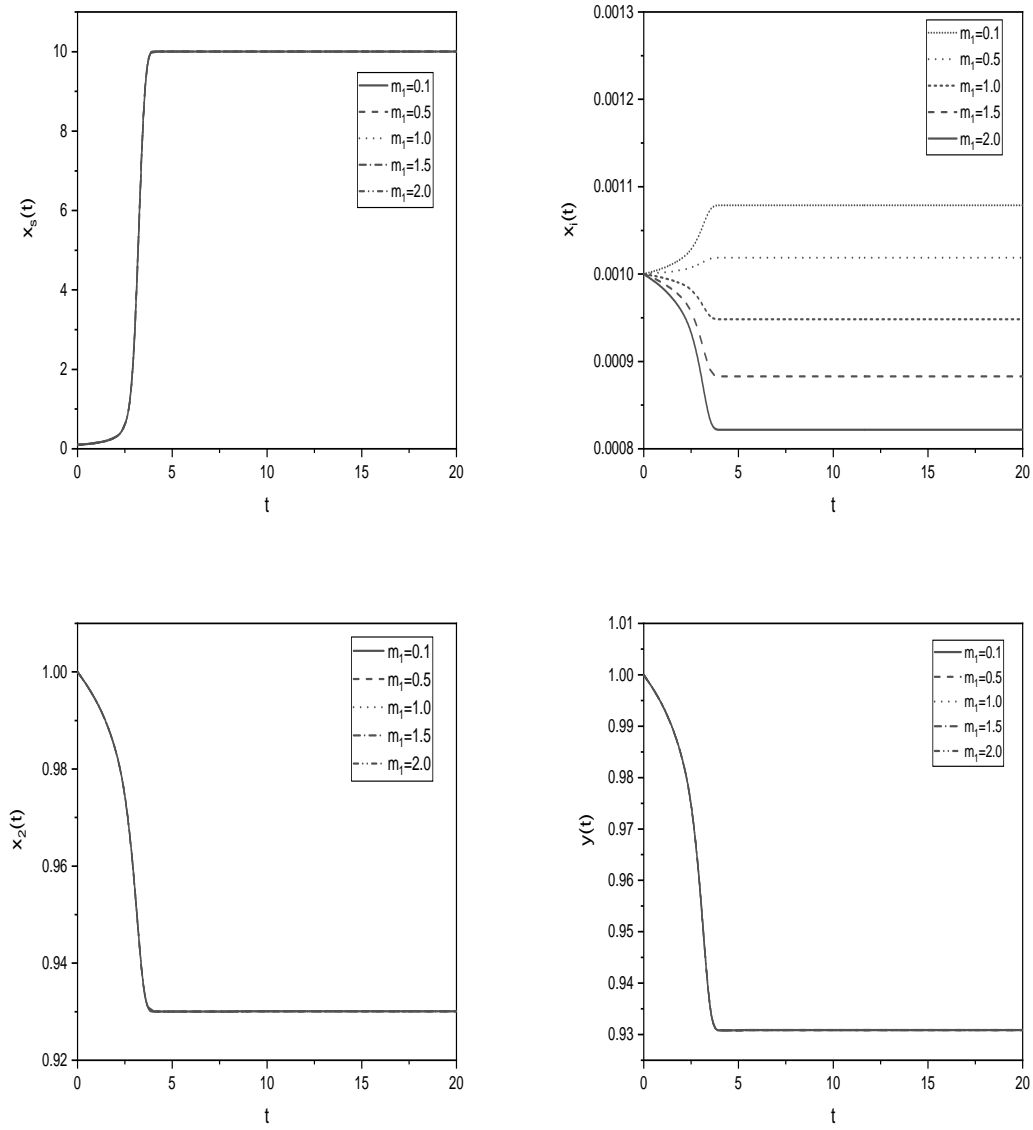
Figure 8.2: Impact of (d_1) on prey-predator system

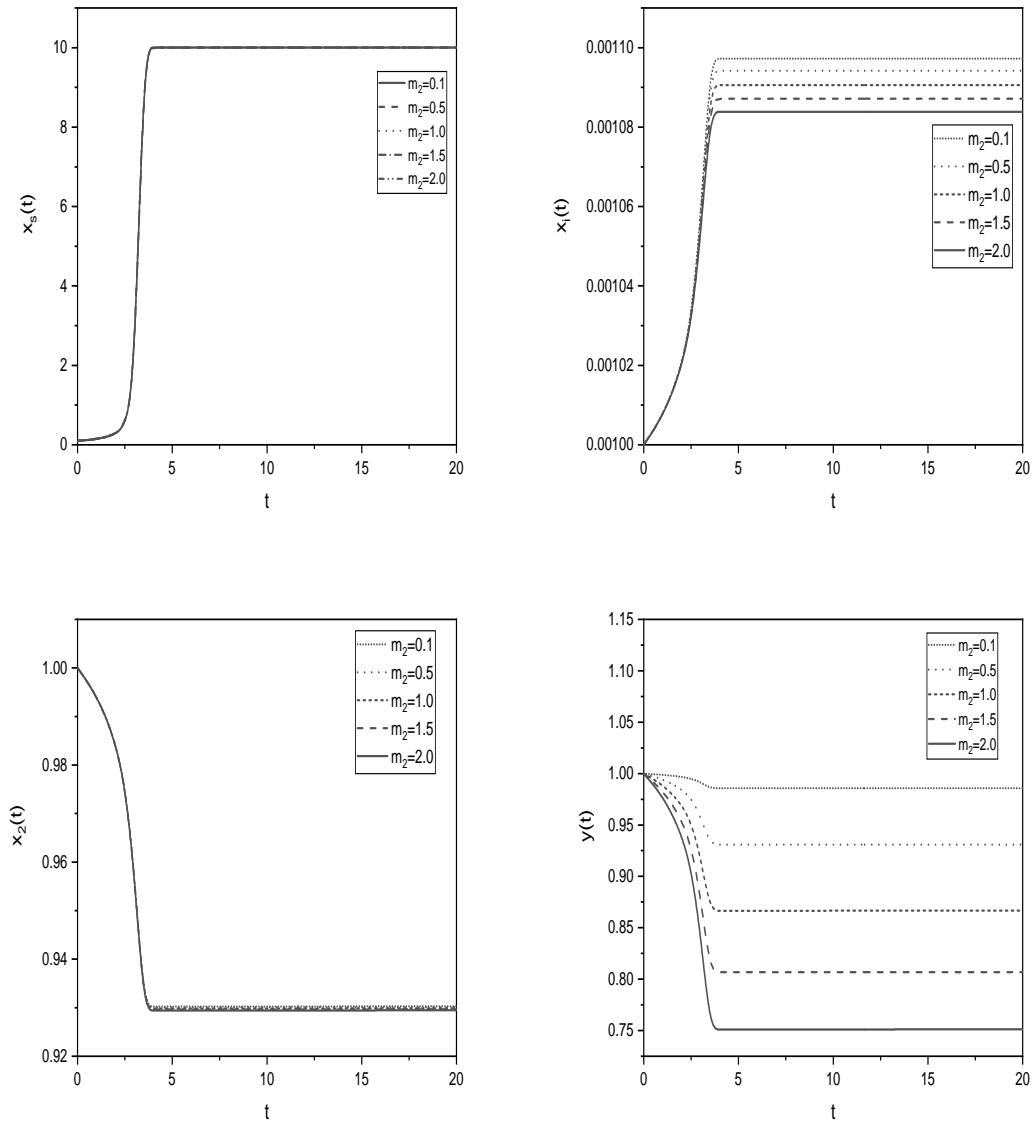
Figure 8.3: Impact of (d_2) on prey-predator system

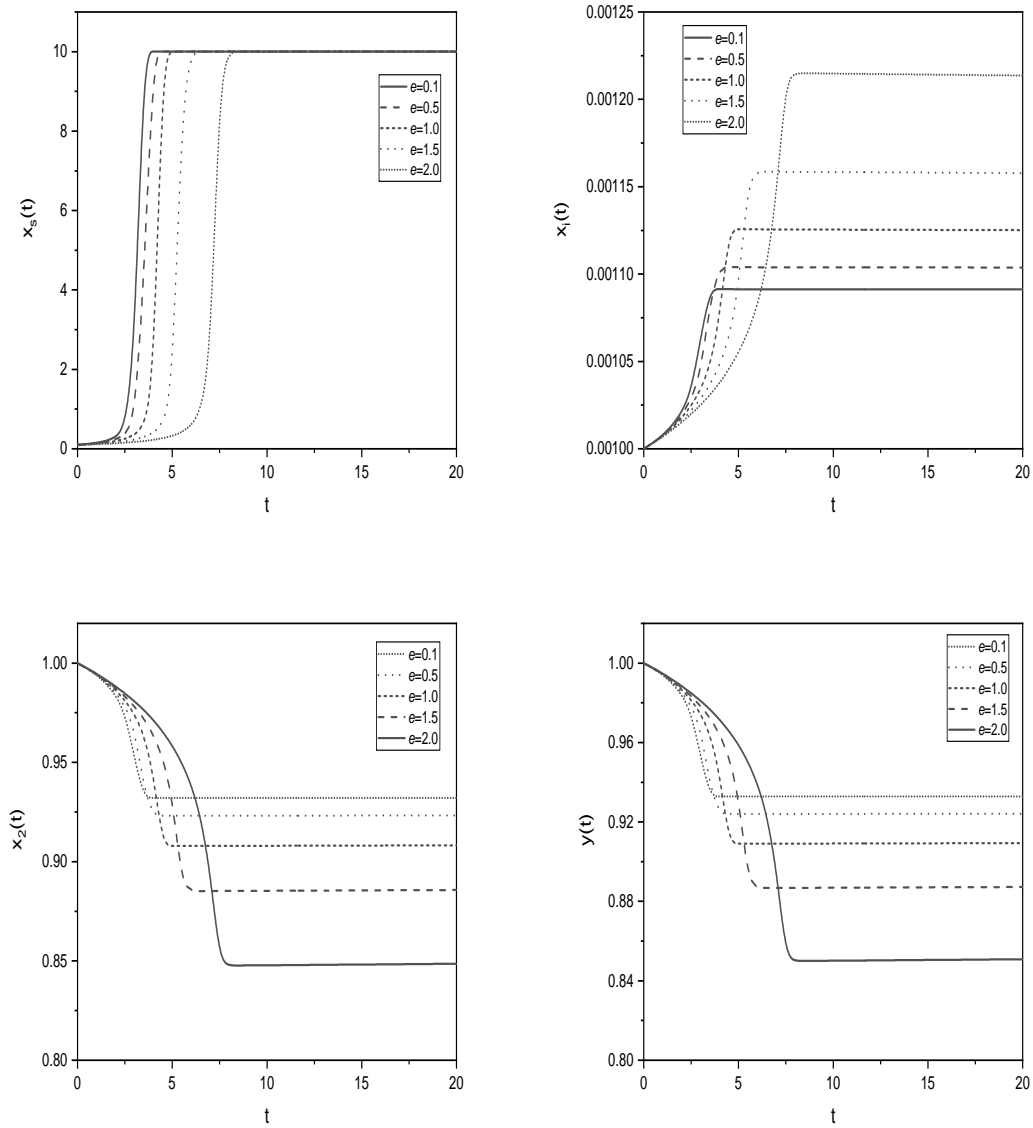
Figure 8.4: Impact of (d_3) on prey-predator system

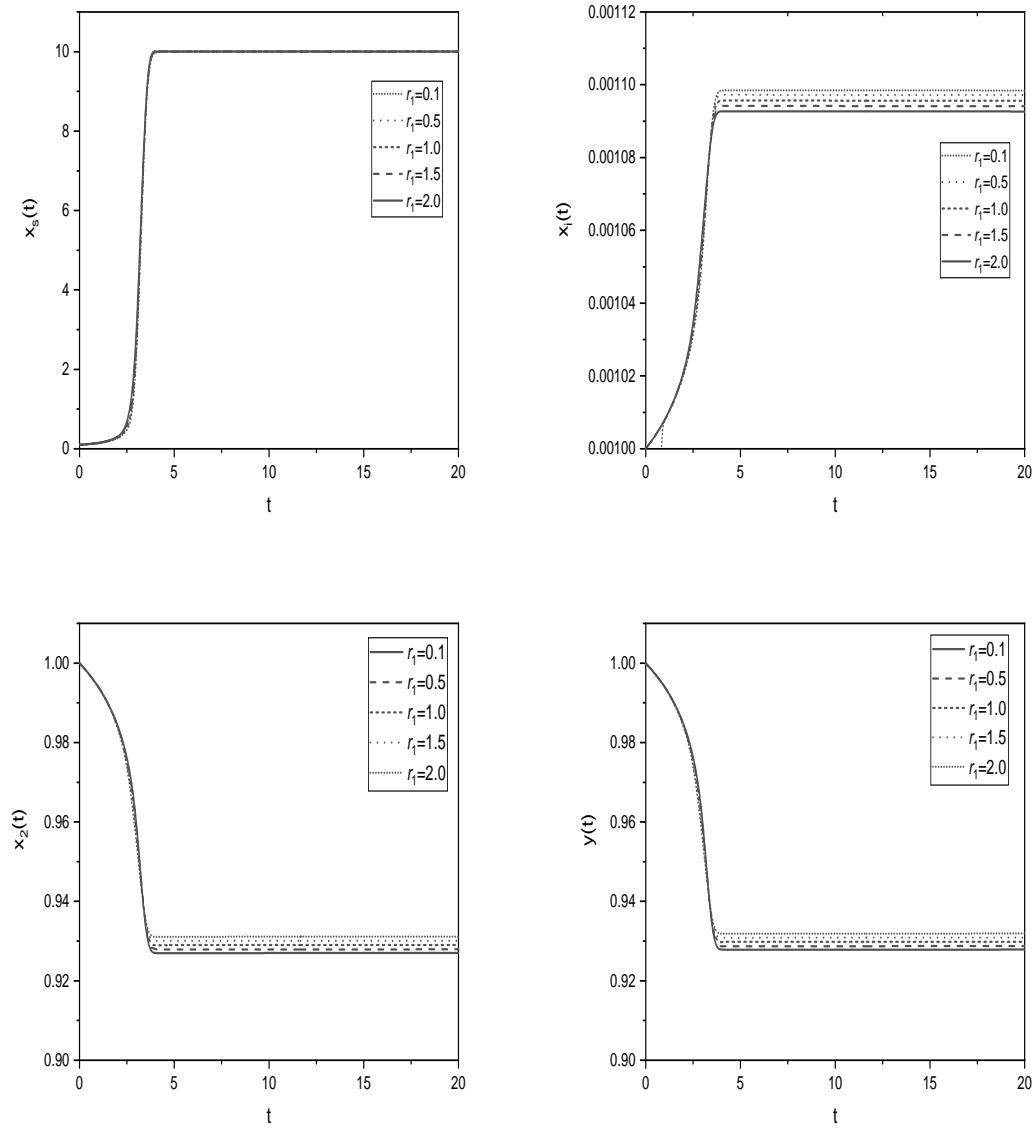
Figure 8.5: Impact of (d_4) on prey-predator system

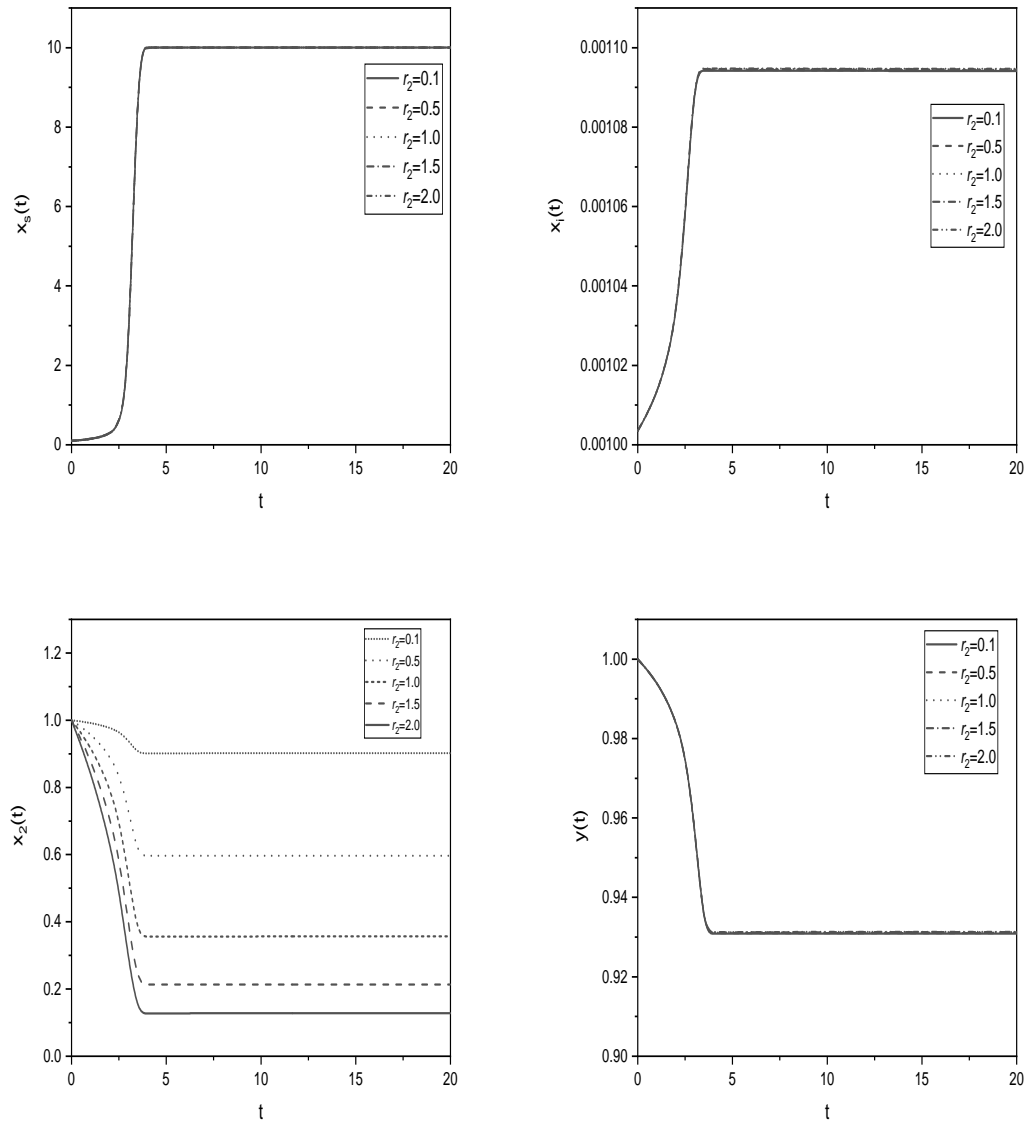
Figure 8.6: Impact of (α) on prey-predator system

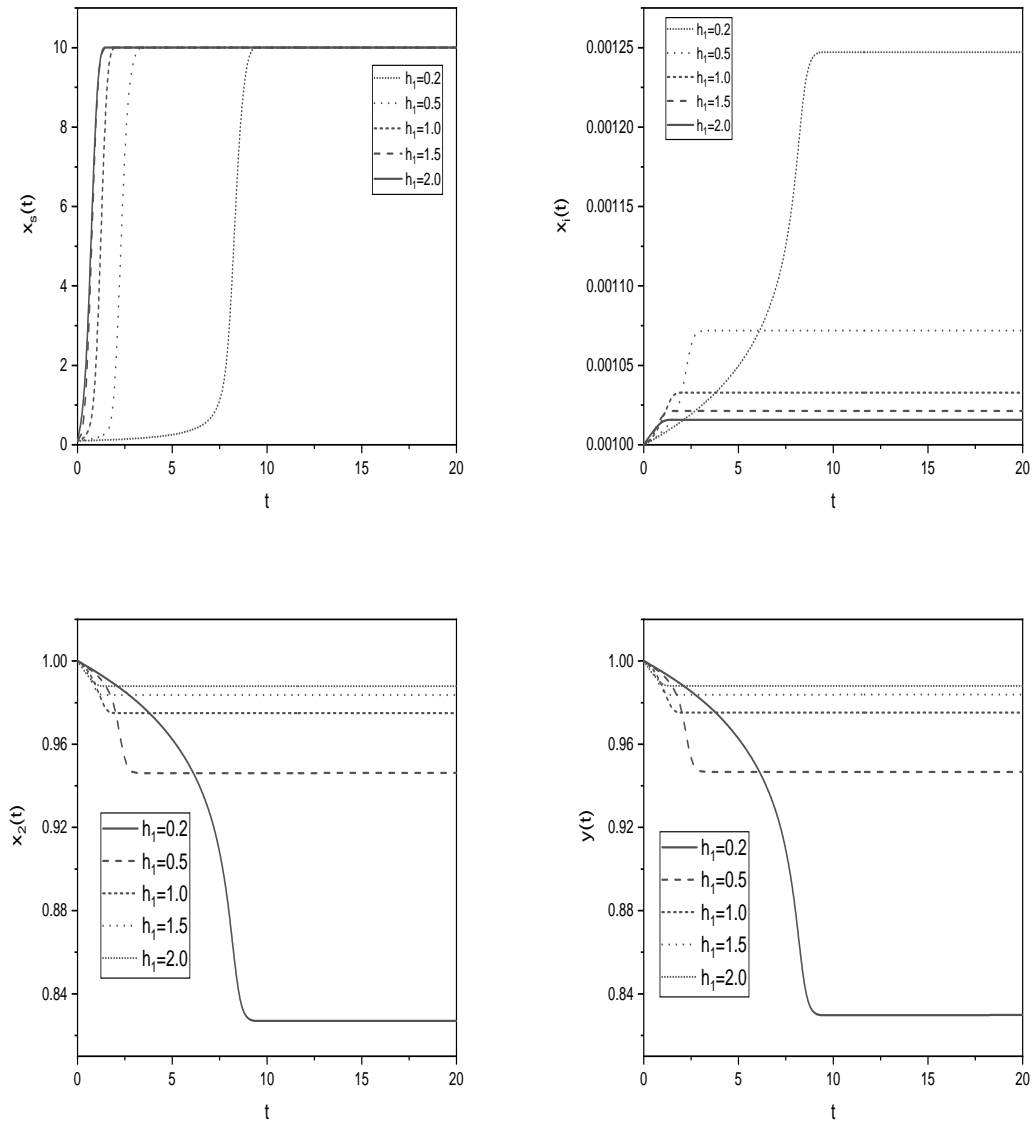
Figure 8.7: Impact of (m_1) on prey-predator system

Figure 8.8: Impact of (m_2) on prey-predator system

Figure 8.9: Impact of (e) on prey-predator system

Figure 8.10: Impact of (r_1) on prey-predator system

Figure 8.11: Impact of (r_2) on prey-predator system

Figure 8.12: Impact of (h_1) on prey-predator system

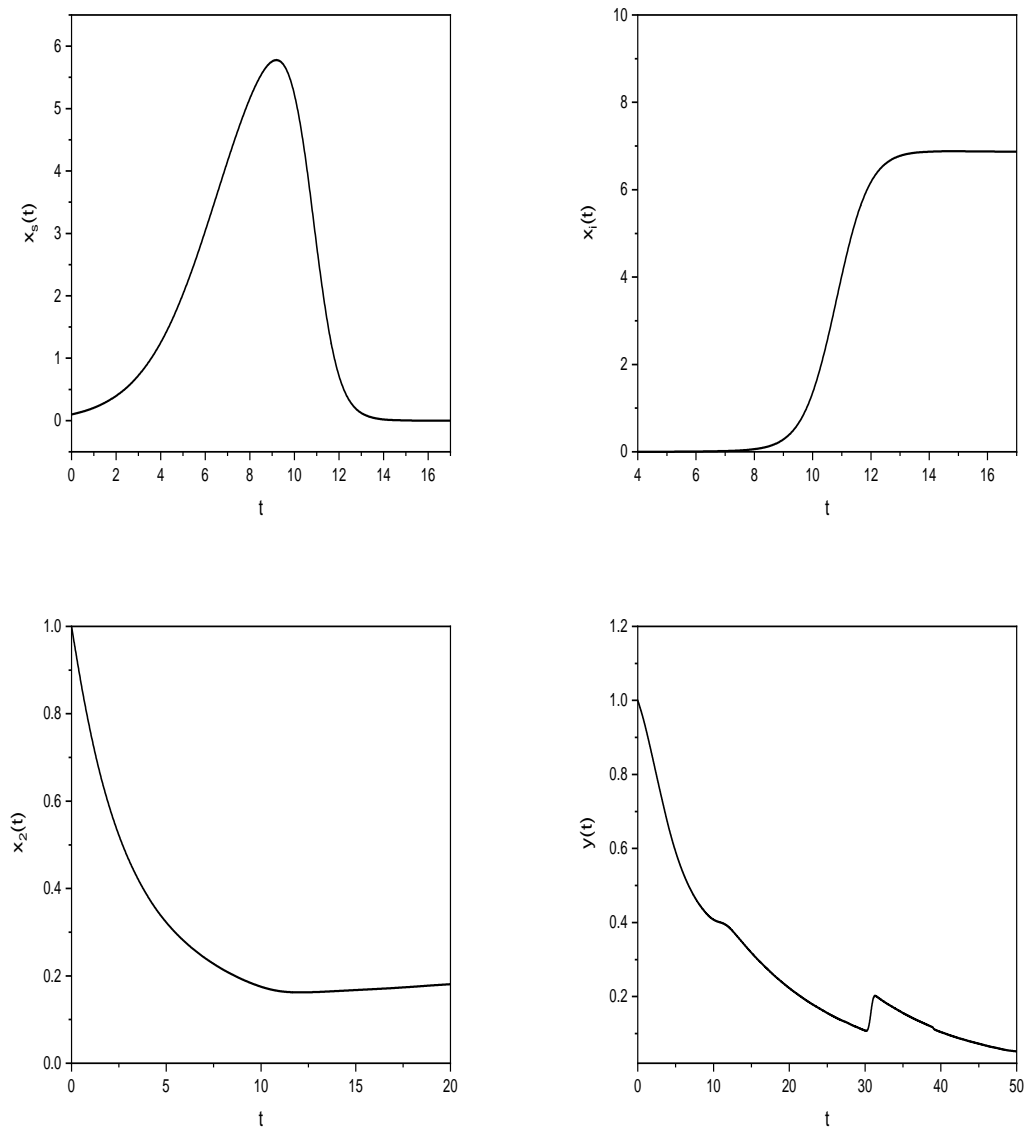


Figure 8.13: Different scenarios for predator-prey system without impulse control

Chapter 9

A Mathematical Study for the Stability of Two Predator and One Prey with Infection in First Predator

1

9.1 Introduction

In this chapter, we have considered Lotka-Volterra predator-prey model with one prey and two predators. Also, considered that the only first predator population got infected by an infectious disease, i.e., the first predator population is divided into two sub-classes: susceptible and infected. The global and asymptotic stability of this model was studied as explained in the earlier chapters [77]. Finally, presented the graphical solutions for the considered problem.

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9.2 Model Formation

Our mathematical model is based on the following assumptions

- Let x be the total population density of the prey.
- The initial group of predators is the only one to have an infectious disease.
- The overall population of first predators is divided into two sub classes when a disease is present: (i) the susceptible first predator population (y_s) and (ii) the infected first predator population (y_i).
- According to the rule of mass action, the disease in the first predator population is spread horizontally from the susceptible to the infected first predator population at a constant rate of infection β .
- Let the second predator total population density is denoted by y_2 .
- Let t be the number of years.

The following model is proposed utilizing a set of non-linear ordinary differential equations based on the aforementioned presumptions.

$$\begin{aligned}
 \frac{dx}{dt} &= rx - ex^2 - \frac{P_1 y_s x}{a_0 + x} - \frac{P_2 y_i x}{a_0 + x} - \frac{P_3 y_2 x}{a_0 + x} \\
 \frac{dy_s}{dt} &= \frac{C_1 P_1 y_s x}{a_0 + x} - \frac{C_1 P_1 y_s y_2}{a_1 + x} - \beta y_s y_i - m_1 y_s \\
 \frac{dy_i}{dt} &= \beta y_s y_i + \frac{C_2 P_2 y_i x}{a_0 + x} - \frac{C_2 P_2 y_i y_2}{a_2 + x} - m_2 y_i \\
 \frac{dy_2}{dt} &= \frac{C_3 P_3 x y_2}{a_0 + x} - \frac{C_3 P_3 y_s y_2}{a_1 + x} + \frac{C_3 P_3 y_i y_2}{a_2 + x} - m_3 y_2
 \end{aligned} \tag{9.1}$$

where $x_0, y_{s0}, y_{i0}, y_{20}$ are the initial populations and all of the parameters are positive with initial conditions as $x = x_0 > 0, y_s = y_{s0} > 0, y_i = y_{i0} > 0, y_2 = y_{20} > 0$. Here r is the intrinsic growth rate of prey, e is the intra-specific competition, β is the infection transmission, P_1 is the predation rate of susceptible first predator, P_2

is the predation rate of infected first predator, P_3 is the predation rate of second predator, C_1 is the efficiency of first susceptible predator, C_2 is the conversion efficiency of first infected predator, C_3 is the conversion efficiency of second predator, m_1 the mortality rate of first susceptible predator, m_2 the mortality rate of first infected predator, m_3 is the mortality rate of second predator, a_0, a_1, a_2 are the half-saturation constants.

A matrix differential equation is stated as follows to analyze the system's stability:

$$\dot{x} = Ax + \phi(x) \quad (9.2)$$

where

$$\dot{x} = \begin{pmatrix} \dot{x}(t) \\ \dot{y}_s(t) \\ \dot{y}_i(t) \\ \dot{y}_2(t) \end{pmatrix}, A = \begin{bmatrix} r & 0 & 0 & 0 \\ 0 & -m_1 & 0 & 0 \\ 0 & 0 & -m_2 & 0 \\ 0 & 0 & 0 & -m_3 \end{bmatrix}, \phi(x) = \begin{bmatrix} -ex^2 - \frac{P_1 y_s x}{a_0 + x} - \frac{P_2 y_i x}{a_0 + x} - \frac{P_3 y_2 x}{a_0 + x} \\ \frac{C_1 P_1 y_s x}{a_0 + x} - \frac{C_1 P_1 y_s y_2}{a_1 + x} - \beta y_s y_i \\ \beta y_s y_i + \frac{C_2 P_2 y_i x}{a_0 + x} - \frac{C_2 P_2 y_i y_2}{a_2 + x} \\ \frac{C_3 P_3 y_2 x}{a_0 + x} - \frac{C_3 P_3 y_s y_2}{a_1 + x} + \frac{C_3 P_3 y_i y_2}{a_2 + x} \end{bmatrix}$$

9.3 T-S Fuzzy model with Impulsive effects

The non-linear equations can be transformed into the following linear equation as explained in earlier chapter(s).

If $x(t)$ is M_i then

$$\dot{x}(t) = A_i x(t), t \neq \tau_j$$

$$\Delta(x) = K_{ij} x(t), t = \tau_j \quad (9.3)$$

$$i = 1, 2, 3 \dots r; j = 1, 2, \dots$$

$$\text{where, } A_i = \begin{bmatrix} r - ex - \frac{P_1 y_s}{a_0+x} - \frac{P_2 y_i}{a_0+x} - \frac{P_3 y_2}{a_0+x} & 0 & 0 & 0 \\ \frac{C_1 P_1 y_s}{a_0+x} & -\frac{C_1 P_1 y_2}{a_1+x} - \beta y_i - m_1 & 0 & 0 \\ \frac{C_2 P_2 y_i}{a_0+x} & \beta y_i & -\frac{C_2 P_2 y_2}{a_2+x} - m_2 & 0 \\ \frac{C_3 P_3 y_2}{a_0+x} & -\frac{C_3 P_3 y_2}{a_1+x} & \frac{C_3 P_3 y_2}{a_2+x} & -m_3 \end{bmatrix},$$

$i = 1$ to 511, where the matrices A'_i s are generated using maximum and minimum values of z'_k s; $k = 1$ to 9 and $z_1, z_2, z_3, z_4, z_5, z_6, z_7, z_8, z_9$ are related to the values of $x(t) \in [0, d_1], y_s(t) \in [0, d_2], y_i(t) \in [0, d_3], y_2(t) \in [0, d_4]$, here $z_1 = ex, z_2 = \frac{P_1 y_s}{a_0+x}, z_3 = \frac{P_2 y_i}{a_0+x}, z_4 = \frac{P_3 y_2}{a_0+x}, z_5 = \frac{P_1 y_2}{a_1+x}, z_6 = \beta y_i, z_7 = \frac{P_2 y_2}{a_2+x}, z_8 = \frac{P_3 y_2}{a_1+x}, z_9 = \frac{P_3 y_2}{a_2+x}$. $M_i, x(t), A_i \in R^{4 \times 4}$, r is the number of the IF-THEN rules, $K_{i,j}$ denotes the control of the j^{th} impulsive instant, $\Delta(x)|_{t=\tau_j} = x(\tau_j - \tau_{j-1})$

9.4 Numerical Simulation

By using fuzzy impulsive T-S design model on (9.2), the membership functions [96] obtained as

$$\begin{aligned} M_1 &= \frac{z_1}{ed_1}, M_2 = \frac{ed_1 - z_1}{ed_1}, N_1 = \frac{z_2}{\frac{P_1 d_2}{a_0+d_1}}, N_2 = \frac{\frac{P_1 d_2}{(a_0+d_1)} - z_2}{\frac{P_1 d_2}{(a_0+d_1)}}, K_1 = \frac{z_3}{\frac{P_2 d_3}{a_0+d_1}}, \\ K_2 &= \frac{\frac{P_2 d_3}{a_0+d_1} - z_3}{\frac{P_2 d_3}{a_0+d_1}}, L_1 = \frac{z_4}{\frac{P_3 d_4}{a_0+d_1}}, L_2 = \frac{\frac{P_3 d_4}{a_0+d_1} - z_4}{\frac{P_3 d_4}{a_0+d_1}}, O_1 = \frac{z_5}{\frac{P_1 d_4}{a_1+d_1}}, O_2 = \frac{\frac{P_1 d_4}{a_1+d_1} - z_5}{\frac{P_1 d_4}{a_1+d_1}}, \\ R_1 &= \frac{z_6}{\beta d_3}, R_2 = \frac{\beta d_3 - z_6}{\beta d_3}, S_1 = \frac{z_7}{\frac{P_2 d_4}{a_2+d_1}}, S_2 = \frac{\frac{P_2 d_4}{a_2+d_1} - z_7}{\frac{P_2 d_4}{a_2+d_1}}, T_1 = \frac{z_8}{\frac{P_3 d_4}{a_1+d_1}}, T_2 = \frac{\frac{P_3 d_4}{a_1+d_1} - z_8}{\frac{P_3 d_4}{a_1+d_1}}, \\ P_1 &= \frac{z_9}{\frac{P_3 d_4}{a_2+d_1}}, P_2 = \frac{\frac{P_3 d_4}{a_2+d_1} - z_9}{\frac{P_3 d_4}{a_2+d_1}} \end{aligned}$$

and the matrices A'_i s are calculated using

$$A_i = \begin{bmatrix} r - ex - \frac{P_1 y_s}{a_0+x} - \frac{P_2 y_i}{a_0+x} - \frac{P_3 y_2}{a_0+x} & 0 & 0 & 0 \\ \frac{C_1 P_1 y_s}{a_0+x} & -\frac{C_1 P_1 y_2}{a_1+x} - \beta y_i - m_1 & 0 & 0 \\ \frac{C_2 P_2 y_i}{a_0+x} & \beta y_i & -\frac{C_2 P_2 y_2}{a_2+x} - m_2 & 0 \\ \frac{C_3 P_3 y_2}{a_0+x} & -\frac{C_3 P_3 y_2}{a_1+x} & \frac{C_3 P_3 y_2}{a_2+x} & -m_3 \end{bmatrix},$$

$i = 1$ to 511, where the matrices A'_i s are generated using maximum and minimum

values of $z'_k s$; $k = 1$ to 9 and, the Defuzzification can be represented as:

$$\dot{x}(t) = \sum_{i=1}^r h_i(z(t))(A_i x(t)) \quad (9.4)$$

here $h'_i s$ are given as $h_i(z(t)) = \omega_i(z(t)) / \sum_{i=1}^r \omega_i(z(t))$, and $\omega_i(z(t)) = \prod_{j=1}^p M_{ij}(z(t))$, $i = 1$ to 511 , $j = 1$ to 9

This Fuzzy model exactly represents the non-linear system 9.1 in the region $[0,5] \times [0,10] \times [0,10] \times [0,10]$.

$$\begin{aligned} \frac{dx}{dt} &= rx - ex^2 - \frac{P_1 y_s x}{a_0 + x} - \frac{P_2 y_i x}{a_0 + x} - \frac{P_3 y_2 x}{a_0 + x} \\ \frac{dy_s}{dt} &= \frac{C_1 P_1 y_s x}{a_0 + x} - \frac{C_1 P_1 y_s y_2}{a_1 + x} - \beta y_s y_i - m_1 y_s \\ \frac{dy_i}{dt} &= \beta y_s y_i + \frac{C_2 P_2 y_i x}{a_0 + x} - \frac{C_2 P_2 y_i y_2}{a_2 + x} - m_2 y_i \\ \frac{dy_2}{dt} &= \frac{C_3 P_3 x y_2}{a_0 + x} - \frac{C_3 P_3 y_s y_2}{a_1 + x} + \frac{C_3 P_3 y_i y_2}{a_2 + x} - m_3 y_2 \end{aligned} \quad (9.5)$$

9.5 Results and discussion

In this section, the global stability of the considered intra-specific competition predator-prey model (9.2) is discussed as discussed in earlier chapters. We have studied the system (9.1) numerically using MATHEMATICA software to get better insight of the proposed model.

Calculations were carried by taking the values of the parameters at $r = 1.5$, $e = 0.2$, $\beta = 0.4$, $P_1 = 0.7$, $P_2 = 0.06$, $P_3 = 0.8$, $C_1 = 0.5$, $C_2 = 0.4$, $C_3 = 0.36$, $m_1 = 0.1$, $m_2 = 0.5$, $m_3 = 0.4$, $a_0 = 1.0$, $a_1 = 1.0$, $a_2 = 1.0$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$ in 9.3 to get the eigen values of $[A_i^T + A_i](i = 1, 2, 3 \dots r)$ as explained in the theorems ([83]). It is found that $\max(\lambda_i) = \lambda(\alpha) = 3$ then we have chosen $\text{diag}[-0.99, -0.99]$ as impulsive control matrix, such that $\omega = \|I + K\| = 0.01$.

It is noted that the system 9.3 is stable globally when $\epsilon=1.5$, $\delta_j=0.1$ (at those above values, $\ln(\epsilon\omega) + \lambda(\alpha)\delta_j = -3.899 < 0$). Further, it is observed that the prey-predator model is unstable when $r = 21.2$, $e = 5.5$, $\beta = 5.5$, $P_1 = 2.8$, $P_2 = 2.8$, $P_3 = 2.0$, $C_1 = 1.8$, $C_2 = 2.2$, $C_3 = 5.6$, $m_1 = 5.2$, $m_2 = 5.4$, $m_3 = 0.5$, $a_0 = 1.0$, $a_1 = 1.0$, $a_2 = 1.0$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, since $\max(\lambda_i) = \lambda(\alpha) = 42.4$, $\implies \ln(\epsilon\omega) + \lambda(\alpha)\delta_j = 0.041 > 0$ for $\omega = 0.01$, $\epsilon = 1.5$, $\delta_j = 0.1$.

Table. 9.1 presents the stability of the system at various values of the present study.

r	e	β	P_1	P_2	P_3	C_1	C_2	C_3	m_1	m_2	m_3	a_0	a_1	a_2	d_1	d_2	d_3	d_4	$\max(\lambda_i) = \lambda(\alpha)$	$\ln(\epsilon\omega) + \lambda(\alpha)\delta_j$	conclusion
1.5	0.2	0.4	0.7	0.06	0.8	0.5	0.4	0.36	0.1	0.5	0.4	1.0	1.0	1.0	10	10	10	10	3	-3.899	stable
2.0	0.5	0.5	0.8	0.6	1.0	0.5	0.4	0.6	0.5	0.2	0.5	1.0	1.0	1.0	10	10	10	10	4.0	-3.799	stable
2.5	1.5	0.2	1.8	1.6	2.0	0.8	0.2	0.6	0.2	0.4	0.5	1.0	1.0	1.0	10	10	10	10	5	-3.699	stable
21.2	5.5	5.5	2.8	2.8	2.0	1.8	2.2	5.6	5.2	5.4	0.5	1.0	1.0	1.0	10	10	10	10	42.4	0.041	unstable

Table 9.1: Stability Analysis by taking different values of the parameters

The impact of the emerging parameters on prey-predator system 9.1 with T-S fuzzy impulsive control model is presented in figs. 9.1 - 9.10 by fixing other parameters at $P_1 = 0.7$, $P_2 = 0.06$, $P_3 = 0.8$, $C_1 = 0.5$, $C_2 = 0.4286$, $C_3 = 0.36$, $a_0 = 1.0$, $a_1 = 1.0$, $a_2 = 1.0$, $m_1 = 0.1$.

The dynamical change on prey-predator population system (x, y_s, y_i, y_2) by varying intrinsic growth rate of prey (r) parameter under fuzzy impulsive control can be noted in fig. 9.1 at $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. It is observed from this figure that, increase in r increases population of prey.

The effectiveness by varying intra-specific competition (e) parameter of prey-predator population system (x, y_s, y_i, y_2) under fuzzy impulsive control can be noted in fig. 9.2 at $r = 1.5$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly shows that an increase in e leads to the decreases in the population of prey, but increases the infected first predator and the second predator

population.

The influence of prey max time (d_1) on prey-predator system is shown in fig. 9.3 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This graph makes it abundantly evident that as d_1 increases, population increases for prey and first predator but decreases for second predator.

The influence of susceptible first predator max time (d_2) on prey-predator system is shown in fig. 9.4 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_3 = 10$, $d_4 = 10$. This graph shows that as d_2 increases, prey population decreases but predators population increases.

The change on prey-predator system with max time of infected first predator (d_3) is shown in fig. 9.5 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_4 = 10$. This figure clearly exhibits that as d_3 increases, prey and susceptible first predator population decreases.

The outcome with varying max time of second predator (d_4) on prey-predator system is shown in fig. 9.6 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$. This graph illustrates clearly how an increase in second predator max time decreases the prey population and first predator population and increases the second predator population.

The effect of transmission coefficient parameter (β) from susceptible first predator to infected first predator on prey-predator system is shown in fig. 9.7 at $r = 1.5$, $e = 0.2$, $m_2 = 0.5$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This graph shows that as transmission coefficient from susceptible first predator to infected first predator rise, the population of susceptible first predator decreases.

The vital pattern of prey- predator population (x, y_s, y_i, y_2) by varying mortality rate of infected first predator (m_2) parameter under fuzzy impulsive control can be noted in fig. 9.8 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_3 = 0.4$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly exhibits that as m_2 increases, population of susceptible

and infected first predator decreases.

The change on prey-predator system (x, y_s, y_i, y_2) by varying mortality rate of second predator (m_3) parameter under fuzzy impulsive control can be noted in fig. 9.9 at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $m_2 = 0.5$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$. This figure clearly exhibits that as m_3 increases, population of prey and second predator decreases but population of infected first predator increases.

Finally, the nature of prey-predator system without impulsive control is presented in fig. 9.10 by fixing all the parameters obtained from T-S fuzzy model at $r = 1.5$, $e = 0.2$, $\beta = 0.7$, $P_1 = 0.7$, $P_2 = 0.06$, $P_3 = 0.8$, $C_1 = 0.5$, $C_2 = 0.4$, $C_3 = 0.36$, $m_1 = 0.1$, $m_2 = 0.5$, $m_3 = 0.4$, $a_0 = 1.0$, $a_1 = 1.0$, $a_2 = 1.0$, $d_1 = 10$, $d_2 = 10$, $d_3 = 10$, $d_4 = 10$, and initial conditions $x(0) > 0$, $y_s(0) > 0$, $y_i(0) > 0$, $y_2(0) > 0$, $t = 10$. The figure clearly shows how the prey and predator populations reaches to stability.

9.6 Conclusions

In this work, a predator-prey model with two predator populations and one prey population is built, but only the first predator population is infected.

- Intrinsic growth rate of prey effects all the four populations. Increase in intrinsic growth rate of prey increases population of prey, and decreases population of susceptible first predator, infected first predator and second predator.
- The population of first and second predators that are infected increases while the population of prey falls due to increased intra-specific competition.
- The population of susceptible first predators and infected first predators declines as the mortality rate of infected first predators rises.
- Effect of infected first predator max time is to decrease all the four populations.

-
- The population of predator decreases due to increase in infection transmission.

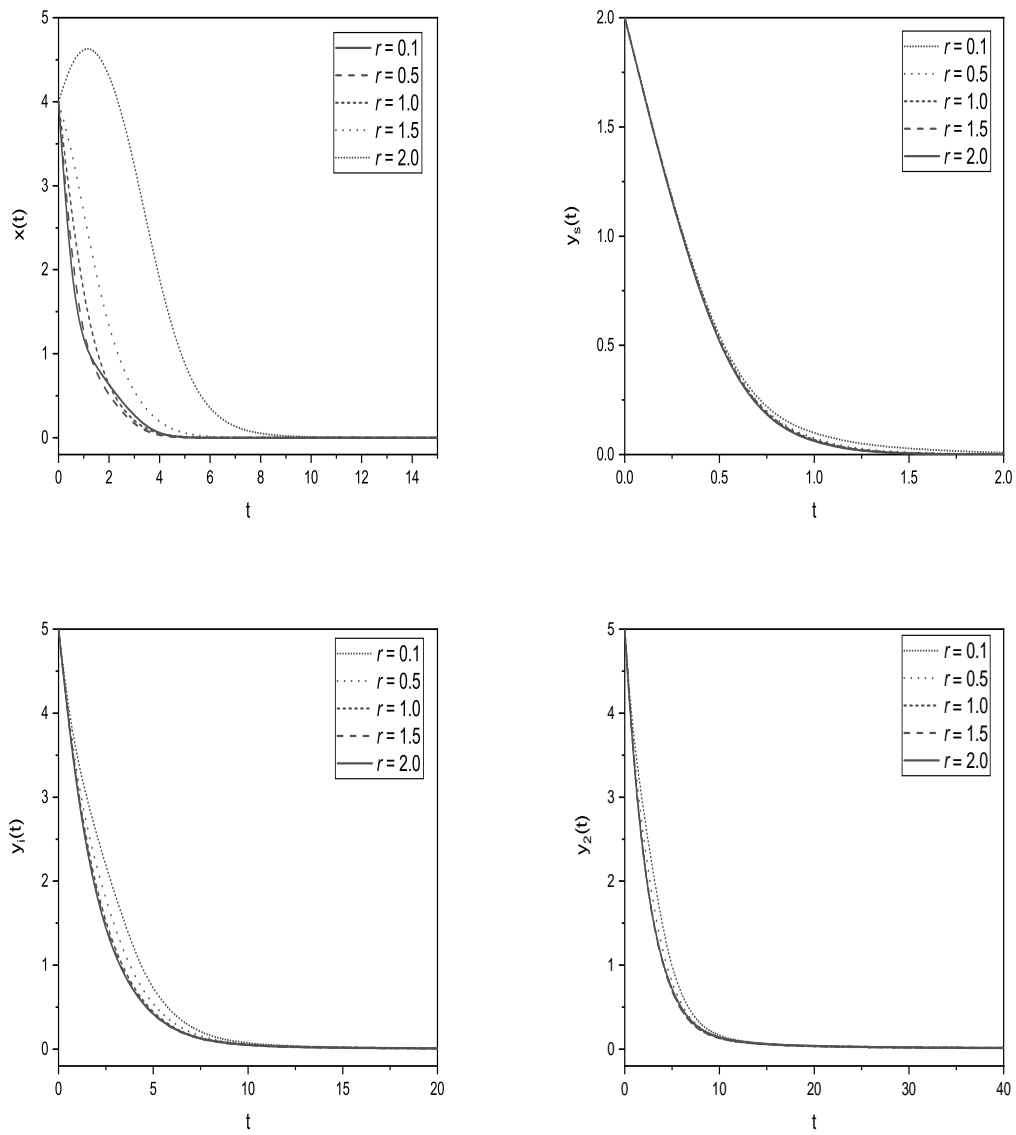


Figure 9.1: The effect of intrinsic growth rate of prey (r) parameter on the prey-predator system

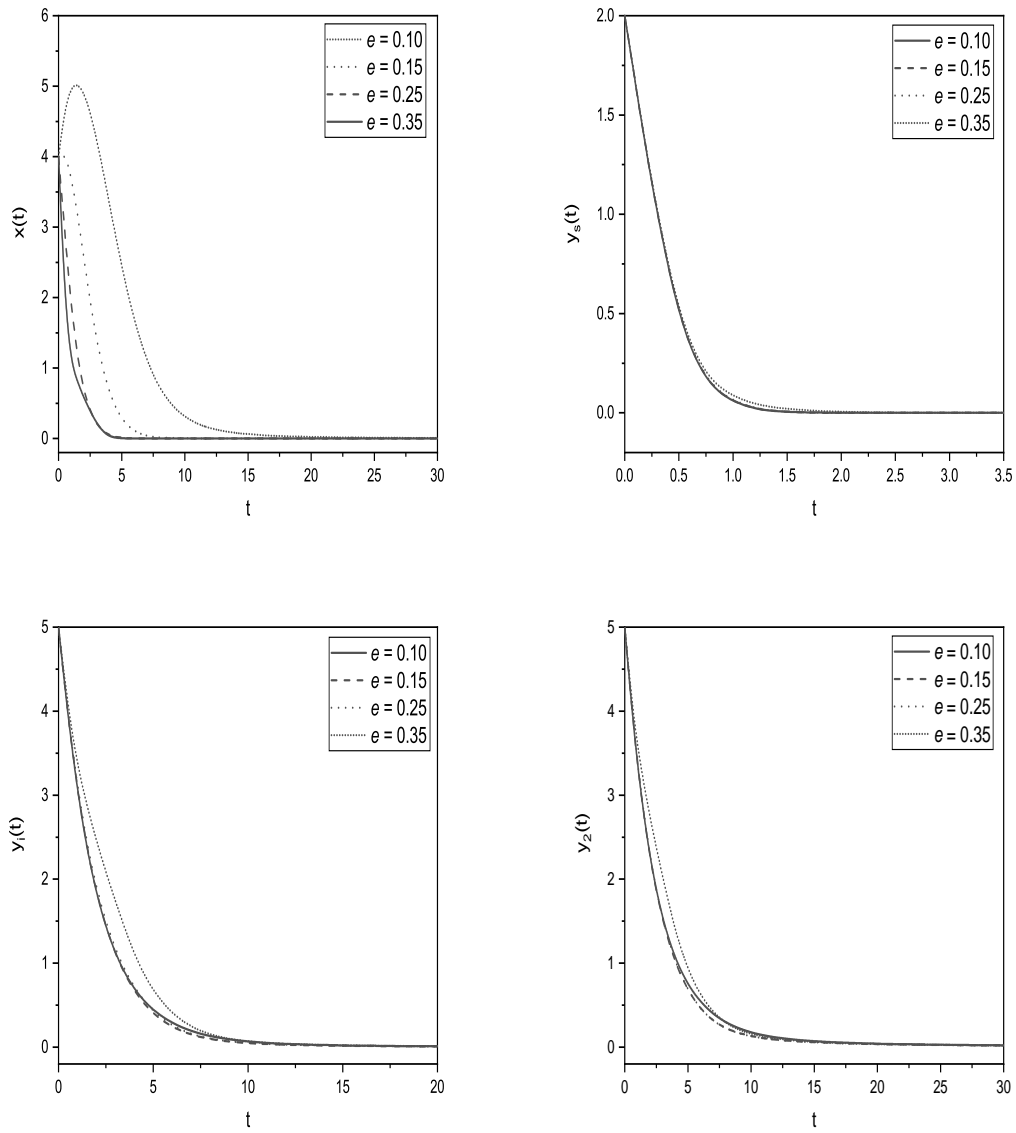


Figure 9.2: The effect of intra-specific competition (e) parameter on the prey-predator system

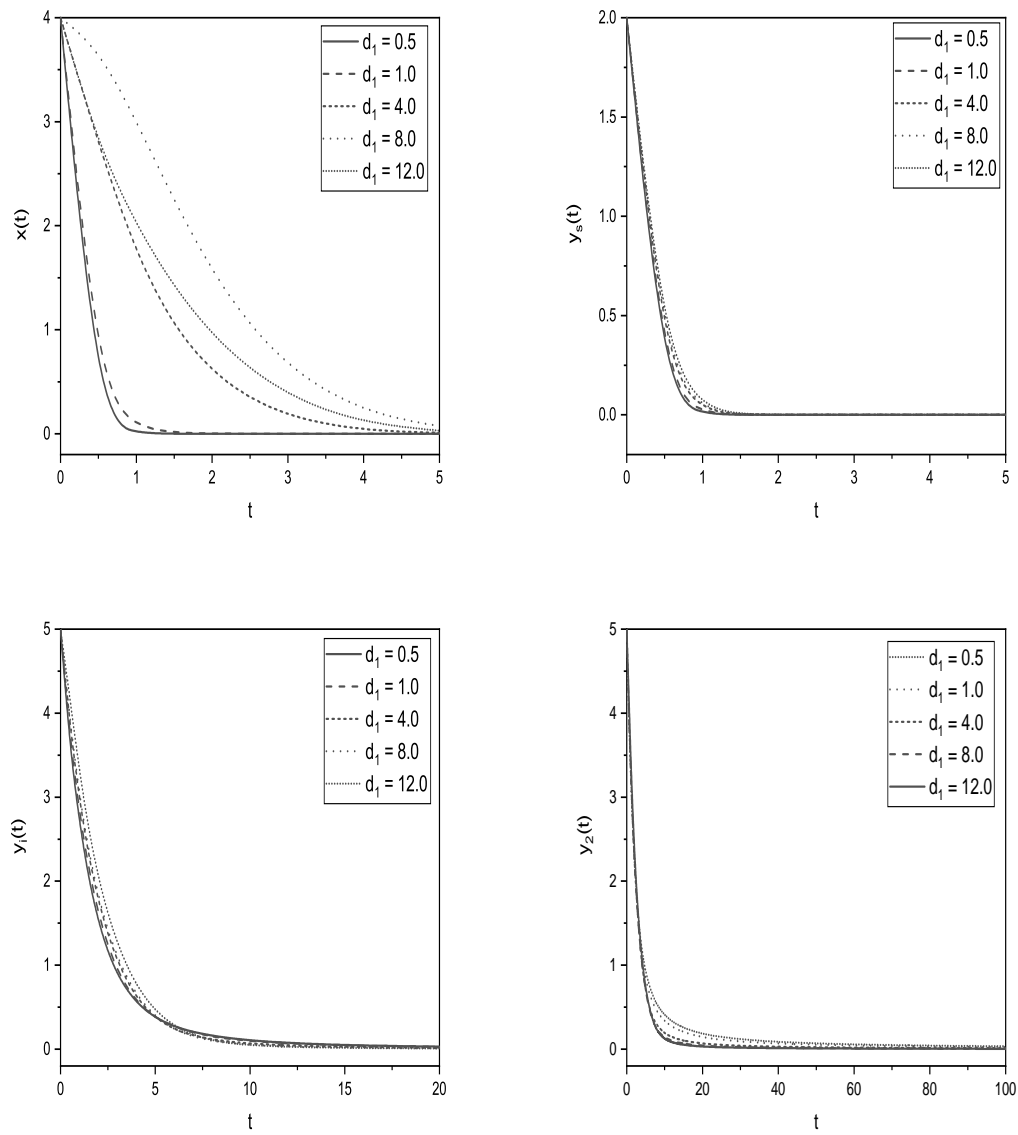


Figure 9.3: The effect of max time of prey (d_1) parameter on the prey-predator system

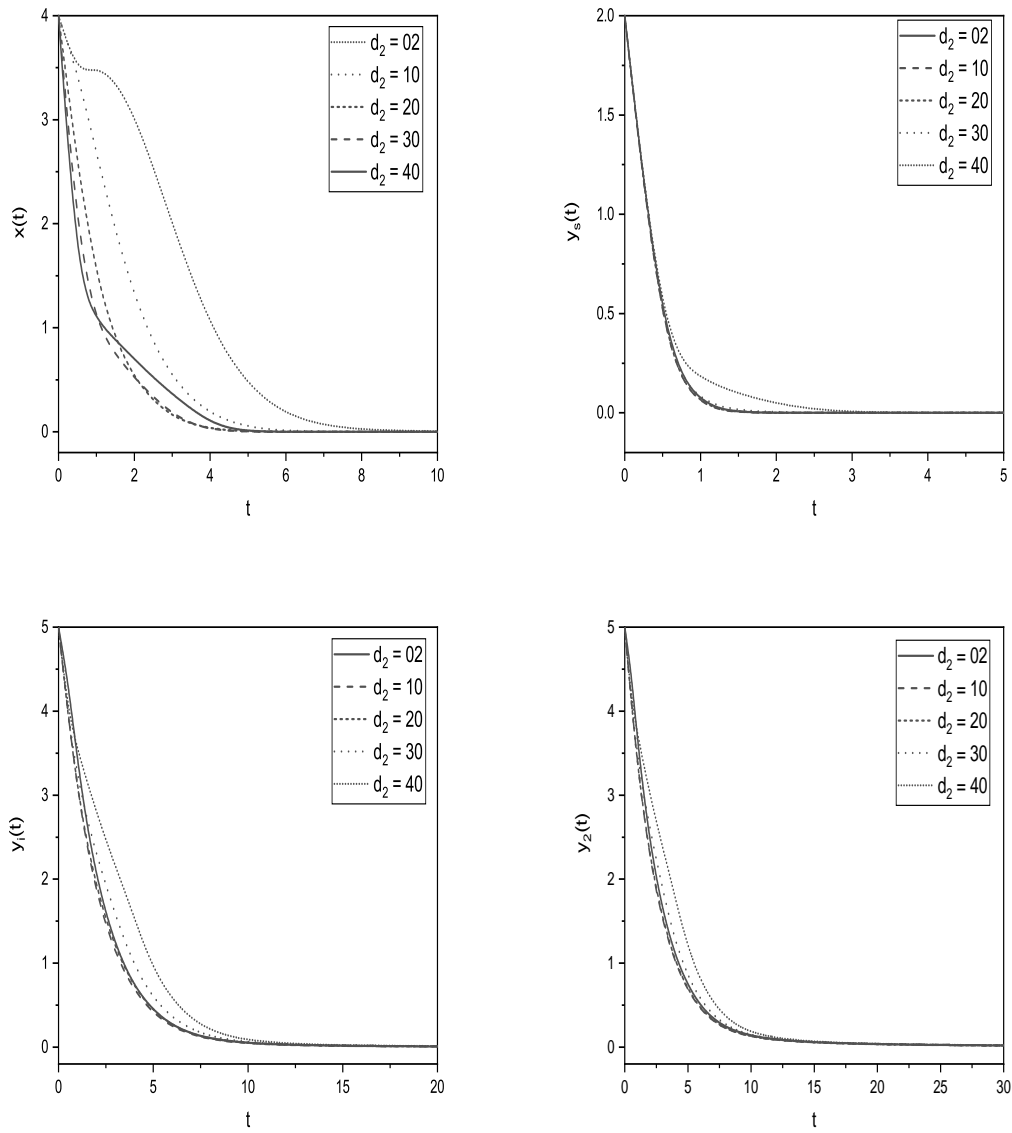


Figure 9.4: The effect of max time of susceptible first predator (d_2) parameter on the prey-predator system

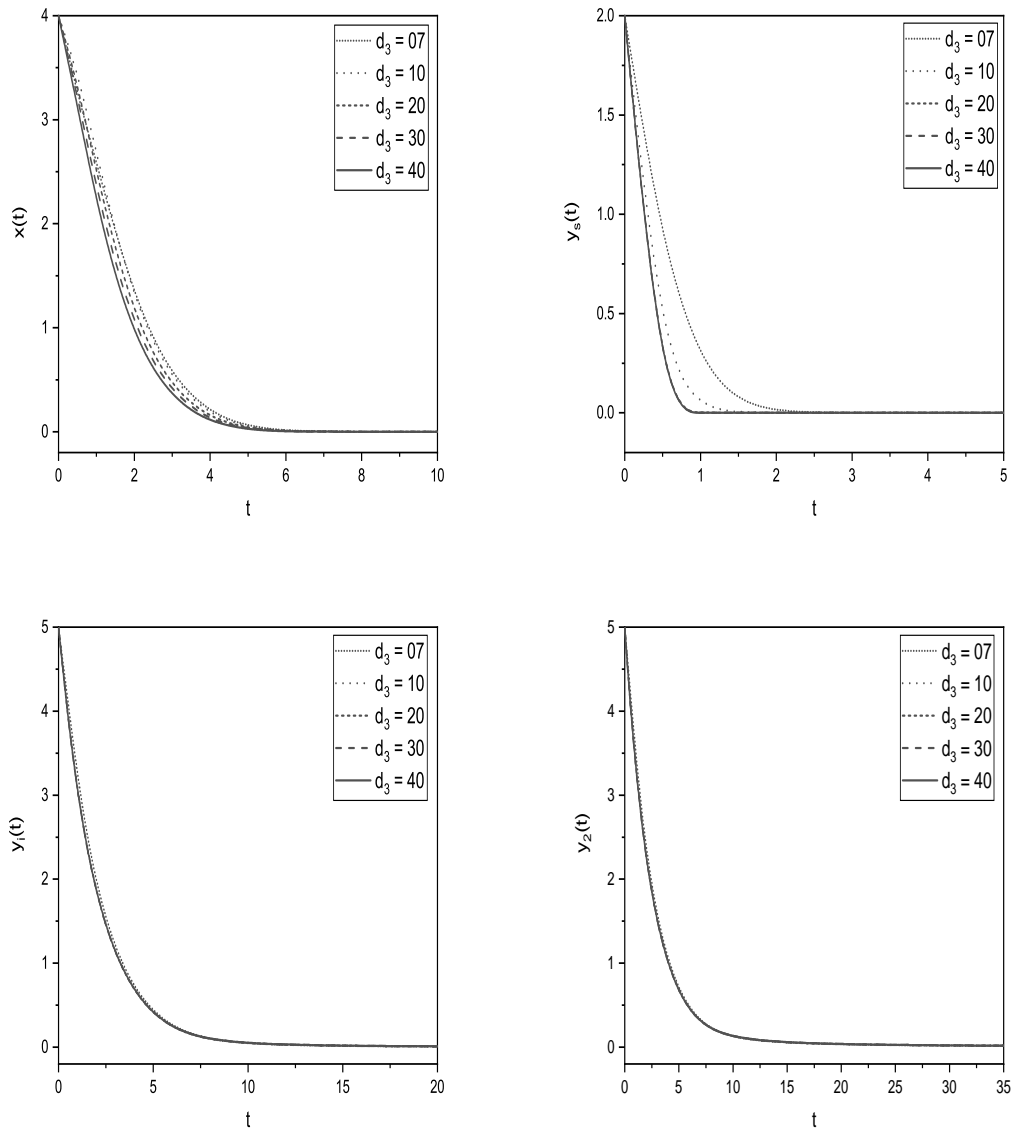


Figure 9.5: The effect of max time of infected first predator (d_3) parameter on the prey-predator system

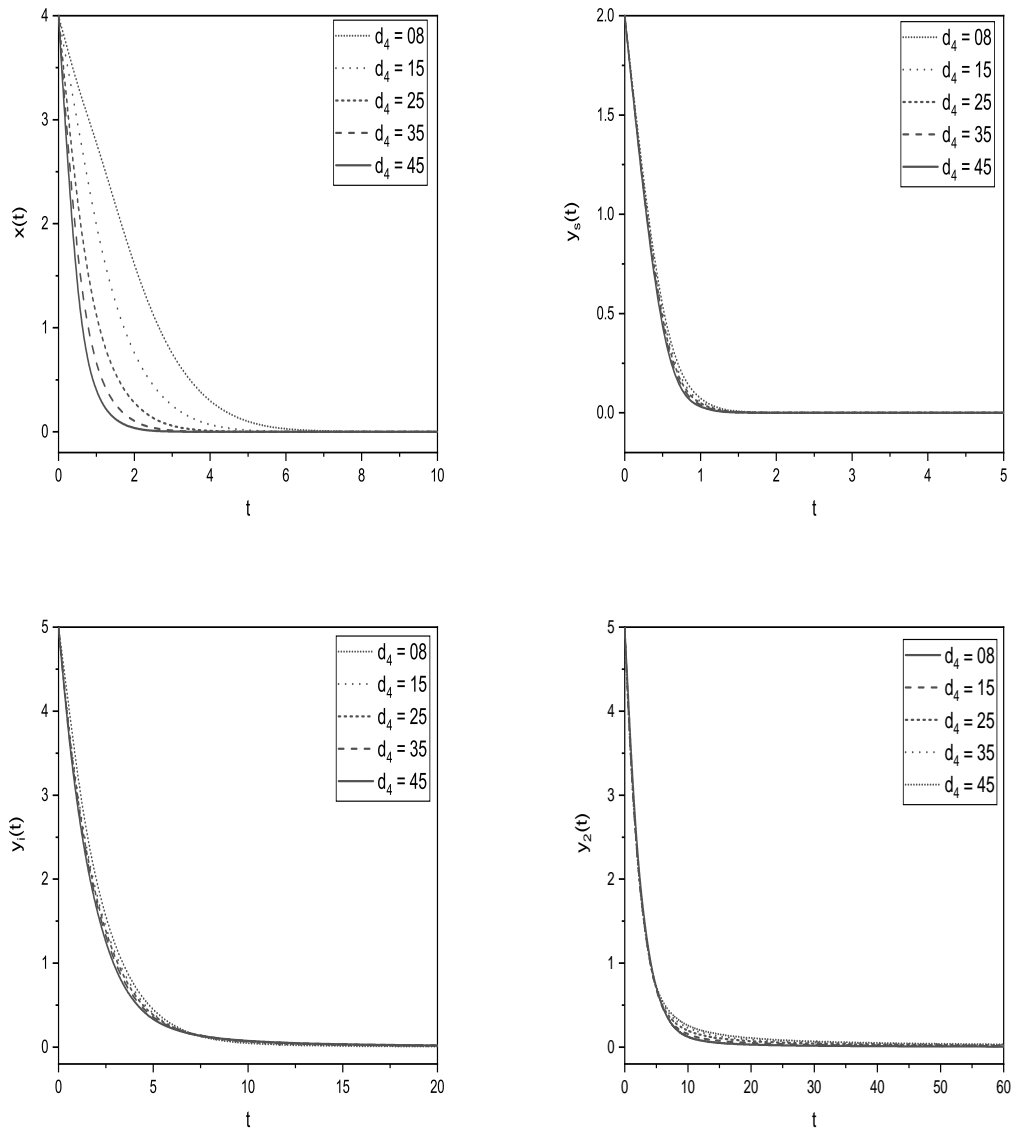


Figure 9.6: The effect of max time of second predator (d_4) parameter on the prey-predator system

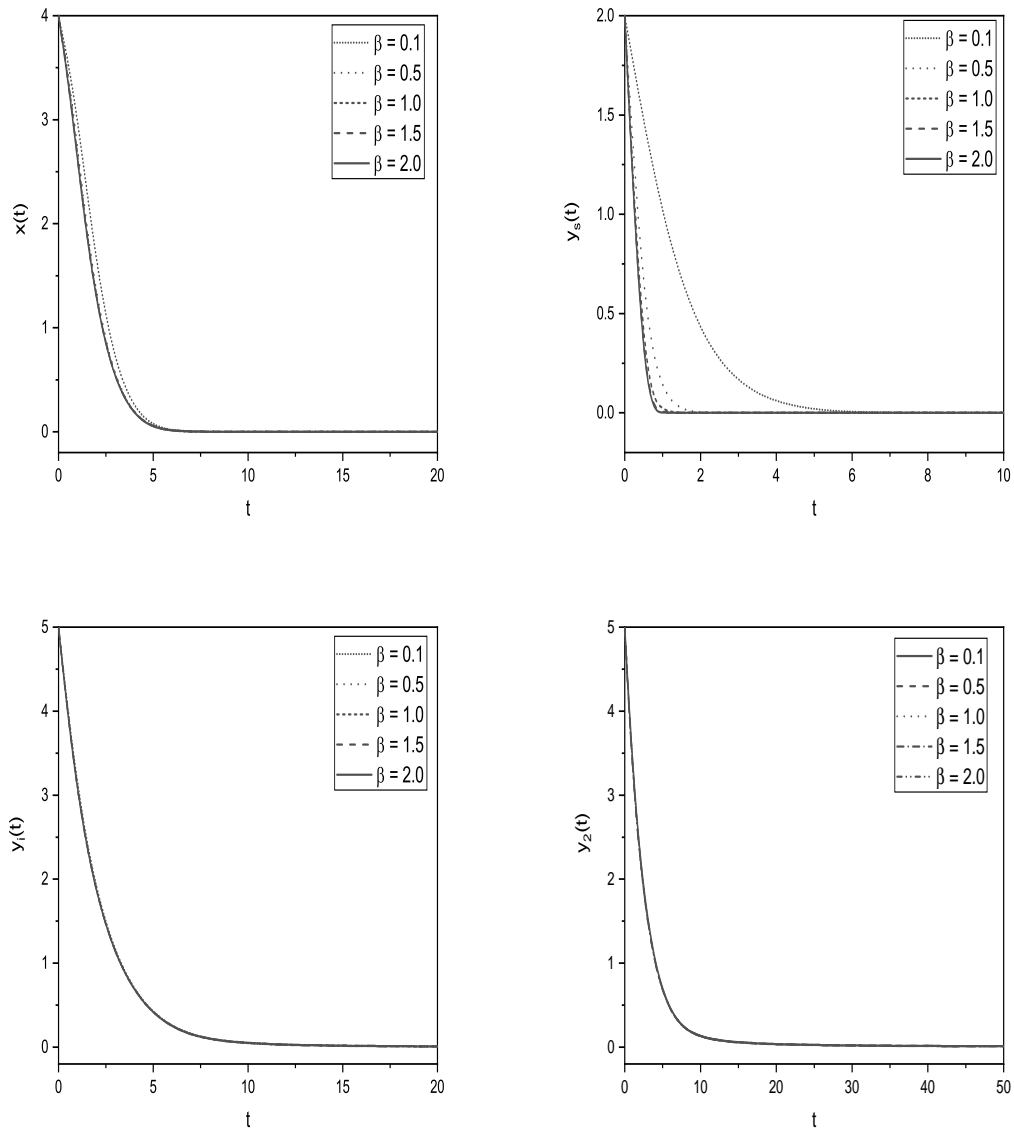


Figure 9.7: The effect of transmission coefficient from susceptible first predator to infected first predator (β) parameter on the prey-predator system

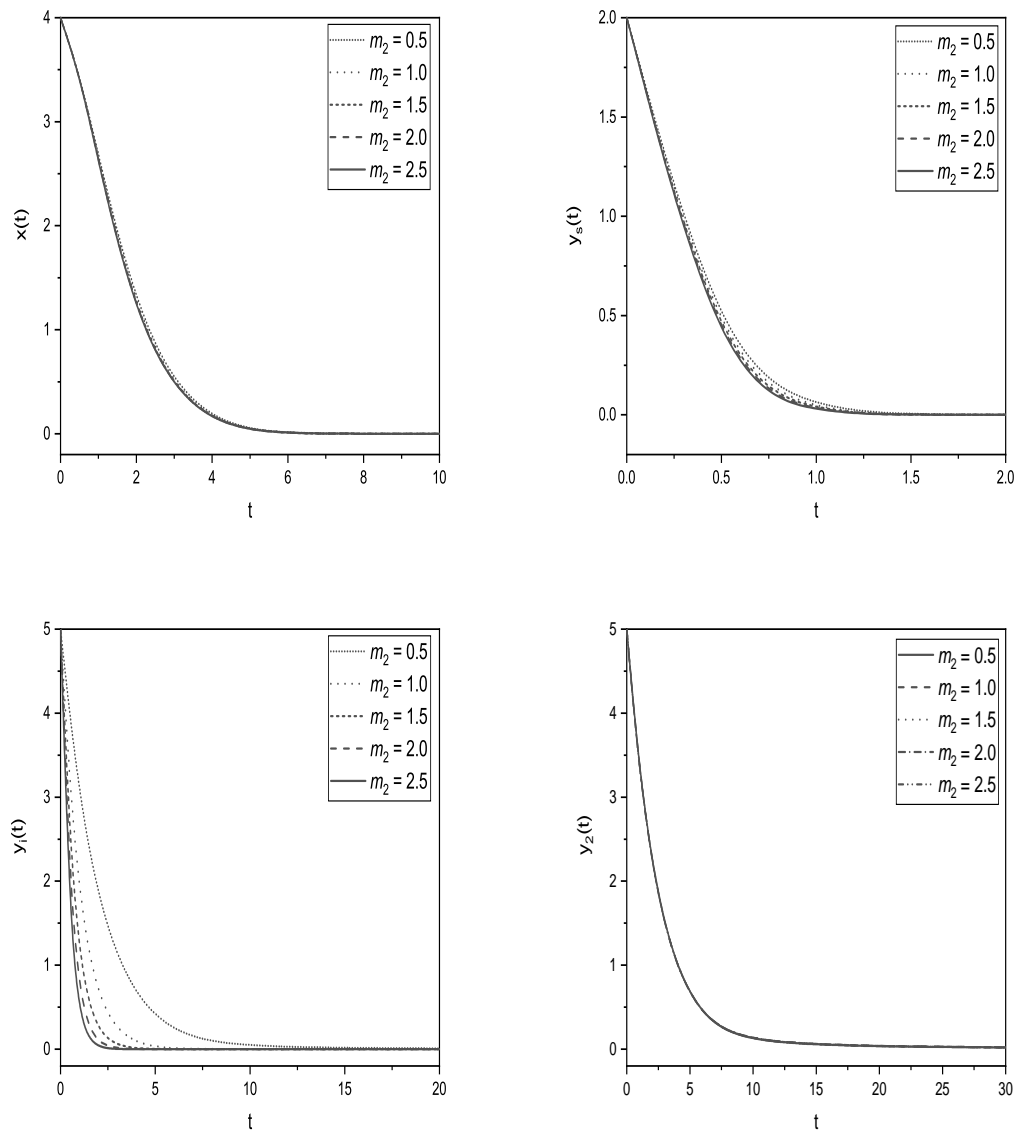


Figure 9.8: The effect of mortality rate of infected first predator (m_2) parameter on the prey-predator system

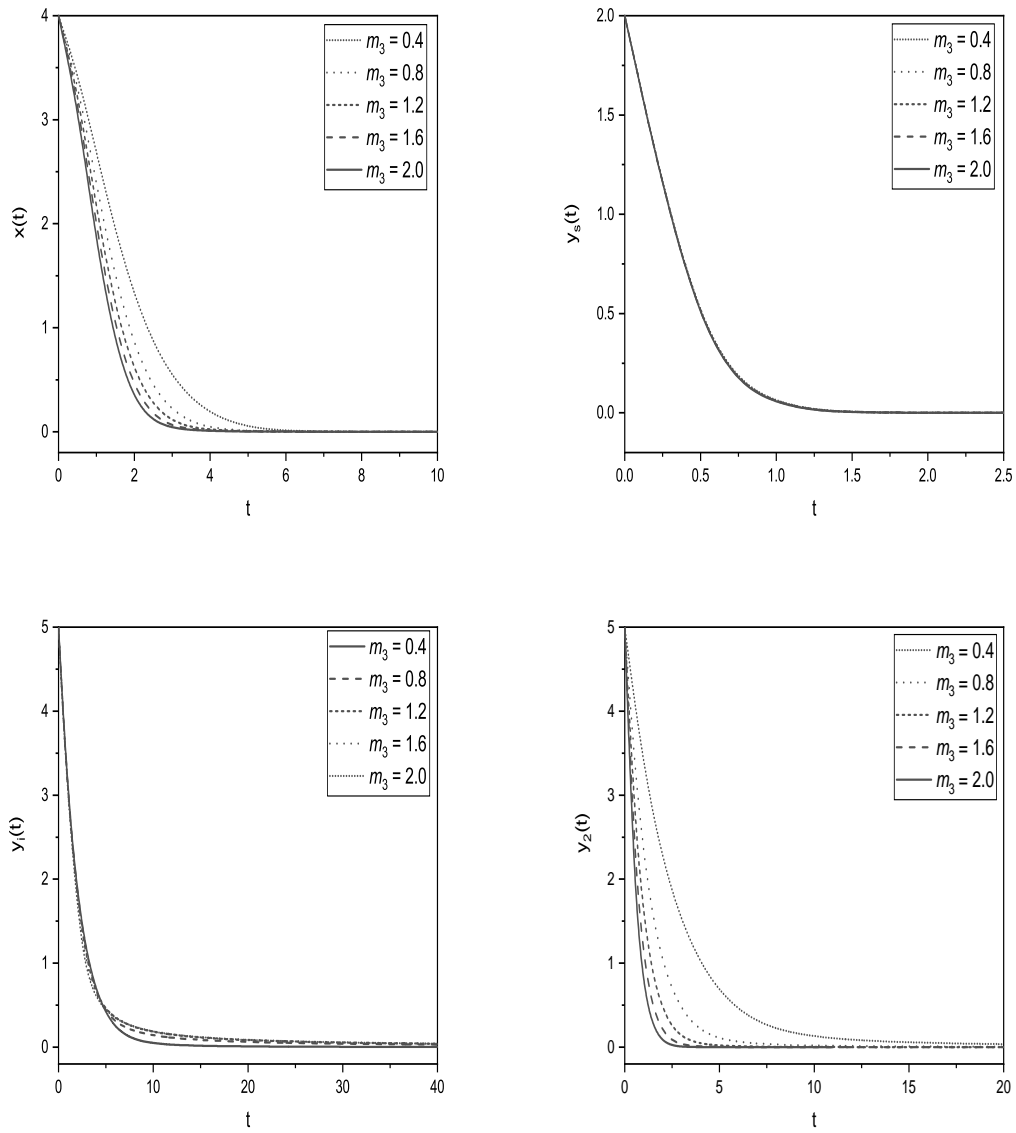


Figure 9.9: The effect of mortality rate of second predator (m_3) parameter on the prey-predator system

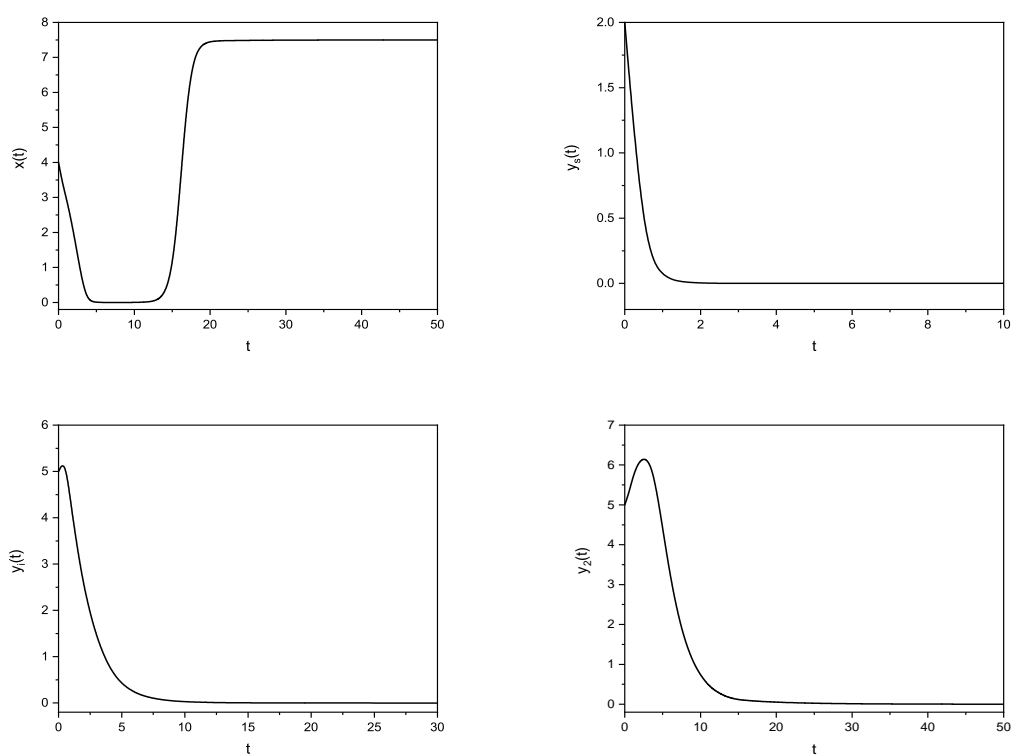


Figure 9.10: Plot of predator-prey system without impulsive control.

Part IV

Conclusions and Scope for Future Work

Chapter 10

Conclusions and Scope for Future Work

The Takagi-Sugeno method has diverse applications in modeling, control, pattern recognition, and decision-making in systems where uncertainty and non-linearity play a significant role. Its ability to combine fuzzy logic with traditional mathematical models provides a powerful tool for addressing complex real-world problems.

Many fields are based on mathematical models, including ecology, epidemiology, physics, algorithms, and infectious diseases. In this thesis, we analyzed the stability of a predator-prey competition model with fuzzy impulsive control by T-S fuzzification. According to the references already in existence, the current investigation covers a variety of ecological consequences and got adequate results.

Significance of Research Findings:

- Stability analysis of prey-predator helps in predicting how populations of predators and prey will change over time.
- It provides insights into how ecosystems respond to perturbations, such as environmental changes or species introduction.

- For conservation biology and wildlife management, stability analysis can guide strategies to maintain or restore balanced populations.
- Stability analysis can predict the outcomes of introducing natural predators to control pest populations and avoid unintended consequences.
- This method contributes to the development of mathematical techniques and theories applicable to various complex systems.
- It aids in understanding the spread and control of diseases, guiding public health interventions.

Practical implications of this model:

- Enhanced Control for Nonlinear Systems: The T-S fuzzy impulsive control model excels in managing nonlinear systems where traditional control methods may fail.
- Robustness to Disturbances and Uncertainties: The model's ability to manage impulses (sudden changes or disturbances) makes it robust against uncertainties. This is particularly useful in environments where external disturbances are frequent and unpredictable, such as in robotic systems or aerospace applications.
- Improved Stability and Performance: T-S fuzzy impulsive control can improve the stability and performance of dynamic systems. By applying impulsive control actions at specific instances, the system can quickly adjust to changes, maintaining desired performance levels.
- Applications in Engineering Systems: The model is widely applicable in engineering fields such as automotive systems, power systems, and mechanical systems.

In [113], Wang et.al. discusses the application of impulsive control in stabilizing T-S fuzzy systems with time delays, highlighting its practical implications in real-world systems. In [114], Zhang et.al. offers a comprehensive overview of fuzzy modeling and control, including the T-S fuzzy model with impulsive control, and discusses various applications in engineering and technology.

The main results indicate the following findings.

Conclusions from Part-II

For the investigation of global and asymptotic stability of two and three species Lotka-Volterra predator-prey model, we have used the T-S method, and then presented the graphical solutions of the problems.

The following are some of the important observations.

- The effect of intra-species competition is to decrease the rate of population growth as population density increases.
- It has been observed that intrinsic growth rate of prey increases with an increase in prey's population.
- The half-saturation constants are inversely proportional to predators population. Decrease in half saturation constant indicate that predators can achieve half of their maximum consumption rate at a lower prey density, which means they are more efficient in utilizing the preys and hence predator population rises.

Conclusions from Part-III

We have analyzed the effect of disease in prey and predator populations. The diseased population has been split into two sub-classes: susceptible and infected.

The following are some of the important observations.

- There are less healthy people in the population when the rate of disease transfer from susceptible to sick people rises.
- A higher death rate results in a smaller population.

Future Scope:

The work presented in the thesis can be extended to analyze the Allee effect and Time-delay in four species predator-prey models and to study about their stability using Takagi-Sugeno method. Further, we can use T-S method in several engineering fields, including near space vehicles, sewage treatment processes and nonlinear active suspension systems and for analyzing the share market.

List of Papers Published/Accepted/Communicated

List of Publications

1. Khushbu Singh, Kaladhar Kolla, "Stability analysis of prey-predator model with two prey and one predator using fuzzy impulsive control", International Journal of Dynamics and Control, 12:1116–1129 (2024), <https://doi.org/10.1007/s40435-023-01189-3>
2. Khushbu Singh, Kolla Kaladhar, "A Mathematical Study for the Stability of Two Predator and One Prey with Infection in First Predator using Fuzzy Impulsive Control", Ann. Appl. Math., Vol. 39, pp. 29-48 (2023)
3. Khushbu Singh, K. Kaladhar, "Population Dynamic study of interaction between two Predator and one Prey", Physica Scripta, 99 (2024) 025023, DOI:10.1088/1402-4896/ad1dd2
4. Khushbu Singh, Kaladhar Kolla, "Population Dynamic Study of two prey one predator system with disease in first prey using Fuzzy Impulsive Control", Epidemiological Methods, 2024; 13(1): 20230019, DOI: <https://doi.org/10.1515/em-2023-0037>
5. K. Kaladhar, Khushbu Singh, "Stability analysis of a T-S based intra-specific predator-prey competition model with Fuzzy Impulsive Control", Journal of Applied Nonlinear Dynamics, 13(2) (2024) 269-277, DOI:10.5890/JAND.2024.06.007
6. K. Kaladhar, Khushbu Singh, "Stability analysis of a T-S Prey-Predator model with disease in both species using Fuzzy Impulsive Control", Journal of Environmental Accounting and Management, 12(3), (2024), 231-244. DOI:10.5890/JEAM.2024.09.002
7. Kaladhar Kolla, Khushbu Singh, "Stability Analysis of a T-S Predator-Prey model with infection in Predator using Fuzzy Impulsive Control", Network Biology, 14(3): 215-227, (2024)

List of the Papers Communicated for Publication

1. A Takagi-Sugeno based study of population dynamics with infected prey, communicated to *Journal of Applied Mathematics and Computing*.

Bibliography

- [1] Alfred James Lotka. *Elements of physical biology*. Williams & Wilkins, 1925.
- [2] Vito Volterra. Fluctuations in the abundance of a species considered mathematically. *Nature*, 118(2972):558–560, 1926.
- [3] V Volterra. Variations and fluctuations of the number of individuals in animal species living together. *Memorie della R. Accademia Nazionale dei Lincei*, 2:31–113, 1926.
- [4] William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721, 1927.
- [5] Roy M Anderson and Robert M May. *Infectious diseases of humans: dynamics and control*. Oxford university press, Oxford, 1992.
- [6] Herbert W Hethcote. A thousand and one epidemic models. In *Frontiers in mathematical biology*, pages 504–515. Springer, U.S.A, 1994.
- [7] Huaguang Zhang, Jun Yang, and Chun Yi Su. Ts fuzzy-model-based robust design for networked control systems with uncertainties. *IEEE Transactions on Industrial Informatics*, 3(4):289–301, 2007.
- [8] Ted Cohen and Peter White. Transmission-dynamic models of infectious diseases. *Infectious Disease Epidemiology (Oxford Specialist Handbooks)*; Abubakar, I., Stagg, HR, Cohen, T., Rodrigues, LC, Eds, pages 223–242, 2016.
- [9] Fred Brauer, Carlos Castillo-Chavez, Zhilan Feng, et al. *Mathematical models in epidemiology*, volume 32. Springer, 2019.

- [10] Assane Savadogo, Boureima Sangaré, and Hamidou Ouedraogo. A mathematical analysis of prey-predator population dynamics in the presence of an infectious disease. *Research in Mathematics*, 9(1):2020399, 2022.
- [11] Plamen P Angelov and Dimitar P Filev. An approach to online identification of takagi-sugeno fuzzy models. *IEEE Transactions on Systems, Man, and Cybernetics, Part B (Cybernetics)*, 34(1):484–498, 2004.
- [12] Zsófia Lendek, Thierry Marie Guerra, Robert Babuska, and Bart De Schutter. *Stability analysis and nonlinear observer design using Takagi-Sugeno fuzzy models*, volume 262. Springer, 2011.
- [13] Zhixiong Zhong, Xingyi Wang, and Hak-Keung Lam. Finite-time fuzzy sliding mode control for nonlinear descriptor systems. *IEEE/CAA Journal of Automatica Sinica*, 8(6):1141–1152, 2021.
- [14] Jiun-Hong Lai and Chin-Teng Lin. Application of neural fuzzy network to pyrometer correction and temperature control in rapid thermal processing. *IEEE Transactions on Fuzzy Systems*, 7(2):160–175, 1999.
- [15] V.G. Moudgal, W.A. Kwong, K.M. Passino, and S. Yurkovich. Fuzzy learning control for a flexible-link robot. *IEEE Transactions on Fuzzy Systems*, 3(2):199–210, 1995.
- [16] J.T. Spooner and K.M. Passino. Stable adaptive control using fuzzy systems and neural networks. *IEEE Transactions on Fuzzy Systems*, 4(3):339–359, 1996.
- [17] J Maynard Smith and M Slatkin. The stability of predator-prey systems. *Ecology*, 54(2):384–391, 1973.
- [18] Dong Li, Shilong Wang, Xiaohong Zhang, and Dan Yang. Impulsive control of uncertain lotka–volterra predator–prey system. *Chaos, Solitons & Fractals*, 41(4):1572–1577, 2009.
- [19] Sanyi Tang and Lansun Chen. The periodic predator-prey lotka–volterra model with impulsive effect. *Journal of Mechanics in Medicine and Biology*, 2(03n04):267–296, 2002.
- [20] Stephen Cecil Ewing. *Application of the lotka-volterra dynamical equations to natural populations*. PhD thesis, Citeseer, 1986.

- [21] Guangye Chen, Zhidong Teng, and Zengyun Hu. Analysis of stability for a discrete ratio-dependent predator-prey system. *Indian Journal of Pure and Applied Mathematics*, 42(1):1–26, 2011.
- [22] Mainul Haque and Ezio Venturino. The role of transmissible diseases in the holling–tanner predator–prey model. *Theoretical Population Biology*, 70(3):273–288, 2006.
- [23] GS Mahapatra and P Santra. Prey–predator model for optimal harvesting with functional response incorporating prey refuge. *International Journal of Biomathematics*, 9(01):1650014, 2016.
- [24] Rong Liu and Guirong Liu. Dynamics of a stochastic three species prey–predator model with intraguild predation. *Journal of Applied Analysis & Computation*, 10(1):81–103, 2020.
- [25] Dongpo Hu, Ying Zhang, Zhaowen Zheng, and Ming Liu. Dynamics of a delayed predator-prey model with constant-yield prey harvesting. *Journal of Applied Analysis & Computation*, 12(1):302–335, 2022.
- [26] Peter W Price, Carl E Bouton, Paul Gross, Bruce A McPherson, John N Thompson, and Arthur E Weis. Interactions among three trophic levels: influence of plants on interactions between insect herbivores and natural enemies. *Annual review of Ecology and Systematics*, 11:41–65, 1980.
- [27] Yang Kuang and Edoardo Beretta. Global qualitative analysis of a ratio-dependent predator–prey system. *Journal of mathematical biology*, 36(4):389–406, 1998.
- [28] Lee A Segel. *Modeling dynamic phenomena in molecular and cellular biology*. Cambridge University Press, Cambridge University, 1984.
- [29] Kevin McCann and Peter Yodzis. Biological conditions for chaos in a three-species food chain. *Ecology*, 75(2):561–564, 1994.
- [30] MA Aziz-Alaoui. Study of a leslie-gower-type tritrophic population model. *Chaos, Solitons & Fractals*, 14(8):1275–1293, 2002.
- [31] Alison B Peet, Peter A Deutsch, and Enrique Peacock-Lopez. Complex dynamics in a three-level trophic system with intraspecies interaction. *Journal of Theoretical Biology*, 232(4):491–503, 2005.

- [32] Xinhong Zhang and Xiaoling Zou. Sufficient and necessary conditions for persistence and extinction of a stochastic two-prey one-predator system. *Journal of Applied Analysis & Computation*, 12(5):1861–1884, 2022.
- [33] Roy Malcolm Anderson and Robert Mccredie May. The invasion, persistence and spread of infectious diseases within animal and plant communities. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences*, 314(1167):533–570, 1986.
- [34] Joydev Chattopadhyay and Ovide Arino. A predator-prey model with disease in the prey. *Nonlinear analysis*, 36:747–766, 1999.
- [35] Chung-Shi Tseng, Bor-Sen Chen, and Huey-Jian Uang. Fuzzy tracking control design for nonlinear dynamic systems via ts fuzzy model. *IEEE Transactions on fuzzy systems*, 9(3):381–392, 2001.
- [36] Xin Wang, Junzhi Yu, Chuandong Li, Hui Wang, Tingwen Huang, and Junjian Huang. Robust stability of stochastic fuzzy delayed neural networks with impulsive time window. *Neural Networks*, 67:84–91, 2015.
- [37] Yanni Xiao and Lansun Chen. Modeling and analysis of a predator–prey model with disease in the prey. *Mathematical biosciences*, 171(1):59–82, 2001.
- [38] Mainul Haque, Ezio Venturino, et al. Increase of the prey may decrease the healthy predator population in presence of a disease in the predator. In *Hermis*, volume 7, pages 38–59. LEA., Athens, 2006.
- [39] Mainul Haque and Ezio Venturino. An ecoepidemiological model with disease in predator: the ratio-dependent case. *Mathematical methods in the Applied Sciences*, 30(14):1791–1809, 2007.
- [40] Saswati Biswas and Arindam Mandal. Cooperation-mediated regime shifts in a disease-dominated prey–predator system. *Chaos, Solitons & Fractals*, 170:113352, 2023.
- [41] Mainul Haque, Jin Zhen, and Ezio Venturino. An ecoepidemiological predator-prey model with standard disease incidence. *Mathematical Methods in the Applied Sciences*, 32(7):875–898, 2009.
- [42] Biswajit Sarkar, Santanu Bhattacharya, and Nandadulal Bairagi. Dynamic behaviour of a single-species nonlinear fishery model with infection: the role

- of fishing tax and time-dependent market price. *Journal of Nonlinear Sciences & Applications (JNSA)*, 16(3), 2023.
- [43] E Venturino. Epidemics in predator models: disease among the prey. *Mathematical Population Dynamics: Analysis of Heterogeneity, Theory of Epidemics*, 1:381–393, 1995.
- [44] Herbert W Hethcote, Wendi Wang, Litao Han, and Zhien Ma. A predator–prey model with infected prey. *Theoretical population biology*, 66(3):259–268, 2004.
- [45] Pei Yongzhen, Li Shuping, and Li Changguo. Effect of delay on a predator–prey model with parasitic infection. *Nonlinear Dynamics*, 63(3):311–321, 2011.
- [46] Soufiane Bentout, Salih Djilali, and Abdon Atangana. Bifurcation analysis of an age-structured prey–predator model with infection developed in prey. *Mathematical Methods in the Applied Sciences*, 45(3):1189–1208, 2022.
- [47] Nazmul Sk and Samares Pal. Dynamics of an infected prey–generalist predator system with the effects of fear, refuge and harvesting: deterministic and stochastic approach. *The European Physical Journal Plus*, 137(1):138, 2022.
- [48] Crawford Stanley Holling. The functional response of predators to prey density and its role in mimicry and population regulation. *The Memoirs of the Entomological Society of Canada*, 97(S45):5–60, 1965.
- [49] Huanmeng Li and Yuan Tian. Dynamic behavior analysis of a feedback control predator-prey model with exponential fear effect and hassell-varley functional response. *Journal of the Franklin Institute*, 360(4):3479–3498, 2023.
- [50] Vito Volterra. *Variazioni e fluttuazioni del numero d'individui in specie animali conviventi*. Società anonima tipografica" Leonardo da Vinci", 1926.
- [51] Robert T Paine. Road maps of interactions or grist for theoretical development. *Ecology*, 69(6):1648–1654, 1988.
- [52] Kirk O Winemiller and Gary A Polis. Food webs: what can they tell us about the world. In Gary A Polis and Kirk O Winemiller, editors, *Food Webs*, pages 1–22. Springer, Boston, 1996.
- [53] Alan Hastings and Thomas Powell. Chaos in a three-species food chain. *Ecology*, 72(3):896–903, 1991.

- [54] Nazmul Sk, Pankaj Kumar Tiwari, and Samares Pal. A delay nonautonomous model for the impacts of fear and refuge in a three species food chain model with hunting cooperation. *Mathematics and Computers in Simulation*, 192:136–166, 2022.
- [55] Ezio Venturino. The influence of diseases on lotka-volterra systems. *The Rocky Mountain Journal of Mathematics*, 24:381–402, 1994.
- [56] Ezio Venturino. Epidemics in predator–prey models: disease in the predators. *Mathematical Medicine and Biology*, 19(3):185–205, 2002.
- [57] Ying-Hen Hsieh and Chin-Kuei Hsiao. Predator-prey model with disease infection in both populations. *Mathematical medicine and biology: a journal of the IMA*, 25(3):247–266, 2008.
- [58] Mainul Haque. Ratio-dependent predator-prey models of interacting populations. *Bulletin of mathematical biology*, 71(2):430–452, 2009.
- [59] Mainul Haque. A predator–prey model with disease in the predator species only. *Nonlinear Analysis: Real World Applications*, 11(4):2224–2236, 2010.
- [60] Mainul Haque, Sahabuddin Sarwardi, Simon Preston, and Ezio Venturino. Effect of delay in a lotka–volterra type predator–prey model with a transmissible disease in the predator species. *Mathematical Biosciences*, 234(1):47–57, 2011.
- [61] Yanni Xiao and Lansun Chen. Analysis of a three species eco-epidemiological model. *Journal of mathematical analysis and applications*, 258(2):733–754, 2001.
- [62] Xueyong Zhou, Xiangyun Shi, and Xinyu Song. Analysis of a delay prey–predator model with disease in the prey species only. *Journal of the Korean Mathematical Society*, 46(4):713–731, 2009.
- [63] Jean Jules Tewa, Valaire Yatat Djeumen, and Samuel Bowong. Predator–prey model with holling response function of type ii and sis infectious disease. *Applied Mathematical Modelling*, 37(7):4825–4841, 2013.
- [64] Peter J Hudson, Andrew P Dobson, and David Newborn. Do parasites make prey vulnerable to predation. red grouse and parasites. *Journal of animal ecology*, 61:681–692, 1992.

-
- [65] Meiling Deng and Yingbo Fan. Invariant measure of a stochastic hybrid predator–prey model with infected prey. *Applied Mathematics Letters*, 124:107670, 2022.
- [66] KP Hadeler and HI Freedman. Predator-prey populations with parasitic infection. *Journal of mathematical biology*, 27(6):609–631, 1989.
- [67] Alfred Hugo, Oluwole Daniel Makinde, Santosh Kumar, and Fred F Chibwana. Optimal control and cost effectiveness analysis for newcastle disease eco-epidemiological model in tanzania. *Journal of Biological Dynamics*, 11(1):190–209, 2017.
- [68] Bin Liu, Guanrong Chen, Kok Lay Teo, and Xinzhi Liu. Robust global exponential synchronization of general lur’e chaotic systems subject to impulsive disturbances and time delays. *Chaos, Solitons & Fractals*, 23(5):1629–1641, 2005.
- [69] Li Dong, Wang Shi-Long, Zhang Xiao-Hong, Yang Dan, and Wang Hui. Fuzzy impulsive control of permanent magnet synchronous motors. *Chinese Physics Letters*, 25(2):401, 2008.
- [70] Chun-Fu Chuang, Wen-June Wang, and Ying-Jen Chen. H- synchronization of fuzzy model based chen chaotic systems. In *2010 IEEE International Conference on Control Applications*, pages 1199–1204. IEEE, 2010.
- [71] Sanyi Tang and Lansun Chen. Density-dependent birth rate, birth pulses and their population dynamic consequences. *Journal of Mathematical Biology*, 44(2):185–199, 2002.
- [72] Sanyi Tang and Lansun Chen. Multiple attractors in stage-structured population models with birth pulses. *Bulletin of Mathematical Biology*, 65(3):479–495, 2003.
- [73] R Rakkiyappan and Pagavathigounder Balasubramaniam. On exponential stability results for fuzzy impulsive neural networks. *Fuzzy Sets and Systems*, 161(13):1823–1835, 2010.
- [74] AA Martynyuk. Stability in the models of real world phenomena. *Nonlinear Dynamics and Systems Theory*, 11(1):7–52, 2011.

- [75] Zhengrong Xiang, Guoxin Chen, et al. Stability analysis and robust control of switched stochastic systems with time-varying delay. *Journal of Applied Mathematics*, 2012, 2012.
- [76] Hui Zhang, Zhihui Ma, Gongnan Xie, Lukun Jia, et al. Effects of behavioral tactics of predators on dynamics of a predator-prey system. *Advances in Mathematical Physics*, 2014, 2014.
- [77] Yann-Horng Lin, Wen-Jer Chang, and Cheung-Chieh Ku. Solving the formation and containment control problem of nonlinear multi-boiler systems based on interval type-2 takagi-sugeno fuzzy models. *Processes*, 10(6):1216, 2022.
- [78] Chang-Woo Park, Chang-Hoon Lee, and Mignon Park. Design of an adaptive fuzzy model based controller for chaotic dynamics in lorenz systems with uncertainty. *Information Sciences*, 147(1-4):245–266, 2002.
- [79] Cheng Hu, Haijun Jiang, and Zhidong Teng. Fuzzy impulsive control and synchronization of general chaotic system. *Acta Applicandae Mathematicae*, 109:463–485, 2010.
- [80] Wu-Hua Chen, Dan Wei, and Wei Xing Zheng. Delayed impulsive control of takagi-sugeno fuzzy delay systems. *IEEE Transactions on Fuzzy Systems*, 21(3):516–526, 2012.
- [81] Xinsong Yang and Zhichun Yang. Synchronization of ts fuzzy complex dynamical networks with time-varying impulsive delays and stochastic effects. *Fuzzy Sets and Systems*, 235:25–43, 2014.
- [82] Shaocheng Tong, Wei Wang, and Lianjiang Qu. Decentralized robust control for uncertain ts fuzzy large-scale systems with time-delay. *International Journal of Innovative Computing, Information and Control*, 3(3):657–672, 2007.
- [83] Yuangan Wang, Honglin Yu, Xiaohong Zhang, and Dong Li. Stability analysis and design of time-varying nonlinear systems based on impulsive fuzzy model. *Discrete Dynamics in Nature and Society*, 2012, 2012.
- [84] Ligang Wu, Xiaojie Su, and Peng Shi. Model approximation of continuous-time ts fuzzy stochastic systems. In *Fuzzy Control Systems with Time-Delay and Stochastic Perturbation*, pages 269–286. Springer, 2015.

-
- [85] S Paul, P Bhattacharya, and Ks Chaudhuri. Stability analysis of a two species competition model with fuzzy initial conditions: Fuzzy differential equation approach environment. In *BIOMAT 2015: International Symposium on Mathematical and Computational Biology*, pages 334–344. World Scientific, 2016.
- [86] Kazuo Tanaka and Hua O Wang. *Fuzzy control systems design and analysis*. Wiley Online Library, 2001.
- [87] Swarnali Sharma and GP Samanta. Optimal harvesting of a two species competition model with imprecise biological parameters. *Nonlinear Dynamics*, 77:1101–1119, 2014.
- [88] Yasuhiro Takeuchi, Yorimasa Oshime, and Hiroyuki Matsuda. Persistence and periodic orbits of a three-competitor model with refuges. *Mathematical biosciences*, 108(1):105–125, 1992.
- [89] GP Samanta, Debasis Manna, and Alakes Maiti. Bioeconomic modelling of a three-species fishery with switching effect. *Journal of Applied Mathematics and Computing*, 12(1):219–231, 2003.
- [90] P Mishra and SN Raw. Dynamical complexities in a predator-prey system involving teams of two prey and one predator. *Journal of Applied Mathematics and computing*, 61:1–24, 2019.
- [91] Debgopal Sahoo and GP Samanta. Impact of fear effect in a two prey-one predator system with switching behaviour in predation. *Differential equations and dynamical systems*, pages 1–23, 2021.
- [92] Maria Elena Fritzsche Hoballah and Ted CJ Turlings. Experimental evidence that plants under caterpillar attack may benefit from attracting parasitoids. *Evolutionary ecology research*, 3(5):583–593, 2001.
- [93] Malay Bandyopadhyay and CG Chakrabarti. Deterministic and stochastic analysis of a nonlinear prey-predator system. *Journal of Biological Systems*, 11(02):161–172, 2003.
- [94] Alakes Maiti and GP Samanta. Deterministic and stochastic analysis of a prey-dependent predator-prey system. *International Journal of Mathematical Education in Science and Technology*, 36(1):65–83, 2005.

- [95] Yongai Zheng and Guanrong Chen. Fuzzy impulsive control of chaotic systems based on ts fuzzy model. *Chaos, Solitons & Fractals*, 39(4):2002–2011, 2009.
- [96] Hua O Wang and Kazuo Tanaka. *Fuzzy control systems design and analysis: A linear matrix inequality approach*. John Wiley & Sons, Tokyo, Japan, 2004.
- [97] Magda da Silva Peixoto, Laécio Carvalho de Barros, and Rodney Carlos Basanezi. Predator–prey fuzzy model. *Ecological Modelling*, 214(1):39–44, 2008.
- [98] Chengdai Huang. Bifurcation behaviors of a fractional-order predator–prey network with two delays. *Fractals*, 29(06):2150153, 2021.
- [99] D Didiharyono. Stability analysis of one prey two predator model with holling type iii functional response and harvesting. *Journal of Math Sciences*, 1(2 October):50–54, 2016.
- [100] Zeynep Yilmaz, Selahattin Maden, and Aytul Gokce. Dynamics and stability of two predators–one prey mathematical model with fading memory in one predator. *Mathematics and Computers in Simulation*, 202:526–539, 2022.
- [101] Yuhua Long, Lin Wang, and Jia Li. Uniform persistence and multistability in a two-predator–one-prey system with inter-specific and intra-specific competition. *Journal of Applied Mathematics and Computing*, 68(2):767–794, 2022.
- [102] D Buche, Peter Stoll, Rolf Dornberger, and Petros Koumoutsakos. Multiobjective evolutionary algorithm for the optimization of noisy combustion processes. *IEEE Transactions on Systems, Man, and Cybernetics, Part C (Applications and Reviews)*, 32(4):460–473, 2002.
- [103] Hiba Abdullah Ibrahim and Raid Kamel Naji. The impact of fear on a harvested prey–predator system with disease in a prey. *Mathematics*, 11(13):2909, 2023.
- [104] SP Bera, A Maiti, and GP Samanta. A prey-predator model with infection in both prey and predator. *Filomat*, 29(8):1753–1767, 2015.
- [105] E Beretta and V Capasso. On the general structure of epidemic systems. global asymptotic stability. *Computers & Mathematics with Applications*, 12(6):677–694, 1986.

- [106] Carlos Castillo-Chavez and Zhilan Feng. Global stability of an age-structure model for tb and its applications to optimal vaccination strategies. *Mathematical biosciences*, 151(2):135–154, 1998.
- [107] Jianquan Li, Yali Yang, and Yicang Zhou. Global stability of an epidemic model with latent stage and vaccination. *Nonlinear Analysis: Real World Applications*, 12(4):2163–2173, 2011.
- [108] Rukhsar Ikram, Amir Khan, Mostafa Zahri, Anwar Saeed, Mehmet Yavuz, and Poom Kumam. Extinction and stationary distribution of a stochastic covid-19 epidemic model with time-delay. *Computers in Biology and Medicine*, 141:105115, 2022.
- [109] HI Freedman and Paul Waltman. Persistence in models of three interacting predator-prey populations. *Mathematical biosciences*, 68(2):213–231, 1984.
- [110] Swarnali Sharma and GP Samanta. Dynamical behaviour of a two prey and one predator system. *Differential Equations and Dynamical Systems*, 22(2):125–145, 2014.
- [111] Amartya Das, Debgopal Sahoo, Guruprasad Samanta, and Juan J Nieto. Deterministic and stochastic analysis of a two-prey–one-predator system with fear effect and switching behaviour in predation. *International Journal of Dynamics and Control*, 11(3):1076–1101, 2023.
- [112] M Haque, Ezio Venturino, et al. Mathematical models of diseases spreading in symbiotic communities. 2009.
- [113] Yibo Wang, Changchun Hua, and Peng Shi. Improved admissibility criteria for takagi-sugeno fuzzy singular systems with time-varying delay. *IEEE Transactions on Fuzzy Systems*, 2023.
- [114] Min Wang, SiYing Zhang, Bing Chen, and Fei Luo. Direct adaptive neural control for stabilization of nonlinear time-delay systems. *Science China Information Sciences*, 53:800–812, 2010.