A PREY-PREDATOR MATHEMATICAL MODEL WITH DISEASE INFECTION AND TREATMENT IN BOTH PREY AND PREDATOR POPULATION

BY

Yerosan Ketema Jima

A Thesis Submitted to Department of Applied Mathematics School of Applied Natural

Science

Office of Graduate Studies

Adama Science and Technology University

July, 2020

Adama, Ethiopia

A PREY-PREDATOR MATHEMATICAL MODEL WITH DISEASE INFECTION AND TREATMENT IN BOTH PREY AND PREDATOR POPULATION

BY

Yerosan Ketema Jima

Advisor: Tamirat Temesgen (PhD)

A Thesis Submitted to Department of Applied Mathematics

School of Applied Natural Science

Office of Graduate Studies

Adama Science and Technology University

July, 2020

Adama, Ethiopia

Approval Sheet

We, the undersigned, members of the Board of Examiners of the final open defened by Yerosan Ketema have read and evaluated her thesis entitled "A Prey-predator Mathematical Model with disease infection and treatment in both prey and predator population" and examined the candidate. This is therefore to certify that the thesis has been accepted in partial fulfillment for the requirement of the degree of Master of Science in Applied Mathematics.

Name	Signiture	Date
Yerosan Ketema Name of Student		04/07/2020
TANIBAT TEMESGEN (PhD) Advisor		04/07/2020
Slifenaw Feyisa Internal Examiner		August 04/2020
External Examiner		
...ay Mitiku Damtew(PhD)	TADESSE ABDI	23/07/020 07/08/2020
...egene Desalegn Zeleke (PhD)		
School Dean ...Legesse Lemecha Obsu (Ph.D)		07/08/20
Post graduate Dean		04/07/2020
Chair Person		

Table of Contents

List of Figures

List of Tables

Abstract

We know that the infectious disease plays important roles in the dynamics of a predator prey system with infection in prey and predator population. But in our model system infection in predator plays an important role since the inclusion of disease in predator population and the transmission of disease from infected prey to predator in our model is vital modification of most of the earlier models. So, we have focused our study in observing the role of infection and transmission rate upon predator prey dynamics. The combined effect of infection and predator on prey population leads to a larger effect on the dynamics of the population sizes. In this study a prey predator system is considered. Both population is categorized into susceptible and infected due to the presence of disease and population under treatment is included into the model. Predators are assumed to consume both the susceptible and infected prey with some partial preference given to susceptible ones. Thus, a mathematical model is developed to describing the population dynamics of susceptible prey Infected prey Predator system. Positivity and boundedness of the model are verified. The equilibrium points is found and analysed. Simulation study is conducted so as to verify the results of mathematical analysis. Different simulation scenarios are presented by assigning varying values to the parameters of the system using mathlab. Lastly, conclusions of the results are forwarded.

Acknowledgements

First and foremost, I offer my humble thanks to Almighty God for giving me the opportunity, strength and guidance to undertake my study. Secondly, I would like to thank my Advisor, Tammirat Temesegen (PhD) for his valuable assistance, counselling, support, patience,and kindness during my thesis work.

My sincere thanks also go to all staffs in the Department of Applied Mathematics who contributed for the success of both my courses and thesis work, for their suggestions and help over the past two years.

Most importantly, I would like to give my special thanks to all my families whose love and support have sustained me during my studies and my relatives, for their consistence support, concern and encouragement throughout my studies.

And last but not least, all my friends and colleagues surely have provided support that I can never thank enough. I am very grateful with this life that I have been bestowed with, in which I have and know you all in my life.

Chapter 1

Introduction

1.1 Background of the Study

A predator is an organism that eats another organism. The prey is the organism which the predator eats. Some examples of predator and prey are lion and zebra, shark and fish, fox and rabbit, and bat and moth. The words "predator "and "prey "are almost always used to mean only organism that eat another organism, but the same concept also applies to plants: Bear and berry, rabbit and lettuce, grasshopper and leaf.

Both the theoretical ecology and the theoretical epidemiology are developed research fields and are treated separately. However, there are some common features between these two systems and merging the two areas may show interesting dynamics. This is a branch in mathematical biology which considers both the ecological and epidemiological issues simultaneously is called eco-epidemiology.

Mathematical biologists have been working on merging these two major areas of interest (or Ecology and Epidemiology) for a long time. Ever since the pioneering work of Lotka and Volterra, a sufficient number of prey-predator models have been introduced and studied extensively to describe the complex relationship between interacting these Species.

Mathematical modelling and analysis of multiple species ecological problems was first done by Volterra (1927) which is valid first-order approximations of more general

interaction. Since then, many mathematical models, some reviewed in this study, have been constructed based on more realistic explicit and implicit biological assumptions. Some of the aspects that need to be critically considered in a realistic and plausible mathematical model include; carrying capacity which is the maximum number of prey that the ecosystem can sustain in absence of predator, competition among prey and functional responses of the species. In the classical Lotka-Volterra model, the functional response is linear. The predator-prey model is a representation of the interaction between two species of animals that live in the same ecosystem whereby the quantity of each group of these species depends on the birth or death rate and the successful meetings with the individuals of the other species[5]

There are many factors affecting the dynamics of predator-prey models. One of the familiar factors is the functional response, referring to the change in the density of prey attached per unit time per predator as the prey density changes.

It has been observed that the inclusion of the logistic growth and Holling Type II functional response in the model, guarantees permanence. This showed the importance of incorporating logistic growth in prey-predator models. In the Holling type II functional response, the rate of prey consumption by a predator rises as prey density increases, but eventually levels off at a plateau (or asymptote) at which the rate of consumption remains constant regardless of increases in prey density. Holling (1959) studied predation of small mammals on pine sawflies, and he found that predation rates increased with increasing prey population density. This resulted from 2 effects:

(1) each predator increased its consumption rate when exposed to a higher prey density, and

(2) predator density increased with increasing prey density.

It is well known that, in nature species do not exist in exclusion. In fact, any given habitat may contain dozens or hundreds of species, sometimes thousands. Since any species has at least the potential to interact with any other species in its habitat, the

spread of a disease in a community may rapidly become astronomical as the number of infected species in the habitat increases. Therefore, it is more of a biological significance to study the effect of disease on the dynamical behavior of interacting species.[9]. Eco-epidemiology studies the direct and indirect effects that diseases have on interacting populations. Infectious diseases have been known to be an important regulating factor for human and animal population sizes. In particular, for predator-prey ecosystems, infectious diseases coupled with predator-prey interaction to produce a complex combined effect as regulators of predator and prey sizes. In many ecological studies of predator-prey systems with disease, it is reported that the predators take a disproportionately high number of parasite of infected prey.

During the last three decades, there has been growing interest in the study of infectious disease coupled with prey-predator interaction model. Some studies have even shown that parasite could change the external features or behaviour of the prey so that infected prey are more vulnerable to predation, see [2] and the references their in. Diseases that affect the prey in particular may affect the entire predator-prey system. The pathogen may not infect the predator but it creates a differential pressure on the predator-prey dynamics, causing destabilization of equilibria or reducing natural oscillations. The parasite population could affect the demographic behaviour of the host population. Eco-epidemiological models could also address scenarios, where a parasitic disease has been found to regulate the host population density. Furthermore it is shown that a disease might be the sole reason for co-existence of the species.

In ecology, predation explains a biological interaction where a predator forages on its prey. The predator-prey relationship is substantial in maintaining the equilibrium between various animal species, for the detailed see [17]. The study of prey-predator dynamics with an infected predator has a great importance in controlling the predator population. But this area has been neglected for a long time in theoretical ecology. Recently, a few researchers have cultured some prey-predator models with infection in

the predator

The focus of the thesis is that in the realm of biological mathematics, it is possible to mathematically represent the population variations of a predation relationship to a certain extent of accuracy. In this study we will try to formulate a predator-prey systems with infection in both populations to examine the behaviour of the two species under a few assumptions. In order to illustrate some of the analytical results of the study, numerical simulations of the model will be carried out.

1.2 Statement of the problem

Ecological populations suffer from various infectious diseases and these diseases have a significant role in regulating population size. Thus, it is worth while to study the combined effect of epidemiological and demographic features on the real ecological populations[2]. The dynamic relationships between species and their complex properties are at the heart of many ecological and biological processes. One of such relationships is the dynamical relationship between a predator and their prey which has long been and will continue to be one of the dominant themes in both ecology and mathematical ecology due to its universal existence and importance [4].

These problems may appear to be simple mathematically at first sight, they are, in fact, often very challenging and complicated. Mathematical study of such eco-epidemiological model has explored various unknown aspects of ecological population. However, in ecosystem, the interaction between the predator and prey is a non-linear and complex process. This complexity has attracted the attention of both theoretical and mathematical ecologists to have extensive investigation concerning the interaction which calls for the development of mathematical models that are essential tools in understanding the interaction mechanisms for persistence or extinction of species in natural systems.

This study is intended to answer the following basic questions:

- How we formulate a mathematical model that describes the correlation between the disease and the predator-prey system?
- What is the role of ecological interactions such as competition, predation and parasite infection (in the prey and predator populations) in regulating population sizes?
- What is the biological implication of the solution of the developed mathematical model?

1.3 The Objective of the study

This study aims at achieving the following major and specific objectives.

1.3.1 General objective

The general objective of this study is to investigate the correlation between the disease and the predator-prey system.

1.3.2 Specific Objective

The specific objectives of this study are to:

- formulate a mathematical model that describes the correlation between the disease and the predator-prey system,
- investigate Stability of the equilibrium points of the model and carry out Computer simulations to illustrate our analytical findings, and
- interpret the biological implications of analytical and numerical findings of the developed mathematical model.

1.4 Significant and Beneficiaries

Mathematical models have become important tools in analyzing the dynamical relationship between predator and their prey. So this study used to study the dynamics

of prey predator system. Species interaction in natural and wildlife environments is unavoidable. Different species occupying similar ecosystems and living amongst each other will regularly interact. These leads to the question, how does this primal concern on survival impact the way in which species interact? this question may be answered by this study.

Eco-epidemic models describe ecosystems of interacting populations among which a disease spreads. It has been established that invading diseases tend to destabilize the predator-prey communities. However, it is showed that the predator infection can also have a stabilizing effect. Most of the existing models in eco-epidemiology consider a disease in prey population.

On the other hand, over the last few decades, mathematics has been used to understand and predict the spread of disease relating important public-health questions to basic transmission parameters.

1.5 Expected Outcome

The expected outcomes of the proposed study are:

- A mathematical model that might provide a detailed description of the correlation between the disease and the predator-prey system, and may also help us in the understanding of the interaction mechanisms for persistence or extinction of species in natural systems.

- mathematical model that describe interactions between infected and susceptible predator-prey population, disease and it's treatment.

- Numerical solution of the developed mathematical model, from which the biological implication of the solution of the model interpreted.

Chapter 2

Literature Review

Mathematical biologists have been working on merging two major areas of interest Ecology and Epidemiology for a long time . A number of attempts have been made to predict the evolution and existence of species mathematically, particularly the interaction between prey and predator [17]. Mathematical study of such populations has attracted attentions of both ecologists and mathematicians from several years past. As a result numerous mathematical models have been developed, and these models have become essential tools in analyzing the interaction of different populations.

Alfred *et al.* [1] proposed a mathematical model and analyzed it to study the functional response of the predator toward a susceptible as well as infected prey. Their model consists of three populations: the prey population density, the predator population density, and population of infected prey and predator under treatment

By the developed mathematical model ,they examined an eco-epidemiological mathematical model with treatment and disease infection in both prey and predator population. A system of differential equations for the problem is proposed and analyzed qualitatively using the stability theory of the differential equations. A local and global study of the model is performed around the disease-free equilibrium and the endemic equilibrium to estimate the effect of incorporated parameters that control disease eradication and species coexistence. Numerical simulations are carried out to justify analytical results. The model with and without infected predator together with

treatment classes are finally compared.

Their paper investigates the dynamical behaviour of an eco-epidemiological model. The model integrates treatment and disease infection in both prey and predator populations. Incorporating treatment in the model provides a more realistic and plays an important role for biological control of disease, and increasing the rate of treatment can increase prey and predator density population. The boundedness and positivity of the system hold which implies that the system is biologically valid and well behaved. Disease free equilibrium points were obtained and their stability analysis investigated. The analysis on interior equilibrium indicates that under a complicated set of conditions, it is possible to have multiple interior equilibria numerically. The model analysis shows that treatment of infected populations has the effect of reducing the spread of the disease. It is observed that incorporating of treatment of infected prey and predator into the system may save the population from extinction.

A numerical study of the model was carried out and it was observed that the increase of the number of infected prey tends to lower the whole population. It could be concluded that the disease can be eradicated in a population through treatment.

S.A. Wuhaib and Y. Abu Hasan [3] developed predator-prey model, where the prey followed the susceptible-infected-susceptible cycle. Within this cycle, the infected prey is harvested and the predators consumed the susceptible prey only. There are many documented cases on this, an example is the relationship between aquatic snails and fishes. The model includes the harvesting of infected prey. They assumed that the predator avoids the infected prey. The susceptible prey becomes infected when they are in contact with infected prey and recover to be susceptible again. In order to maintain a healthy population, the infected prey was harvested. They discussed the effect of effort of harvest on the disease. First, they fixed all parameters to ensure all populations survive. Then they find the effect of harvest on the disease. Conditions for stability of the equilibrium points for the two populations were obtained. They obtained also were

the region of the solutions, where the solutions are bounded. It is also observed that the increase in harvest affects the disease and thus prevents the occurrence of an epidemic. They found the equilibrium points and the conditions for their existence and stability. they also showed the non-existence of periodic solutions. Numerical simulations explained the effect of the parameters on the behaviour of the three classes of populations. The simulations also gave the region of the solution and guarantees that all solutions of the system lie within the region. they analyzed stability of the equilibrium points. Next, they discussed the nature of the solutions and finally the numerical simulations to support the model.

S. P. Bera, A. Maiti and G. P. Samanta[2] have formulated a prey-predator model with disease in both the populations. The model they introduced consists of two populations: the prey population and the predator population. Both the populations have two sub classes: susceptible and infected.

By this model the authors studied the dynamical behaviours of a prey-predator system where both prey and predator populations are affected by diseases. A system of four differential equation has been proposed and analyzed. Stability of the equilibrium points of the model has been investigated. Computer simulations are carried out to illustrate their analytical findings. The biological implications of analytical and numerical findings are discussed.

In this study , the role of ecological interactions such as competition, predation etc., are well recognized. On the other hand, parasite infection in the prey and predator populations is shown to be an issue which cannot be ignored, and also it has an important role in regulating population sizes. It is observed that parasites affect the internal mechanism of the hosts. Parasites weaken the survival and fecundity of infected host. Sometimes parasites may also influence aspects of their hosts 'sexual life cycle. For example, they may reduce the hosts' likelihood of finding mates or may increase or decrease the frequency with which a female produces ephippia and male offspring. Many

parasites are able to redirect the metabolic pathways of the host to favor their own growth. Thus, reality dictates that, we should be concerned with prey-predator systems, where both the populations are disease affected.

Sachin Kumar and Harsha Khrbanda [11] formulated and analyzed four dimensional ecoepidemiological model consisting of susceptible prey, infected prey, vaccinated prey and predator. They have considered this model to study the influence of disease, migration and vaccination on an environment where two or more interacting species are present. In their work the functional response was assumed to be of Lotka-Volterra type. they studied systematically the behaviour of the model with and without disease in prey. They analyzed mathematically the dynamics of the system such as boundedness of the solutions, existence and stability conditions of equilibria.

Numerical simulations are also carried out for the analytical results. It is observed that the number of infected prey population decreases when we consider migration in our model. This also implies the increment in the population of healthy prey, it simply means that the infection is reducing within the population. Similarly, it has been observed that the number of vaccinated prey increases with the effect of migration in main model. The dependency of equilibria on migration is described. They have seen that the conditions of existence and stability of equilibrium points depend on the parameters. Thus, the estimation of parameters is an important phase for numerical simulations of a mathematical model.

Most of the earlier studies [1][2][3][11] pay attention to the disease transmission in prey population only. To the best of our knowledge, the influence of disease in predator on epidemics and treatment and also the transmission of disease from prey to predator while feeding has not yet been studied together considerably. In the proposed study we tried to merge the models and employ numerical method which filled this gap.

Chapter 3

Methodology of the Study

3.1 Study Design

The aim of this study is to formulate a mathematical model using a system of ordinary differential equation which describes the interaction between prey-predator system as well as disease that affect them and may help us for understanding the interaction mechanisms of persistence or extinction of these species in natural systems. In order to investigate the effect of the disease and treatment, on the process we will be consider it together in the model equation. After we non-dimensionalized the formulated mathematical model, the boundedness and positivity of the solutions of the model are established, all the possible equilibrium points of the model and their feasibility conditions will be discussed. Linear stability analysis will be carried out to see the dynamical behaviour of the system under consideration. Numerical simulations will be done by using MATLAB to illustrate our analytical findings. Finally, biological significance of our analytical finding is discussed from the solution of system of the differential equation with its graph.

3.2 Procedures of the Study

To attain the objective of the study, the following procedures will be undertaken:

- Defining the problem,

- Collecting information,
- Formulating the model by adding some assumption on the existing model,
- Qualitative analysis of the model,
- Solving the model equation by employing numerical methods,
- discussing the results with its graph.

Chapter 4

Model Formulation and Analysis

4.1 Model Formulation

Eco-epidemiology studies the direct and indirect effects that diseases have on interacting populations. Infectious diseases have been known to be an important regulating factor for human and animal population sizes . In particular for predator prey ecosystems, infectious diseases coupled with predator prey interaction to produce a complex combined effect as regulators of predator and prey sizes. In many ecological studies of predatorprey systems with disease, it is reported that the predators take a disproportionately high number of parasite of infected prey [11].

In the present paper, we study an eco-epidemiological mathematical model with treatment and disease infection in both Prey and Predator population. This is an extension of the study of the eco-epidemiological model which was studied by [1] by incorporating infected predator group together and treatment. A mathematical model is proposed and analyzed to study the functional response of the predator toward a susceptible as well as infected prey. The model consists of three populations:

- The prey whose total population density is denoted by P, the predator whose total population density is denoted by Q and population of infected prey and predator under treatment is denoted by T.

- in the presence of disease, total prey population is divided into two classes (i) susceptible class (W) and (ii) infected class (X). And also the total predator population is divided into two classes (i) susceptible predator class (Y) and (ii) infected predator class (Z). So, at time t total prey population is $P(t) = W(t) + X(t)$, total predator population is $Q(t) = Y(t) + Z(t)$ and population under treatment is $T(t)$.

  In our model we assume that diseases are transmitted vertically in both populations. That is disease transmits from infected prey (X) to susceptible prey (W) and from infected predator (Z) to susceptible predator (Y). Furthermore we assume that Disease can spread from prey to predator by feeding on infected prey species only and this assumption is not considered in other comparable model.

- We assume that both the susceptible and the infected prey are capable of reproducing in logistic growth with carrying capacity $K(K > 0)$, intrinsic birth rate $r_1 > 0$ and $r_2 > 0$ respectively. so the equation governing the situation is:

$$\frac{dW}{dt} = r_1 W\left(1 - \frac{W}{K}\right)$$

$$\frac{dX}{dt} = r_2 X\left(1 - \frac{W + X}{K}\right)$$

  Since here we assume that W feels only population pressure of W where as X feels the population pressure of both populations W and X because it is assumed that infected can not exert population pressure on W due to less weak and inactive.

In addition, in the formulation of the proposed model, the following assumptions are taken into consideration:

- It is assumed that the disease spreads among the prey population and can be transmitted to predator population during the predation leading $Z(t)$ predator population at a rate $\beta_3 XY$. Moreover, the disease is genetically inherited. The infected population can only recover through treatment.

- Susceptible prey becomes infected when it comes in contact with the infected prey and this contact process is assumed to follow the simple mass action $\beta_1 WX$, where β_1 is the rate of conversion. Further, susceptible prey can be eaten by both predator at a rates $b_1 WY$ and $b_2 WZ$ respectively. but healthy predator can caught easily.

- The healthy predators hunts with possibly different predation rate a_1 and β_3 on susceptible and infected preys respectively with $\beta_3 > a_1$. We also assume that The infected predators hunts with possibly different predation rate a_2 and a_3 on susceptible and infected preys respectively with $a_3 > a_2$. This is in accordance with the fact that the infected individuals can be caught more easily. That is infected prey become less active and therefore they could get caught easily by the predator compared to healthy prey. Thus, we assume that searching coefficient of the predator for infected prey is greater than that of healthy prey. Moreover, Predators get the same reward out of predating on healthy and infected prey with different search efficiencies denoted by a_1 , a_2, β_3 and a_3 respectively.

- It is assumed that coefficients of conversing of healthy prey to healthy predator is b_1 , healthy prey to infected predator is b_2, infected prey to infected predator is β_3.Furthermore the consumption of infected prey by healthy predator results change in infected predator that represented by crowded term $\beta_3 XY$ in our model. Where β_3 the transmission rate of disease from infected prey to healthy predator.

- The infected prey and predator are respectively, treated at the rates e_1 and e_2, removed and can recover to susceptible with the coefficient rate λ_1 and λ_2 respectively,while δ_1 , δ_2 and δ_3 are respectively the death of infected prey, infected predator and population under treatment.

- It is assumed that all the five species may have different natural death rates d_1 , d_2 , d_3 , d_4 and d_5 .

Taking into account the aforementioned considerations, we then have the schematic flow diagram shown in figure 4.1.

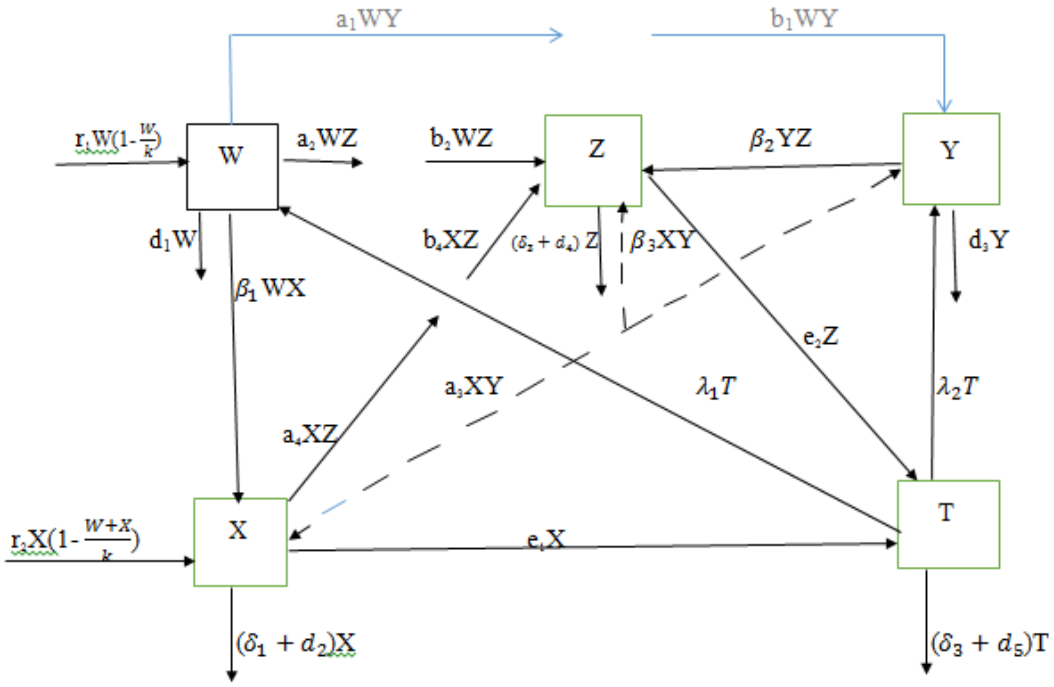

Figure 4.1: Model flowchart

.

From the flow chart(figure 4.1),we motivated to form the following set of five non-linear ordinary differential equations(ODE):

$$\frac{dW}{dt} = \underbrace{r_1 W\left(1-\frac{W}{K}\right)}_{\textbf{Growth}} - \underbrace{\beta_1 WX}_{Infection} - \underbrace{a_1 WY - a_2 WZ}_{Predation} + \underbrace{\lambda_1 T}_{Treated} - \underbrace{d_1 W}_{Mortality} \tag{4.1.1}$$

$$\frac{dX}{dt} = \underbrace{r_2 X\left(1-\frac{W+X}{K}\right)}_{\textbf{Growth}} + \underbrace{\beta_1 WX}_{Infection} - \underbrace{\beta_3 XY - a_3 XZ}_{Predation} - \underbrace{e_1 X}_{Treatment} - \underbrace{(\delta_1 + d_2)X}_{Mortality} \tag{4.1.2}$$

$$\frac{dY}{dt} = \underbrace{b_1 WY}_{Preyconsumption} - \underbrace{\beta_2 YZ}_{Infection} + \underbrace{\lambda_2 T}_{Treated} - \underbrace{d_3 Y}_{Mortality} \tag{4.1.3}$$

$$\frac{dZ}{dt} = \underbrace{b_2 WZ + \beta_3 XY + b_3 XZ}_{Preyconsumption} + \underbrace{\beta_2 YZ}_{Infection} - \underbrace{e_2 Z}_{Treatment} - \underbrace{(\delta_2 + d_4)Z}_{Mortality} \tag{4.1.4}$$

$$\frac{dT}{dt} = \underbrace{e_1X + e_2Z}_{Treatment} - \underbrace{(\lambda_1 + \lambda_2)T}_{Treated} - \underbrace{(\delta_3 + d_5)T}_{Mortality} \tag{4.1.5}$$

Consumed prey is converted into predator with efficiency ϵ ($0 < \epsilon < 1$) that is $b_1 = \epsilon a_1$, $b_2 = \epsilon a_2$, and $b_3 = \epsilon a_3$. For a biological meaning, we further assume that $e_1 < \lambda_1$ and $e_2 < \lambda_2$. Furthermore, we assume that all the constant coefficients are positive. The initial condition of equation (4.1.1-4.1.5) are given as $W(0) = W_0 > 0$, $X(0) = X_0 \geq 0$, $Y(0) = Y_0 > 0$, $Z(0) = Z_0 \geq 0$ and $T(0) = T_0 \geq 0$.

4.1.1 Model Without Disease

In this section, model is transformed with the assumption that there does not occur any infection within the population. Therefore, the model equation 4.1.1-4.1.5 is reduced into two dimensional prey-predator model 4.1.6-4.1.7 so it takes the form:

$$\frac{dW}{dt} = \underbrace{r_1W\left(1 - \frac{W}{K}\right)}_{\textbf{growth}} - \underbrace{a_1WY}_{Predation} - \underbrace{d_1W}_{mortality} \tag{4.1.6}$$

$$\frac{dY}{dt} = \underbrace{b_1WY}_{preyconsumption} - \underbrace{d_3Y}_{Mortality} \tag{4.1.7}$$

and has to be analyzed with the following initial conditions : $W(0) > 0$ and $Y(0) > 0$.

4.1.2 Model Without Treatment

Under this, the model is transformed With the assumption that there does not treatment for the population and the model equation 4.1.1-4.1.5 is reduced into four dimensional prey-predator model having the following form.

$$\frac{dW}{dt} = \underbrace{rW\left(1 - \frac{W}{K}\right)}_{\textbf{growth}} - \underbrace{\beta_1WX}_{infection} - \underbrace{a_1WY - a_2WZ}_{Predation} - \underbrace{d_1W}_{mortality} \tag{4.1.8}$$

$$\frac{dX}{dt} = \underbrace{r_2X\left(1 - \frac{W + X}{K}\right)}_{\textbf{growth}} + \underbrace{\beta_1WX}_{infection} - \underbrace{\beta_3XY - a_3XZ}_{Predation} - \underbrace{(\delta_1 + d_2)X}_{Mortality} \tag{4.1.9}$$

$$\frac{dY}{dt} = \underbrace{b_1WY}_{preyconsumption} - \underbrace{\beta_2YZ}_{Infection} - \underbrace{d_3Y}_{Mortality} \tag{4.1.10}$$

$$\frac{dZ}{dt} = \underbrace{b_2WZ + \beta_3XY + b_3XZ}_{preyconsumption} + \underbrace{\beta_2YZ}_{Infection} - \underbrace{(\delta_2 + d_4)Z}_{Mortality} \tag{4.1.11}$$

4.2 Analysis of Model System

4.2.1 Non dimensionalization

The model we have just specified has thirteen parameters, which makes analysis difficult. To reduce the number of parameters and to determine which combinations of parameters control the behaviour of the system, we non-dimensionalize the system (4.1.1-4.1.5) with the following scaling

$$\tilde{t} = r_1 t, \tilde{W} = \frac{W}{K}, \tilde{X} = \frac{X}{K}, \tilde{Y} = \frac{Y}{K}, \tilde{Z} = \frac{Z}{K}, \tilde{T} = \frac{T}{K}. \tag{4.2.1}$$

as well as the full set of non-dimensional parameters are given by table4.2.1 .

Table 4.1: Set of non-dimensional Parameter from which the non dimensional variables are obtained

$\alpha_{12} = \frac{\beta_1 K}{r_1}$,	$\alpha_{13} = \frac{a_1 K}{r_1}$,	$\alpha_{14} = \frac{a_2 K}{r_1}$,	$\alpha_{15} = \frac{\lambda_1 K}{r_1}$,	$\alpha_{16} = \frac{d_1 K}{r_1}$,
$r = \frac{r_2}{r_1}$,	$\alpha_{23} = \frac{\beta_3 K}{r_1}$,	$\alpha_{24} = \frac{a_3 K}{r_1}$,	$\alpha_{25} = \frac{e_1+\delta_1+d_2}{r_1}$,	$\alpha_{31} = \frac{b_1 K}{r_1}$,
$\alpha_{32} = \frac{\beta_2 K}{r_1}$,	$\alpha_{33} = \frac{\lambda_2}{r_1}$,	$\alpha_{34} = \frac{d_3}{r_1}$,	$\alpha_{41} = \frac{b_2 K}{r_1}$,	$\alpha_{43} = \frac{b_3 K}{r_1}$,
$\alpha_{45} = \frac{e_2+\delta_2+d_4}{r_1}$,	$\alpha_{51} = \frac{e_1}{r_1}$,	$\alpha_{52} = \frac{e_2}{r_1}$,	$\alpha_{53} = \frac{\lambda_1+\lambda_2+\delta_3+d_5}{r_1}$,	

Inserting the non-dimensional variables (4.2.1) and parameters (given in table 4.2.1) above into the system (4.1.1) -(4.1.5) and by omitting the tildes for notational simplicity, we obtain the non-dimensionalised form of the system of ODEs given by (4.2.2)-(4.2.6):

$$\frac{dW}{dt} = W(1 - W) - \alpha_{12}WX - \alpha_{13}WY - \alpha_{14}WZ + \alpha_{15}T - \alpha_{16}W \tag{4.2.2}$$

$$\frac{dX}{dt} = rX(1 - W - X) + \alpha_{12}WX - \alpha_{23}XY - \alpha_{24}XZ - \alpha_{25}X \tag{4.2.3}$$

$$\frac{dY}{dt} = \alpha_{31}WY - \alpha_{32}YZ + \alpha_{33}T - \alpha_{34}Y \tag{4.2.4}$$

$$\frac{dZ}{dt} = \alpha_{41}WZ + \alpha_{23}XY + \alpha_{43}XZ + \alpha_{32}YZ - \alpha_{45}Z \tag{4.2.5}$$

$$\frac{dT}{dt} = \alpha_{51}X + \alpha_{52}Z - \alpha_{53}T \tag{4.2.6}$$

4.2.2 Boundedness

All the solutions of the system4.1.1-4.1.5 which start in $\mathbf{R^{5+}}$ are bounded.

4.2.3 Equilibrium Points, Feasibility Conditions and Stability Analysis

In this section,to study the dynamical behaviour of the system, the existence and local stability analysis of all possible equilibrium points of system equations4.2.2-4.2.6 are discussed and the following results are obtained:

Equilibrium Points Feasibility Conditions

Now getting the equilibrium points is the first step and the fixed points are obtained as the solutions of $\frac{dW}{dt} = \frac{dX}{dt} = \frac{dY}{dt} = \frac{dZ}{dt} = \frac{dT}{dt} = 0$. Using this and the model equations4.2.2-4.2.6 system may have the following equilibrium points:

1. The trivial equilibrium point $E_0(0, 0, 0, 0, 0)$; This equilibrium always exists unconditionally.

2. The axial equilibrium point $E_1(w', 0, 0, 0, 0)$; This disease and predator free equilibrium exists when $\alpha_{16} > 1$. When this condition is satisfied $w' = \alpha_{16} - 1$.In terms of original parameters of the system, this condition $\alpha_{16} > 1$ indicates that $\frac{d_1 k}{r_1} > 1$. That is healthy population do not have death rate greater than the production rate of infected prey, then the predator and disease-free equilibrium exists.

3. The disease free equilibrium point $E_2(w', 0, y', 0, 0)$; This equilibrium exists when $\alpha_{31} > (\alpha_{34}+\alpha_{31}\alpha_{16})$. When this condition is satised, $w' = \frac{\alpha_{34}}{\alpha_{31}}$ and $y' = \frac{\alpha_{31}(1-\alpha_{16})-\alpha_{34}}{\alpha_{31}\alpha_{13}}$.

In terms of original parameters of the system, this condition $(\alpha_{34} + \alpha_{31}\alpha_{16}) < \alpha_{31}$ indicates that $d_3 + b_1 d_1 k < b_1 k$. That is, if the predator is high capacity consumer, then the disease-free equilibrium exists.

4. The predator-free equilibrium point $E_3(w', x', 0, 0, t')$; This equilibrium point exist when

$$t' = \frac{\alpha_{51}}{\alpha_{53}} x',$$
$$w' = \frac{rx' + \alpha_{25} - r}{\alpha_{12-r}}$$

and x' is a positive root of

$$\frac{\alpha_{53}[\alpha_{25}(\alpha_{16} - 1) - r - (\alpha_{25} - r)^2] + r\alpha_{53}(2\alpha_{25} + \alpha_{16} - 1)x' - r\alpha_{53}x'^2}{(\alpha_{25} - r)(\alpha_{15}\alpha_{51} - \alpha_{12}\alpha_{53}) - r\alpha_{12}\alpha_{53}x'} = 0$$

are all positive.

5. The interior equilibrium point $E_4(w', x', y', z', t')$; This equilibrium point of co-existence exist when

$$t' = \frac{\alpha_{51}x' + \alpha_{52}z'}{\alpha_{53}},$$
$$z' = \frac{\alpha_{53}\alpha_{33}}{\alpha_{52}\alpha_{33} + \alpha_{53}\alpha_{32}}(\frac{\alpha_{31}}{\alpha_{33}}w' - \frac{\alpha_{51}}{\alpha_{53}}x' - \alpha_{34}),$$
$$y' = \frac{-\alpha_{33}\alpha_{51}x'}{\alpha_{53}\alpha_{31}w' - \alpha_{34}} + \alpha_{34},$$
$$x' = \frac{1}{\alpha_{12}}(1 - w' - \alpha_{13}y' - \alpha_{14}z' - \alpha_{16})$$

and

$$w' = (\frac{\alpha_{32}}{\alpha_{31}} + \frac{\alpha_{52}\alpha_{32}}{\alpha_{31}\alpha_{53}})z' - \frac{\alpha_{33}\alpha_{51}x'}{\alpha_{31}\alpha_{53}y'} + \frac{\alpha_{34}}{\alpha_{31}}$$

are all positive.

Stability Analysis

Theorem of stability: (4.1.1): Assume A is an nxn constant matrix.

(i) If A has an eigenvalue with positive real part, then the trivial solution is unstable on $[0; \infty)$.

(ii) If all the eigenvalues of A with zero real parts are simple (multiplicity one) and all other eigenvalues of A have negative real parts, then the trivial solution is stable on$[0;\infty)$.

(iii) If all the eigenvalues of A have negative real parts, then the trivial solution of $x = Ax$ is globally asymptotically stable on $[0;\infty)$.

The variational matrix V(w; x; y; z,t) of system (4.2.2)-(4.2.6) at any point (w; x; y;z,t) is

$$J(F) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22} & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44} & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55} \end{bmatrix}$$

where

$a_{11} = 1 - 2w - \alpha_{12}x - \alpha_{13}y - \alpha_{14}z - \alpha_{16}$ $\quad$ $a_{12} = -\alpha_{12}w$

$a_{13} = -\alpha_{13}w$ $\quad$ $a_{14} = -\alpha_{14}w$

$a_{15} = \alpha_{15}$ $\quad$ $a_{21} = (\alpha_{12} - r)x$

$a_{22} = r - \alpha_{25} - 2rx + (\alpha_{12} - r)w - \alpha_{23}y$ $\quad$ $a_{23} = -\alpha_{23}x$

$a_{24} = -\alpha_{24}x$ $\quad$ $a_{25} = 0$

$a_{31} = \alpha_{31}y$ $\quad$ $a_{32} = 0$

$a_{33} = \alpha_{31}w - \alpha_{32}z - \alpha_{34}$ $\quad$ $a_{34} = \alpha_{32}y$

$a_{35} = \alpha_{33}$ $\quad$ $a_{41} = \alpha_{41}z$

$a_{42} = \alpha_{23}y + \alpha_{43}z$ $\quad$ $a_{43} = \alpha_{23}x + \alpha_{32}z$

$a_{44} = \alpha_{41}w + \alpha_{43}x + \alpha_{32}y - \alpha_{45}$ $\quad$ $a_{45} = 0$

$a_{51} = 0$ $\quad$ $a_{52} = \alpha_{51}$

$a_{53} = 0$ $\quad$ $a_{54} = \alpha_{52}$

$a_{55} = \alpha_{53}$

At the equilibrium point $E_0(0,0,0,0,0)$; the variational matrix becomes

$$V(E_0) = \begin{bmatrix} 1-\alpha_{16} & 0 & 0 & 0 & \alpha_{15} \\ 0 & r-\alpha_{25} & 0 & 0 & 0 \\ 0 & 0 & -\alpha_{34} & 0 & \alpha_{33} \\ 0 & 0 & 0 & -\alpha_{45} & 0 \\ 0 & \alpha_{51} & 0 & \alpha_{52} & \alpha_{53} \end{bmatrix}$$

The corresponding eigenvalues are $1-\alpha_{16}$ (which is negative since $\alpha_{16} > 1$ for the existence of this equilibrium point) ,$r-\alpha_{25}$, $-\alpha_{34}$, $-\alpha_{45}$ and α_{53} hence the equilibrium point E_0 is unstable.

At the equilibrium point $E_1(\alpha_{16}-1,0,0,0,0)$; the variational matrix becomes

$$V(E_1) = \begin{bmatrix} -1 & -\alpha_{12} & -\alpha_{13} & -\alpha_{14} & \alpha_{15} \\ 0 & \alpha_{22}-\alpha_{25} & 0 & 0 & 0 \\ 0 & 0 & \alpha_{31} & 0 & 0 \\ 0 & 0 & 0 & \alpha_{41}-\alpha_{45} & 0 \\ 0 & \alpha_{51} & 0 & \alpha_{52} & \alpha_{53} \end{bmatrix}$$

The corresponding eigenvalues are -1 , $\alpha_{22}-\alpha_{25}$, α_{31} , $\alpha_{41}-\alpha_{45}$ and α_{53} hence we have that the axial equilibrium point E_1 is also unstable.

At the disease free equilibrium point $E_2(\frac{\alpha_{34}}{\alpha_{31}}, 0, \frac{\alpha_{31}(1-\alpha_{16})-\alpha_{34}}{\alpha_{13}\alpha_{31}}, 0, 0)$; the variational matrix becomes

$$V(E_2) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22} & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44} & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55} \end{bmatrix}$$

where

$$a_{11} = 1 - \alpha_{16} - 2\frac{\alpha_{34}}{\alpha_{31}} - \frac{\alpha_{31}(1-\alpha_{16}) - \alpha_{34}}{\alpha_{31}} \qquad a_{12} = -\frac{\alpha_{12}\alpha_{34}}{\alpha_{31}}$$

$$a_{13} = -\frac{\alpha_{13}\alpha_{34}}{\alpha_{31}} \qquad a_{14} = \frac{\alpha_{14}\alpha_{34}}{\alpha_{31}}$$

$$a_{15} = \alpha_{15} \qquad a_{21} = 0$$

$$a_{22} = \frac{(\alpha_{12}-r)\alpha_{34}}{\alpha_{31}} - \frac{\alpha_{23}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}} + r - \alpha_{25} \qquad a_{23} = 0$$

$$a_{24} = 0 \qquad a_{25} = 0$$

$$a_{31} = \frac{\alpha_{31}(1-\alpha_{16}) - \alpha_{34}}{\alpha_{13}} \qquad a_{32} = 0$$

$$a_{33} = \alpha_{31} \qquad a_{34} = \frac{\alpha_{32}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}}$$

$$a_{35} = \alpha_{33} \qquad a_{41} = 0$$

$$a_{42} = \frac{\alpha_{23}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13}} \qquad a_{43} = 0$$

$$a_{44} = \frac{\alpha_{41}\alpha_{34}}{\alpha_{31}} + \frac{\alpha_{32}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}} \qquad a_{45} = 0$$

$$a_{51} = 0 \qquad a_{52} = \alpha_{51}$$

$$a_{53} = 0 \qquad a_{54} = \alpha_{52}$$

$$a_{55} = \alpha_{53}$$

The corresponding eigenvalues are λ_1 , λ_2 , λ_3 , λ_4 and λ_5 where

$$\lambda_1 = 1 - \alpha_{16} - 2\frac{\alpha_{34}}{\alpha_{31}} - \frac{\alpha_{31}(1-\alpha_{16}) - \alpha_{34}}{\alpha_{31}}, \quad \lambda_2 = \frac{(\alpha_{12}-r)\alpha_{34}}{\alpha_{31}} - \frac{\alpha_{23}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}} + r - \alpha_{25},$$

$$\lambda_3 = \alpha_{31}, \qquad \lambda_4 = \frac{\alpha_{41}\alpha_{34}}{\alpha_{31}} + \frac{\alpha_{32}(\alpha_{31}(1-\alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}}$$

$$\lambda_5 = \alpha_{53}.$$

hence the equilibrium point E_2 is unstable.

At the equilibrium point E_3; the variational matrix becomes

$$V(E_3) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22} & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44} & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55} \end{bmatrix}$$

where

$$a_{11} = 1 - \alpha_{16} - \frac{\alpha_{23}\alpha_{31} + \alpha_{25}\alpha_{32}}{\alpha_{53}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}} + \frac{\alpha_{33}\alpha_{51}}{\alpha_{31}\alpha_{53}} \qquad a_{12} = -\alpha_{12}\frac{\alpha_{12}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}{\alpha_{31}\alpha_{34} + \alpha_{25}\alpha_{13}}$$

$$a_{13} = -\alpha_{13}(\frac{\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}{\alpha_{31}\alpha_{15} + \alpha_{31}\alpha_{53}} + \frac{\alpha_{52}\alpha_{32}}{\alpha_{31}\alpha_{53}}) \qquad a_{14} = -\alpha_{14}(\frac{\alpha_{32} + \alpha_{31}\alpha_{53}}{\alpha_{31} + \alpha_{34}\alpha_{52} + \frac{r-\alpha_{25}}{\alpha_{25}\alpha_{13}}})$$

$$a_{15} = \alpha_{15} \qquad a_{21} = \frac{\alpha_{51}\alpha_{32} + \alpha_{31}\alpha_{33}}{\alpha_{34}\alpha_{42} - r\alpha_{42}} + \frac{1}{\alpha_{13}\alpha_{53}}(\alpha_{33}\alpha_{34} + \alpha_{12})$$

$$a_{22} = \frac{\alpha_{41}\alpha_{34} + \alpha_{15}}{\alpha_{31}\alpha_{25} - r\alpha_{32}} - \frac{\alpha_{32}\alpha_{41} - r\alpha_{23}\alpha_{53}}{\alpha_{13}\alpha_{31}} \qquad a_{23} = \frac{r\alpha_{31}\alpha_{51} + \alpha_{23}\alpha_{41}}{r\alpha_{52}\alpha_{53} - r\alpha_{41}} + \frac{\alpha_{23}\alpha_{42} + \alpha_{52}}{\alpha_{41}\alpha_{31}}$$

$$a_{24} = \frac{\alpha_{52} + \alpha_{14}\alpha_{25}}{\alpha_{34}\alpha_{53} - \alpha_{34}} + \frac{\alpha_{31}(\alpha_{23}\alpha_{14} - \alpha_{16}) - \alpha_{23}\alpha_{34}}{\alpha_{23}\alpha_{23} - \alpha_{32}\alpha_{42}} \qquad a_{25} = 0$$

$$a_{31} = \frac{\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}{\alpha_{23}\alpha_{43}} + \frac{\alpha_{31}(1 - \alpha_{16}) - \alpha_{34}}{\alpha_{13}} \qquad a_{32} = 0$$

$$a_{33} = \frac{\alpha_{24}\alpha_{22} - \alpha_{25}\alpha_{15}}{\alpha_{53}\alpha_{33} - r\alpha_{32}} + \frac{1 - r - \alpha_{25}}{\alpha_{25}\alpha_{13}} \qquad a_{34} = \frac{\alpha_{32}\alpha_{23} + r\alpha_{34}\alpha_{33}\alpha_{25} + \alpha_{13}\alpha_{16}}{\alpha_{31} + r\alpha_{15}} + \frac{1 - r\alpha_{15}}{\alpha_{51}\alpha_{32}}$$

$$a_{35} = \alpha_{33} \qquad a_{41} = 0$$

$$a_{42} = \frac{\alpha_{31}\alpha_{16} - \alpha_{34}}{\alpha_{41}\alpha_{13} + \alpha_{52}\alpha_{16}} + \frac{1}{\alpha_{42}\alpha_{24}}(r\alpha_{32}\alpha_{51} + \alpha_{14}) \qquad a_{43} = \frac{\alpha_{25}(\alpha_{13} - \alpha_{32})}{\alpha_{12}\alpha_{13} + \alpha_{52}\alpha_{16}} + \frac{\alpha_{52}\alpha_{16}}{\alpha_{24}\alpha_{53} - r}(r\alpha_{25}\alpha_{13} - \alpha_{31})$$

$$a_{51} = 0 \qquad a_{52} = \alpha_{51}$$

$$a_{53} = 0 \qquad a_{54} = \alpha_{52}$$

$$a_{55} = \alpha_{53}$$

The corresponding eigenvalues are λ_1 , λ_2 , λ_3 , λ_4 and λ_5 where

$$\lambda_1 = \frac{\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}\alpha_{41}\alpha_{14}\alpha_{25}}{\alpha_{12}\alpha_{34}\alpha_{53} - \alpha_{34}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}(\frac{\alpha_{51} + \alpha_{31}\alpha_{32}\alpha_{52}}{\alpha_{23}\alpha_{53}}) + \frac{\alpha_{31}(\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{13}\alpha_{16}) - \alpha_{34}}{\alpha_{13}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}},$$

$$\lambda_2 = \frac{\alpha_{23}\alpha_{51} + \alpha_{52}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}{\alpha_{23}\alpha_{53} - r\alpha_{33}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}} + \frac{\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}} + \frac{\alpha_{12}\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}{\alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}}$$

$$\lambda_3 = \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13} + r\alpha_{32}\alpha_{41}} + \frac{r\alpha_{32}\alpha_{41}}{r\alpha_{32}\alpha_{41} + \alpha_{12}\alpha_{13}} + \frac{r\alpha_{32}\alpha_{41}}{\alpha_{14} - \alpha_{44}\alpha_{34} + \alpha_{51}}(\frac{\alpha_{23}\alpha_{31}(r\alpha_{32}\alpha_{41} - \alpha_{16}\alpha_{34})}{\alpha_{31}\alpha_{13} + \alpha_{12}\alpha_{13} - \alpha_{16}})$$

$$\lambda_4 = \frac{r\alpha_{52}\alpha_{51} + \alpha_{52}}{\alpha_{41}\alpha_{13} + r\alpha_{16}} + \frac{\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13} + \alpha_{41}\alpha_{13} + r\alpha_{16}} + \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{1}{\alpha_{12}}(1 - \alpha_{13} - \alpha_{16}),$$

$$\lambda_5 = \frac{(\alpha_{31}\alpha_{13} - \alpha_{52}\alpha_{16})\alpha_{51} + \alpha_{31}\alpha_{52}}{(\alpha_{43}\alpha_{31} - r\alpha_{23}\alpha_{23})\alpha_{53}} + \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1 - r\alpha_{25}\alpha_{32}\alpha_{13}) - \alpha_{41})}{\alpha_{31}\alpha_{32} + r\alpha_{32}\alpha_{13}}.$$

hence the equilibrium point E_3 is stable if λ_i are all negative and unstable otherwise.

At the interior equilibrium point $E_4(w', x', y', z', t')$; the variational matrix becomes

$$V(E_4) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22} & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44} & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55} \end{bmatrix}$$

where

$$a_{11} = 1 - \alpha_{16} - \frac{\alpha_{32}\alpha_{31} + \alpha_{52}\alpha_{32}}{\alpha_{31}\alpha_{53} + \alpha_{16}} - \frac{\alpha_{33}\alpha_{51} + \alpha_{34}\alpha_{31}}{\alpha_{31}\alpha_{53}\alpha_{13}\alpha_{23} - \alpha_{31}} \qquad a_{12} = -\frac{\alpha_{12}(\frac{\alpha_{32}}{\alpha_{31}} + \frac{\alpha_{52}\alpha_{32}+\alpha_{42}}{\alpha_{31}\alpha_{53}-\alpha_{23}})}{\alpha_{31}\alpha_{34} + r\alpha_{25}}$$

$$a_{13} = -\alpha_{13}(\frac{\alpha_{32}\alpha_{14} + r\alpha_{25}}{\alpha_{31}\alpha_{13} - \alpha_{25}} + \frac{\alpha_{52}\alpha_{32} - \alpha_{12}}{\alpha_{31}\alpha_{53} + \alpha_{25}}) \qquad a_{14} = -\alpha_{14}(\frac{\alpha_{32}\alpha_{34} + \alpha_{31}\alpha_{33}\alpha_{51}}{\alpha_{31}\alpha_{53}})$$

$$a_{15} = \alpha_{15} \qquad a_{21} = \frac{\alpha_{34}\alpha_{13} - r\alpha_{14} - \alpha_{16}}{\alpha_{13} - \alpha_{14}\alpha_{41} - \alpha_{13}\alpha_{16}\alpha_{12}}$$

$$a_{22} = \frac{(\alpha_{12} - r)\alpha_{34} + \alpha_{25}}{\alpha_{31}\alpha_{25} - r\alpha_{12}} - \frac{\alpha_{32}\alpha_{41} - r\alpha_{23}\alpha_{53} - \alpha_{34}}{\alpha_{13}\alpha_{31}} + r - \alpha_{25} \qquad a_{23} = \frac{r\alpha_{23}\alpha_{25} + \alpha_{23}\alpha_{41} - r\alpha_{12}}{r\alpha_{52}\alpha_{53} - r\alpha_{32}}$$

$$a_{24} = \frac{\alpha_{25}r + \alpha_{14}\alpha_{25}}{\alpha_{12}\alpha_{32}\alpha_{52} - \alpha_{34}}(\frac{\alpha_{53} - \alpha_{13}\alpha_{32}\alpha_{52}}{\alpha_{23}\alpha_{53}}) + \frac{\alpha_{31}(1 - r\alpha_{16}) - \alpha_{23}\alpha_{34}}{\alpha_{23}} \qquad a_{25} = 0$$

$$a_{31} = \frac{\alpha_{23}r - \alpha_{41}\alpha_{14}\alpha_{25}}{\alpha_{12}\alpha_{34}\alpha_{53} - \alpha_{34}}(\frac{\alpha_{51} + \alpha_{31}\alpha_{32}\alpha_{52}}{\alpha_{23}\alpha_{53}}) + \frac{\alpha_{31}(1 - \alpha_{16}) - \alpha_{34}}{\alpha_{13}} \qquad a_{32} = 0$$

$$a_{33} = \frac{(1 - \alpha_{25}\alpha_{16})\alpha_{51}\alpha_{53} + \alpha_{52}}{\alpha_{53}} - r\alpha_{32} \qquad a_{34} = \frac{\alpha_{32}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{r\alpha_{32}\alpha_{31} + \alpha_{13}\alpha_{31}}$$

$$a_{35} = \alpha_{33} \qquad a_{41} = 0$$

$$a_{42} = \frac{\alpha_{51} + \alpha_{52}}{\alpha_{53}} + \frac{\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{1}{\alpha_{12}}(1 - \alpha_{13} - \alpha_{16}) \qquad a_{43} = \frac{r\alpha_{14} - \alpha_{25}}{\alpha_{12}\alpha_{34}\alpha_{53}}(\frac{\alpha_{31}\alpha_{32}\alpha_{52}}{\alpha_{23}\alpha_{53}})$$

$$a_{44} = \frac{\frac{1}{\alpha_{12}}(1 - \alpha_{41}\alpha_{13} + \alpha_{14} - r\alpha_{16})}{\alpha_{31}\alpha_{25}(\alpha_{15}\alpha_{25}\alpha_{31} - r\alpha_{24})} + \frac{\alpha_{32}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{13}\alpha_{31}} \qquad a_{45} = 0$$

$$a_{51} = 0 \qquad a_{52} = \alpha_{51}$$

$$a_{53} = 0 \qquad a_{54} = \alpha_{52}$$

$$a_{55} = \alpha_{53}$$

The corresponding eigenvalues are λ_1 , λ_2 , λ_3 , λ_4 and λ_5 where

$$\lambda_1 = \frac{\alpha_{23}r - \alpha_{41}\alpha_{14}\alpha_{25}}{\alpha_{12}\alpha_{34}\alpha_{53} - \alpha_{34}}(\frac{\alpha_{51} + \alpha_{31}\alpha_{32}\alpha_{52}}{\alpha_{23}\alpha_{53}}) + \frac{\alpha_{31}(\alpha_{12}\alpha_{32} + \alpha_{14}\alpha_{16}) - \alpha_{34}}{\alpha_{13}\alpha_{12}\alpha_{32} + \alpha_{14}} + \frac{\alpha_{23}(\alpha_{31}\alpha_{16}\alpha_{34}}{\alpha_{31}\alpha_{13}} + \frac{\alpha_{13}\alpha_{24}}{\alpha_{12}\alpha_{32} + \alpha_{14}},$$

$$\lambda_2 = \frac{\alpha_{23}\alpha_{51} + \alpha_{52}}{\alpha_{23}\alpha_{53} - r\alpha_{33}} + \frac{\alpha_{23}(\alpha_{31}(1 - \alpha_{16}) - \alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{1}{\alpha_{12}}(1 - \alpha_{25}\alpha_{13} + \alpha_{52}\alpha_{16}).$$

$$\lambda_3 = \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1-\alpha_{16})-\alpha_{34})}{\alpha_{31}\alpha_{13}+r\alpha_{23}} + \frac{r\alpha_{32}\alpha_{41}}{\alpha_{14}-\alpha_{44}\alpha_{34}+\alpha_{51}}\left(\frac{\alpha_{23}(\alpha_{31}(1-\alpha_{16})-\alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{1}{\alpha_{12}}\alpha_{13}\alpha_{16}\right),$$

$$\lambda_4 = \frac{r\alpha_{52}\alpha_{51}+\alpha_{52}}{1-\alpha_{24}\alpha_{53}} + \frac{\alpha_{23}(\alpha_{31}(1-\alpha_{16})-\alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1-\alpha_{16})-\alpha_{34})}{\alpha_{31}\alpha_{13}} - \frac{1}{\alpha_{12}}(1-\alpha_{41}\alpha_{13}+r\alpha_{16}),$$

$$\lambda_5 = \frac{(r-\alpha_{51}\alpha_{32}\alpha_{14})\alpha_{51}+\alpha_{31}\alpha_{52}}{(\alpha_{14}\alpha_{31}-r\alpha_{23}\alpha_{32})\alpha_{53}} + \frac{\alpha_{25}\alpha_{23}(\alpha_{31}(1-r\alpha_{25}\alpha_{32}\alpha_{16})-\alpha_{34})}{\alpha_{31}\alpha_{13}} + \frac{1}{r\alpha_{12}\alpha_{13}}(1-\alpha_{31}\alpha_{13}-\alpha_{52}\alpha_{16}).$$

hence the equilibrium point E_4 is stable if λ_i are all negative and unstable otherwise.

4.2.4 Parameter estimation

In this work, whenever possible values of the parameters are estimated from available experimental data used in comparable models; i.e, we took our baseline set of parameters in line with previous comparable models of prey and predator. The majority of these values of the parameters are taken from Alfred[1] and Bera *et al.*[2] except for the values of the new parameters r_2 , δ_1 , δ_2 , δ_3, α_{23} and α_{41} which we choose their values from a reasonable range in order to give the best qualitative results in the simulations. A summary of the values of the non-dimensional parameter set $\mathcal{P}$ used in the solution are given in Table below:

Table 4.2: Parameter set $\mathcal{P}$ of the model and their values

$\alpha_{12} = 3.214,$	$\alpha_{13} = 2.143$	$\alpha_{14} = 1.071$	$\alpha_{15} = 0.000893$	$\alpha_{16} = 0.00036$
$r = 0.465$	$\alpha_{23} = 3.214$	$\alpha_{24} = 0.24107$	$\alpha_{25} = 0.000893$	$\alpha_{31} = 1.875$
$\alpha_{32} = 0.000268$	$\alpha_{33} = 0.000179$	$\alpha_{34} = 0.000358$	$\alpha_{41} = 0.8036$	$\alpha_{43} = 0.0054$
$\alpha_{45} = 0.000893$	$\alpha_{51} = 0.000893$	$\alpha_{52} = 0.0026786$	$\alpha_{53} = 0.0083$	

4.3 Numerical simulation

In this section, we presented the computer simulation of different solutions of the systems using MATLAB. Thus we solved the system equation(4.1.1)-(4.1.5) and its different subsystems by using the in-built ordinary differential equation solver MatLab function ode45. For solving systems and also subsystems, we took the parametric values given in table 4.2.4.

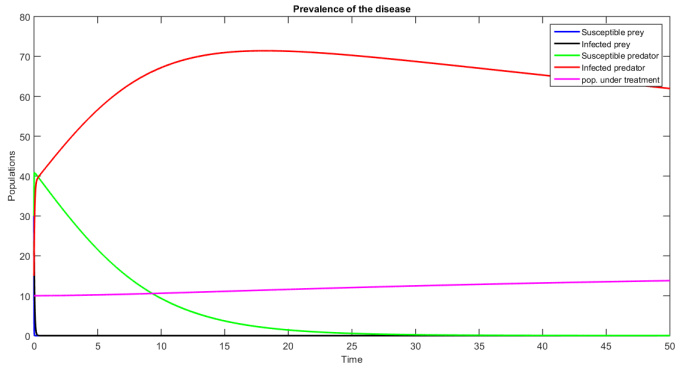

Figure 4.2: Variation in the susceptible prey, infected prey, susceptible predator and infected predator for the model (4.1.7)-(4.1.11) with the parameter values in Table (4.2.4)
.

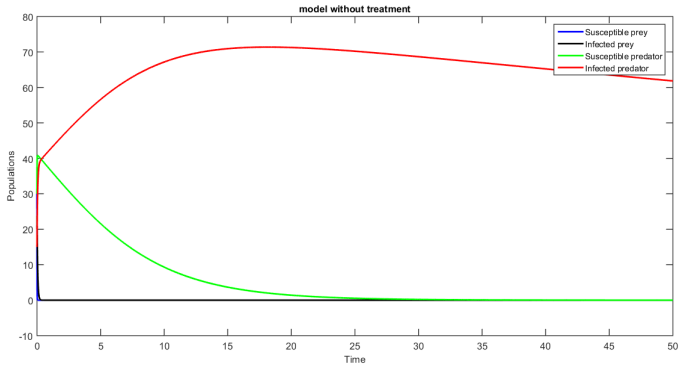

Figure 4.3: The solution of the model equation in the absence of treatment with the parameter values in Table (4.2.4) with the parameter values in Table (4.2.4)
.

A numerical study reveals an interesting result; namely: as shown in

Figure 4.2 the susceptible predator population decreases due to the infection and leads

the increase in infected predator until its steady state because of treatment; the system dynamics changes the behaviour as the result of the incorporated treatment parameters. Figure4.3 shows that the variation of the susceptible predator population due to the decline of susceptible prey and the increase of infected predator and then its decline after the steady state is attained due to treatment.That is the infected prey population increases and then decreases due to the effect of treatment.

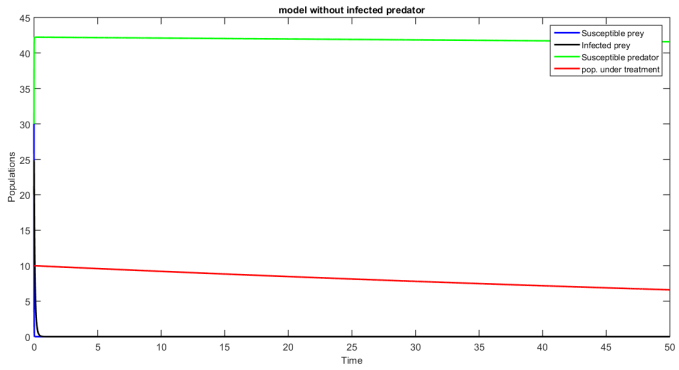

Figure 4.4: The solution of the model equation (4.1.7)-(4.1.11) in the absence of disease in prey with the parameter values given in Table (4.2.4) .

Figure 4.4 and 4.5 show the absence of disease because of treatment cause the increase of healthy population until it attain its maximum point and then decreases due to treatment and other factors.

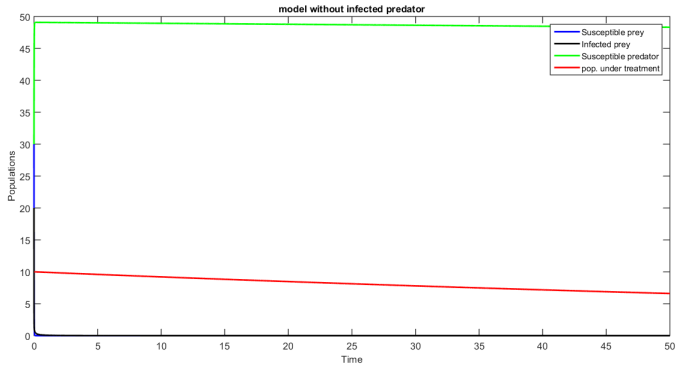

Figure 4.5: The solution of the model equation (4.1.7)-(4.1.11) in the absence of disease in predator with the parameter values in Table (4.2.4)

.

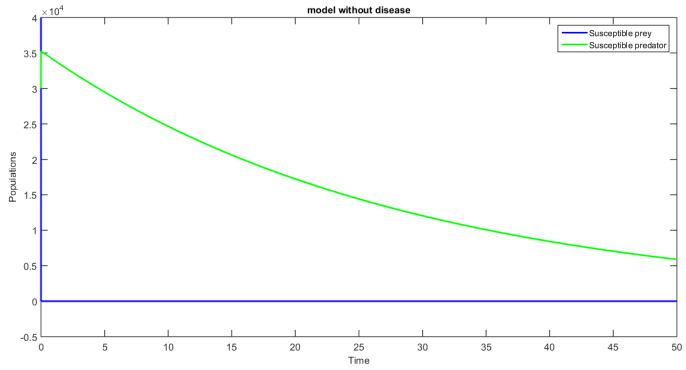

Figure 4.6: The solution of the model equation in the absence of disease with the parameter values in Table (4.2.4)

.

Figure 4.6 shows how the susceptible predator population varying with time depend on the interaction with susceptible prey population. The sharply decrease of the healthy predator population in 4.6 occurs as the result of low number of healthy prey. The high death rate of the predator also contributes in the decline of the population, although the recovery rates λ_1 and λ_2 leads the population to reach its steady state.

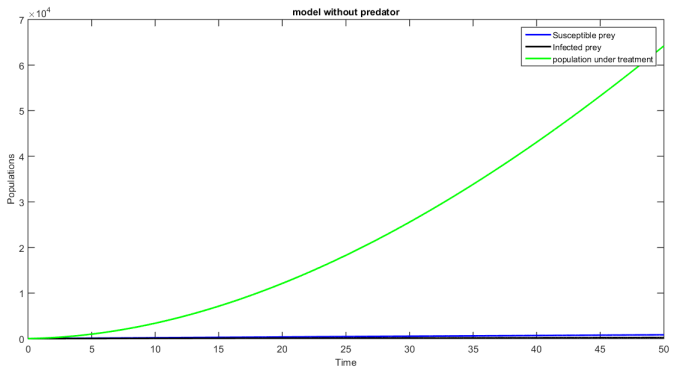

Figure 4.7: The solution of the model equation in the absence of predator with the parameter values in Table (4.2.4)

.

Figure 4.7 shows that untreated population decreases to its optimal point then slowly oscillates with low amplitude to its steady state. This figure shows the sharp increase of the infected predator populations up to its equilibrium point as the result of the disease increase and then lowered by the treatment to a steady state. Figure 4.7 shows that the treated group decreases sharply as the number of infected population is lowered. As the number of infected increases, the graph increases with high amplitude until the equilibrium point is reached. Then modified parameters values are used to obtain susceptible predator population above the infected predator population as seen in figures.

The model analysis shows that treatment of infected populations has the effect of reducing the spread of the disease. It is observed that incorporating of treatment of infected prey and predator into the system may save the population from extinction. A numerical study of the model was carried out and it was observed that the increase of the number of infected prey tends to lower the whole population. It can be concluded that the disease can be eradicated in a population through treatment.

Chapter 5

Result and Discussion

In this study, an eco-epidemiological model has been modified and analyzed. Our main objective is to understand the effect of infection disease and treatment on the dynamics of the prey-predator system. As such, the dynamical behaviour of system (4.1.1)-(4.1.5) and all possible subsystems have been investigated analytically. We then have studied the dynamics of systems in the absence of disease, in the absence of treatment, in the absence of predator,in the absence of disease in prey,in the absence of disease in predator population and then we have studied the system with disease in both the population. Moreover, the effects of changing the parameters on the dynamical behaviour of simplified form of system (4.1.1)-(4.1.5) and also its sub system are discussed numerically and the following results are obtained:

- In this section we illustrate some of the key findings of the systems numerically around the positive steady state for a wide range of parameter values. The following four parameters namely the force of infection rates α_{12} and α_{32} and the predation rates α_{13} and α_{23} are the four key parameters that directly influence the dynamics of the system.

- For the simulation interval, it is found that all the five populations are unstable with respect to time. Therefore, numerical simulations are consistent with the theoretical formulation. If we choose the different set of values of the parameters and with the

same other parameters given in table 4.2.4 and initial values, we may have different figures.

- It is observed from numerical simulations that the phase diagram of the system changes with slight changes in the initial values. From theoretical foundation it is observed that system is stable up to the critical value of time interval.

- In the study of population dynamics, the role of ecological interactions such as competition, predation etc., are well recognized. On the other hand, infection in the prey and predator populations is an issue which cannot be ignored, and also it has an important role in regulating population sizes. There are numerous field evidences where the prey and the predator are infected by disease. Thus, reality dictates that, we should be concerned with prey-predator systems, where both the populations are disease affected.

Chapter 6

Conclusion and Recommendation

6.1 Conclusion

In this study we considered a predator-prey system where both populations are infected by disease and treatment is given for them . The main objective of this study is to observe the effect of disease infection in population composition. We analyzed the local stability of equilibrium points and carried out numerical simulation of the community structure of model system for understanding how infection affect the community structure and composition. The model integrates treatment and disease infection in both prey and predator populations. Incorporating treatment in the model provides a more realistic and plays an important role for biological control of disease, and increasing the rate of treatment can increase prey and predator density population. In almost all the models with diseased prey, it is assumed that the predators eat only the infected preys (as they are weak and more vulnerable). But the susceptible preys are completely out of danger is an over simplification. In our model, we have made more realistic assumptions: (i) the susceptible predators are capable of catching both the susceptible and infected preys,and (ii)infected predators(being weak and having disturbed internal mechanism) can manage only infected preys. It is observed that incorporating of treatment of infected prey and predator into the system may save the population from extinction. It is also observed that the increase of the number of

infected prey tends to lower the whole population. It can be concluded that the disease can be eradicated in a population through treatment.

6.2 Recommendation

The main focus of this study was to introduce modified mathematical model that describe the effect of disease infection and treatment on prey and predator systems and techniques for the analysis. It is better to use this study for those who working in species health to get biological interpretation from this mathematical model to control infectious disease. We recommend increasing treatment capacity as it is a basic idea that can be decide the survival and extinction of species from the world. Ecological environment is very dynamic what we have seen yesterday is new today since this study can be used as a base ground for future other researches.

Research Fund Acknowledgement

This research project is funded by Adama science and technology university under the grant number ASTU/SM-R/026/19,Adama Ethiopia.

Bibliography

[1] Alfred Hugo, Estomih S. Massawe and Oluwole Daniel Makinde (2012), "An eco-epidemiological mathematical model with treatment and disease infection in both prey and predator population ", *Journal of Ecology and the Natural Environment* Vol. 4(10), pp. 266-279.

[2] S. P. Bera , A. Maiti, G. P. Samanta (2015), "A Prey-predator Model with Infection in Both Prey and Predator ", *Journal of Mathematical science*, Filomat 29:8, 1753-1767.

[3] S.A. Wuhaib and Y. Abu Hasan (2013),"A predator-infected prey model with harvesting of infected prey",*doi: 10.2306/scienceasia1513-1874.2013.39S.037*

[4] Ahmed Buseri Ashine and Dawit Melese Gebru (Ph.D.) (2017),"Mathematical Modeling of a Predator-Prey Model with Modified Leslie-Gower and Holling-type II Schemes ", *Global Journal of Science Frontier Research: F Mathematics and Decision Sciences* Volume 17 Issue 3 Version 1.0

[5] Hamid A. Adamu (2018), "Mathematical analysis of predator-prey model with two preys and one predator ", *International Journal of Engineering and Applied Sciences (IJEAS)* ISSN: 2394-3661, Volume-5, Issue-11,

[6] SHashi Kant, Vivex, Kumar(2017,) "Dynamics of a Prey-Predator system with infection in Prey ", *Electronic Journal of Differential Equations*, Vol. 2017, No. 209, pp. 1-27.

[7] T. K. Kar, Kunal CHakraborty and U. K. Pahari (2010),"A Prey-Predator model with Allernative Prey: Mathematical Model and analysis ", *Canadian Applied Mathematics Quarterly* Volume 18, Number 2.

[8] Krishna pada Das (2011),"A Mathematical Study of a Predator-Prey Dynamics with Disease in Predator ", *Research article*

[9] Raid Kamel Naji and Dina Sultan Al-Jaf (2011) "The Dynamics of Stage Structured Prey-Predator Model Involving Parasitic Infectious Disease ", *Applications and Applied Mathematics: An International Journal (AAM)* Vol. 6, Issue 2, pp. 529-551

[10] Prodip Roy, Dr. Krishna Pada Das, Dr. Partha Karmakar and Dr. Seema Sarkar (Mondal) (2016),"Disease and alternative Food in the Intermediate Predator stabilize Chaotic Dynamics-conclusion drawn from a Tri-TrOphic Food chain model ", *An International Journal (ECOJ)* Vol. 1, No. 1.

[11] Sachin Kumar and Harsha Kharbanda (2017),"Stability analysis of Prey-Predator Model With infection, migration and vaccination in prey ", *Math.DS*

[12] Simon, J. S. H. and Rabago, J. F. T.(2018), "Optimal Control for a Predator-Prey Model with Disease in the Prey Population ", *Malaysia journal of mathematical Sciences* 12(2): 269-285

[13] Rachna Soni and Usha Chouhan (2018), "A dynamic effect of infectious disease on prey predator system and harvesting policy", *Biotechnological Communication* 11(2): 231-237

[14] Yanni Xiao and Lansun Chen (2001),"Analysis of a Three Species Eco-Epidemiological Model ", *Journal of Mathematical Analysis and Applications* 258, 733-754 . doi:10.1006rjmaa.2001.7514, available online at http:rrwww.idealibrary.com

[15] Raid Kamel Naji and Arkan N. Mustafa (2012), "The Dynamics of an Eco-Epidemiological Model with Nonlinear Incidence Rate ", *Journal of Applied Mathematics*, Volume 2012, Article ID 852631, 24 pages

[16] David Greenhalgh , Qamar J. A. Khan and Joseph S. Pettigrew (2012), "An Eco-Epidemiological Predator-Prey Model where Predators Distinguish Between Susceptible and Infected Prey ", *Journal of Applied Mathematics*,

[17] Souvik Bhattacharya, Maia Martcheva and Xue-Zhi Li (2013), "A Predator-Prey-Disease model with immune response in infected-prey ",

Appendix

function dydt = zeros(5,1);

global alpha12 alpha13 alpha14 alpha15 alpha16 r alpha22 alpha23 alpha24 alpha25 alpha26 alpha31 alpha32 alpha33 alpha34 alpha41 alpha42 alpha43 alpha44 alpha45 alpha51 alpha52 alpha53

alpha12 =3.214;

alpha13= 2.143;

alpha14 = 1.071;

alpha15 = 0.0000893;

alpha16=0.00036;

r = 0.465;

alpha22= 3.214;

alpha23 = 0.24107;

alpha24 = 0.24107;

alpha25 = 0.000893;

alpha26=0.0384;

alpha31= 1.875;

alpha32=0.00268;

alpha33 =0.000179;

alpha34 = 0.000358;

alpha41 = 0.8036;

alpha42=0.241;

alpha43 = 0.2143;

alpha44 = 0.00268;

alpha45 = 0.0054;

alpha51 = 0.000893;

alpha52 = 0.0026786;

alpha53 = 0.0083;

dydt(1) = y(1)*(1-y(1))-alpha12*y(1)*y(2)-alpha13*y(1)*y(3)-

alpha14*y(1)*y(4)+alpha15*y(5)-alpha16*y(1);

dydt(2) = r*y(2)*(1-y(1)-y(2)) +alpha22* y(1)*y(2)-

alpha23*y(2)*y(3)-alpha24*y(2)*y(4)-alpha25*y(2);

dydt(3) = alpha31*y(1)*y(3)- alpha32*y(3)*y(4)+alpha33*y(5)- alpha34*y(3);

dydt(4) =

alpha41*y(1)*y(4)+alpha42*y(2)*y(3)+alpha43*y(2)*y(4)+alpha44*y(3)*y(4)-

alpha45*y(4);

dydt(5) = alpha51*y(2)+alpha52*y(4)-alpha53*y(5);

clear all

close all

clc

tspan = [0 50];

y0 = [30 20 30 15 10];

$[t, y] = ode45('yeroo', tspan, y0);$

plot(t,y(:,1),'b',t, y(:,2),'black',t, y(:,3),'g',t,y(:,4),'r',t,y(:,5),'m','Linewidth',2)

xlabel('Time(Days)')

ylabel('Number of Population ')

title('Prevalence of the disease') legend('Susceptible prey','Infected prey','Susceptible

predator','Infected predator','Treated population')