

## Mathematical Modeling the Impact of Infectious Diseases in Prey-predator Interactions

Abayneh Fentie Bezabih<sup>\*</sup>, Geremew Kenassa Edessa, Koya Purnachandra Rao

Department of Mathematics, Wollega University, Nekemte, Ethiopia

**Abstract:** This paper aims to show the influence of infectious disease in predator-prey system. In the present work, a four compartment mathematical eco-epidemiology model is formulated which contains susceptible predator-prey, infected predator-prey populations are considered and analyzed with the assumption that infected predators are not involved in predation. The positivity, boundedness, and existence of the solution of the system are studied. Equilibrium points of the model equations are identified. Local and Global Stability analysis has been performed. The basic reproduction number for infected prey and infected predator at disease free equilibrium point  $R_{01} = [\beta\mu k]/[rp_2(kqp_1 - \mu)]$  and  $R_{02} = [\alpha r(kqp_1 - \mu)]/[kq\delta_2(p_1)^2]$  respectively. Finally, numerical simulations are presented to clarify results.

**Keywords:** Mathematical eco-epidemiology, prey-predator system, Stability Analysis, Reproduction Number, Simulation.

Date of Submission: 07-03-2021

Date of Acceptance: 20-03-2021

### I. Introduction

Mathematical modeling is a sub specialty in Applied Mathematics which is a very important tool to understand real life problems in diverse disciplines such as biology, epidemiology, ecology etc [1-3]. In 1798, the British Economist Malthus constructed a single species model [4] and Italian Mathematician Vito Volterra first proposed a simple differential equation of prey-predator model to describe the population dynamics of two interaction species and a chemist Alfred Lotka also derived the same differential equations. Lotka-Volterra prey-predator model (1925) forms the basis of many models used in population dynamics. Afterwards, prey-predator model became an interesting area of research in applied mathematics [1, 2, 11, 12, 24, 25]. Mathematical ecology and mathematical epidemiology are distinct major fields of study in biology. But recently, these two major fields of study are merged and renamed as a new field of study called eco-epidemiology [1-4].

On the other hand, many disease transmission dynamic models originated from the pioneer classic work of Kermack - McKendrick (1927) SIR-type epidemiological model of infectious disease [1-3, 7-11, 14, 24, 25, 26, 27]. Most recent works are done by Anderson R. and May R. (1979) which involves infectious disease and predator-prey interaction of species open a new door for ecoepidemiology research [1-5, 7-11, 25, 26].

Eco-epidemiology is the branch of biomathematics that understands and analyzes the dynamics of infectious disease spread on the predator-prey interaction of species [2]. There are many types of interaction of species that can be observed in ecological systems throughout the world such as (i) predation, (ii) competition, (iii) mutualism, (iv) commensalism, (v) ammensalism [5, 14].

In a certain ecological system, predator-prey species exhibit regular cycles of population increase and decrease. This change occurs due to diseases, over predation, climate change [4, 5, 22]. Interaction of species between predators and prey is non-linear and complex phenomenon in mathematical ecology [2, 4, 5, 10]. Due to this many researchers have proposed and studied a number of ecoepidemiological models involving two or more interacting species [2, 4, 5, 10].

Ecological prey-predator systems are suffering from various infectious diseases. These diseases sometimes play a vital role in regulating the size of predator-prey population. Within a predator-prey population, it is often to see that an infectious disease spreads between predator and prey population [4, 10, 11]. The predator-prey populations could be affected due to the presence of infectious disease in the population [10].

Anderson R. and May R. (1986) were the first who merged ecology and epidemiology and formulated a prey-predator model where infection in the prey diseases. The influence of predation on epidemics has not yet been studied considerably, except the works. For instance, the disease in prey [3, 7, 8, 11, 17, 18, 21, 23, 24, 26], predators consume only infected preys, predators avoid infected prey [15], the disease in predators only [1, 25], predators consume both susceptible and infected preys [4, 5, 10, 22].

In the present paper a mathematical ecoepidemiology model with disease infection in both Prey and Predator population with the assumption that infected predators are not going to catch any of the prey due to disease infection. Here the only active predators are susceptible predators.

## II. Mathematical Model Formulation And Assumptions

The Predator-prey system contains four classes of populations. Let  $x(t)$  denotes susceptible prey,  $w(t)$  denotes infected prey,  $y(t)$  denotes susceptible predator,  $z(t)$  denotes infected predator. Then total number of prey and predator populations is given by  $N(t) = x(t) + w(t) + y(t) + z(t)$ . In formulating the present model, the following assumptions have been used.

- (i) When there is no diseases, prey population grows logistically with intrinsic growth rate  $r$  and environmental carrying capacity of the prey population  $k$
- (ii) Only susceptible prey  $x(t)$  can reproduce reaching to its carrying capacity.
- (iii) Infected prey and infected predator does not grow, recover, reproduce or compete and they are suffering with death rates  $\delta_1$  and  $\delta_2$  respectively due to infection of disease.
- (iv) Susceptible prey becomes infected prey, when it comes in contact with the infected prey and this contact process is assumed to follow simple mass action kinetics with convolution rate  $\beta$ .
- (v) Susceptible predators become infected predator, when it comes in contact with the infected predator and this contact process is assumed to follow simple mass action kinetics with convolution rate  $\alpha$ .
- (vi) The Susceptible predator population  $y(t)$  suffering with natural mortality rate  $\mu$
- (vii) The predation functional response of the susceptible predator towards susceptible prey as well as infected prey are assumed to follow Simple bilinear functional form with  $p_1, p_2$ , be predation coefficients and Consumed prey is converted into susceptible predator with efficiency  $q$
- (viii) Infected predators are assumed to be weak and unfit to catch any of the prey and if susceptible predator is once infected then it remains infected or dies out.
- (ix) All Model variables and parameters are assumed to be non negative.

**Table 1 Notation and Description of model Variables**

Variables	Descriptions
$X(t)$	Population size of susceptible preys at time $t$
$W(t)$	Population size of infected preys at time $t$
$Y(t)$	Population size of susceptible predators at time $t$
$Z(t)$	Population size of infected predators at time $t$

**Table 2 Notations and Description of model parameters**

Parameter	Description of parameter
$r$	Intrinsic growth rate of susceptible prey
$k$	Carrying capacity of susceptible prey
$\alpha$	Convolution rate of susceptible predator to be infected predator
$\beta$	Convolution rate of susceptible prey to be infected prey
$p_1$	Predation coefficient of susceptible prey due to susceptible predator
$p_2$	Predation coefficients of infected prey due to susceptible predator
$q$	Efficiency of predation
$\delta_1, \delta_2$	Death rate due to disease for infected prey and predator respectively.
$\mu$	Natural Death rate of susceptible predator

According to the above assumptions, the description of variables, and parameters the present paper will have the following mode diagram given in Fig. 1

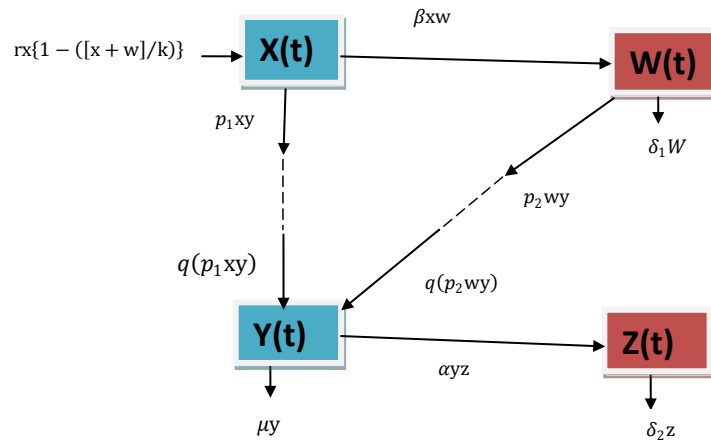


Figure 1 Model Diagram

From the model diagram the phenomenon is governed by the following systems of ordinary differential equations.

$$\frac{dx}{dt} = rx\{1 - [(x + w)/k]\} - \beta xw - p_1xy \quad (1)$$

$$\frac{dw}{dt} = \beta xw - p_2wy - \delta_1w \quad (2)$$

$$\frac{dy}{dt} = qp_1xy + qp_2wy - \alpha yz - \mu y \quad (3)$$

$$\frac{dz}{dt} = \alpha yz - \delta_2z \quad (4)$$

The initial conditions are  $x(0) = x_0 \geq 0$ ,  $w(0) = w_0 \geq 0$ ,  $y(0) = y_0 \geq 0$ ,  $z(0) = z_0 \geq 0$ ,  $p_1, p_2, > 0$  and  $0 < q \leq 1$ .

### III. Mathematical Analysis Of The Model

Model (1) – (4) will be analyzed to get insight into its dynamical features which will give better understanding on the effect of infectious disease in prey- predator System. In this section, we are going to analysis the following features of the model: Positivity, Boundedness and Existence of solutions, Trivial Equilibrium point, Axial Equilibrium point, Disease-free equilibrium point, Endemic equilibrium point, Local stability of disease -free equilibrium point, and Global stability of endemic equilibrium point. All these concepts are presented and discussed in the following sub-sections.

#### 3.1 Positivity of solutions of the model

For model (1) - (4) to be epidemiologically meaningful and well posed, it is necessary to prove that all solutions of system with positive initial data will remain positive for all time  $t \geq 0$ . This will be established by the following theorems.

**Theorem 1 [Positivity]** Let  $x(0) > 0$ ,  $w(0) > 0$ ,  $y(0) > 0$ ,  $z(0) > 0$ ,  $t(0) > 0$ , then the solutions of system equations (1) – (4)  $x(t)$ ,  $w(t)$ ,  $y(t)$ ,  $z(t)$  are positive  $\forall t \geq 0$ .

**Proof:** Positivity of the model variables is shown separately for each of the model variables  $x(t)$ ,  $w(t)$ ,  $y(t)$ ,  $z(t)$ .

**Positivity of  $x(t)$ :** The model equation (1) given by  $\frac{dx}{dt} = rx\{1 - [(x + w)/k]\} - \beta xw - p_1xy$  can be expressed without loss of generality, after eliminating the positive terms  $(rx)$  which are appearing on the right hand side, as an inequality as  $\frac{dx}{dt} \geq -\{[(x + w)/k] + \beta xw + p_1xy\}$ .

This inequality can also be written  $\frac{dx}{(\beta xw + p_1xy)} \geq \frac{dx}{[rx^2 + rwx]/k + \beta xw + p_1xy} \geq -dt$ . Then we have  $\frac{dx}{(\beta w + p_1y)x} \geq -dt$  using separation of variable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as  $x(t) \geq e^{\{(\beta w + p_1y)(-t + c)\}}$ . Recall that an exponential function is always non-negative irrespective of the sign of the exponent, Hence, it can be concluded that  $x(t) \geq 0$ .

**Positivity of  $w(t)$ :** The equation (2) given by  $\frac{dw}{dt} = \beta xw - p_2wy - \delta_1w$  can be expressed without loss of generality, after eliminating the positive term  $\beta xw$  which are appearing on the right hand side, as an inequality as  $\frac{dw}{dt} \geq -(p_2y + \delta_2)w$ . This inequality can be written as  $\frac{dw}{w} \geq -(p_2y + \delta_2)dt$  hence using variables separable method and on applying integration, the solution of the foregoing differential inequality can be obtained as  $w(t) \geq e^{-(p_2y + \delta_2)t}$ . Recall that an exponential function is always non-negative irrespective of the sign of the exponent, i.e., the exponential function  $\exp(-a_1t)$  is a non-negative quantity. Hence, it can be concluded that  $w(t) \geq 0$ .

*Positivity of  $y(t)$ :* The model equation (3) given by  $dy/dt = qp_1xy + qp_2wy - \alpha y - \mu y$  can be expressed without loss of generality, after eliminating the positive term  $(qp_1xy + qp_2wy)$  which are appearing on the right hand side, as an inequality as  $dy/dt \geq -\alpha y - \mu y$ . This inequality can be written as  $dy/dt \geq -(\alpha + \mu)y$  hence  $dy/y \geq -(\alpha + \mu)dt$ . Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as  $y(t) \geq e^{-(\alpha+\mu)t}$ . Recall that an exponential function is always non-negative irrespective of the sign of the exponent, Hence, it can be concluded that  $y(t) \geq 0$ .

*Positivity of  $z(t)$ :* The model equation (4) given by  $dz/dt = \alpha yz - \delta_2 z$  can be expressed without loss of generality, eliminating the positive term  $\alpha yz$  which are appearing on the right hand side, as an inequality as  $dz/dt \geq -(\delta_2)z$ . Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as  $z(t) \geq e^{-(\delta_2)t+C}$ . Recall that an exponential function is always non-negative irrespective of the sign of the exponent. Hence, it can be concluded that  $z(t) \geq 0$ . Thus, the model variables  $x(t), w(t), y(t), z(t)$  representing population sizes of various types of prey and predator are positive quantities and will remain in  $\mathbb{R}_+^4$  for all  $t$ .

### 3.2 Boundedness of the solutions of the Model

In the theoretical eco-epidemiology, the boundedness of the system implies that the system is biologically valid and well behaved. Then, we first show the biological validity of the model by providing the Boundedness of the solution of the model equation (1) – (4) by the following theorems.

**Theorem 2 [Boundedness]** All solutions of the model (1) – (4) are uniformly bounded.

*Proof:* To show that each population size is bounded if and only if the total population size is bounded. Hence, it is sufficient to prove that the total population size  $N = x(t) + w(t) + y(t) + z(t)$  is bounded for all  $t$ . Now, take derivatives of summation of all the five model (1) – (4) results,  $dN(t)/dt = (dx/dt) + (dw/dt) + (dy/dt)$ , then  $[dN(t)/dt + \eta N(t)] \leq rX + qp_1xy + qp_2wy + \eta N(t) = \Lambda$ . It can be shown that all feasible solutions are uniformly bounded in a proper subset  $\Omega \in \mathbb{R}_+^4$  where the feasible region  $\Omega$  is given by  $\Omega = \{(x, w, y, z) \in \mathbb{R}_+^4; N \leq (\Lambda/\eta)\}$ . Without loss of generality, after eliminating the negative terms which are appearing on the right hand side, the foregoing equation can be expressed as an inequality as  $dN(t)/dt \leq [\Lambda - \eta N(t)]$ . Equivalently this inequality can be expressed as a linear ordinary differential inequality as general solution upon solving as  $0 \leq N(x, w, y, z) \leq [\Lambda/\eta][1 - \exp(-\eta t)] + N(0)\exp(-\eta t)$ . But, the term  $N(0)$  denotes the initial values of the respective variable i.e.,  $N(t) = N(0)$  at  $t = 0$ . The particular solution can be expressed as  $N(t) \leq [\Lambda/\eta][1 - \exp(-\eta t)] + N(0)\exp(-\eta t)$ . Further, it can be observed that  $N(t) \rightarrow (\Lambda/\eta)$  as  $t \rightarrow \infty$ . That is, the total population size  $N(t)$  takes off from the value  $N(0)$  at the initial time  $t = 0$  and ends up with the bounded value  $(\Lambda/\eta)$  as the time  $t$  grows to infinity. Thus it can be concluded that  $N(t)$  is bounded as  $0 \leq N(t) \leq (\Lambda/\eta)$ . Therefore,  $(\Lambda/\eta)$  is an upper bound of  $N(t)$ . Hence, feasible solution of the system of model equations (1) – (4) remains in the positively invariant region  $\Omega$ . Thus, the system is biologically meaningful and mathematically well posed in the domain  $\Omega$ . Further, it is sufficient to consider the dynamics of the populations represented by the model system (1) – (4) in that domain. This proves the theorem. Therefore, it can be summarized the result of Theorem 2 as “the model variables  $x(t), w(t), y(t), z(t)$  are bounded for all  $t$ .”

**Theorem 3 [Existence]** Solutions of the model equations (1) – (4) together with the initial conditions  $x(0) > 0, w(0) \geq 0, y(0) \geq 0, z(0) \geq 0$  exist in  $\mathbb{R}_+^4$  i.e., the model variables  $x(t), w(t), y(t), z(t)$  and exist for all  $t$  and will remain in  $\mathbb{R}_+^4$ .

*Proof:* Let the system of equation (1) – (4) be denoted as follows:

$$f_1 = rx\{1 - [(x + w)/k]\} - \beta xw - p_1xy$$

$$f_2 = dw/dt = \beta xw - p_2wy - \delta_1 w$$

$$f_3 = dy/dt = qp_1xy + qp_2wy - \alpha yz - \mu y$$

$$f_4 = dz/dt = \alpha yz - \delta_2 z$$

According to Derrick and Groosman theorem, let  $\Omega$  denote the region  $\Omega = \{(x, w, y, z) \in \mathbb{R}_+^4; N \leq \Lambda/\eta\}$ . Then equations (1) – (4) have a unique solution if  $\partial f_i / \partial x_j, \forall i, j=1, 2, 3, 4$  are continuous and bounded in  $\Omega$ . Here  $x_1 = x, x_2 = w, x_3 = y, x_4 = z$ . The continuity and the boundedness can be verified in Table 3 below:

**Table 3 Partial derivatives of models with respect to the model variables**

<p>Forf<sub>1</sub>:  <math> (\partial f_1)/(\partial x)  =  r(1 - [x + w]/k) - rx/k - \beta w - p_1 y  &lt; \infty</math>  <math> (\partial f_1)/(\partial w)  =  -(rx)/k  &lt; \infty</math>  <math> (\partial f_1)/(\partial y)  =  -p_1 x  &lt; \infty</math>  <math> (\partial f_1)/(\partial z)  =  0  &lt; \infty</math>.</p>	<p>Forf<sub>3</sub>:  <math> (\partial f_3)/(\partial x)  =  qp_1 y  &lt; \infty</math>  <math> (\partial f_3)/(\partial w)  =  qp_2 y  &lt; \infty</math>  <math> (\partial f_3)/(\partial y)  =  qp_1 x + qp_2 w - \mu  &lt; \infty</math>  <math> (\partial f_3)/(\partial z)  =  -\alpha y  &lt; \infty</math></p>
<p>Forf<sub>2</sub>:  <math> (\partial f_2)/(\partial x)  =  \beta w  &lt; \infty</math>  <math> (\partial f_2)/(\partial w)  =  \beta x - (p_2 y + \delta_1)  &lt; \infty</math>  <math> (\partial f_2)/(\partial y)  =  -p_2 w  &lt; \infty</math>  <math> (\partial f_2)/(\partial z)  = 0 &lt; \infty</math></p>	<p>Forf<sub>4</sub>:  <math> (\partial f_4)/(\partial x)  =  0  &lt; \infty</math>  <math> (\partial f_4)/(\partial w)  =  0  &lt; \infty</math>  <math> (\partial f_4)/(\partial y)  =  \alpha z  &lt; \infty</math>  <math> (\partial f_4)/(\partial z)  =  \alpha y - \delta_2  &lt; \infty</math></p>

Thus, all the partial derivatives  $(\partial f_i)/(\partial x_j), \forall i, j = 1, 2, 3, 4$  exist, continuous and bounded in  $\Omega$ . Hence, by Derrick and Grossman theorem, a solution for the model (1) – (4) exists and is unique.

**3.3 Equilibrium Points of the model**

Disease free equilibrium point of model (1) – (4) is obtained by solving  $dx/dt = dw/dt = dy/dt = dz/dt = 0$ . Model Equation (1) – (4) possesses the following equilibrium points: (i) Trivial equilibrium point  $E_0(0, 0, 0, 0)$  (ii) Axial equilibrium point  $E_1(k, 0, 0, 0)$  (iii) disease free equilibrium points  $E_2(\bar{x}, 0, \bar{y}, 0)$  (iv) predator-free equilibrium point  $E_3(x, w, 0, 0)$ , and (v) co-existence equilibrium point/endemic equilibrium point or positive equilibrium point  $E^*(x^*, w^*, y^*, z^*)$  equilibrium points are Computed. Suppose  $dx/dt = dw/dt = dy/dt = dz/dt = 0$ , That is

$$\begin{cases} rx\{1 - [(x + w)/k]\} - \beta xw - p_1 xy = 0 \\ \beta xw - p_2 wy - \delta_1 w = 0 \\ qp_1 xy + qp_2 wy - \alpha yz - \mu y = 0 \\ \alpha yz - \delta_2 z = 0 \end{cases}$$

- i. It is clear that the system has trivial equilibrium point (TEP)  $E_0(0, 0, 0, 0)$
- ii. Axial equilibrium point (AEP)  $E_1(x, 0, 0, 0)$  can be computed as follows:  
 $rx\{1 - [x/k]\} = 0$  which implies that  $x=0$  or  $x=k$ . Since  $x \neq 0$ , hence axial equilibrium point is  $E_1(k, 0, 0, 0)$
- iii. Disease free equilibrium points (DFEP)  $E_2(\bar{x}, 0, \bar{y}, 0)$ . since  $w = z = 0$ , then
 
$$\begin{cases} rx\{1 - [x/k]\} - p_1 xy = 0 \\ qp_1 xy - \mu y = 0 \end{cases}$$

This non-linear system of equations Solved as follows:

$$\begin{cases} x[r\{1 - [x/k]\} - p_1 y] = 0 \\ y[qp_1 x - \mu] = 0 \end{cases}$$

Since  $x \neq 0, y \neq 0$ ,  $x = \mu/(qp_1)$  and  $y = [r\{1 - [x/k]\}]/p_1$ , which implies  $y = [r(kqp_1 - \mu)]/[kq p_1^2]$ , Therefore DFEP will be  $E_2(\bar{x}, 0, \bar{y}, 0) = \{\mu/(qp_1), 0, [r(kqp_1 - \mu)]/[kq p_1^2], 0\}$

- iv. Predator-free equilibrium point (PFEP)  $E_3(x, w, 0, 0)$ . Since  $y = z = 0$ , then

$$\begin{cases} rx\{1 - [(x + w)/k]\} - \beta xw = 0 \\ \beta xw - \delta_1 w = 0 \end{cases}$$

This non-linear system of equations solved as follows:

$$\begin{cases} (r\{1 - [(x + w)/k]\} - \beta w)x = 0 \\ (\beta x - \delta_1)w = 0 \end{cases}$$

since  $x \neq 0, w \neq 0$ , then it is clear that  $x = \delta_1/\beta$ ,  $w = [r(\beta k - \delta_1)]/[\beta(r + \beta k)]$ , Therefore the PFEP will be  $E_3(\delta_1/\beta, [r(\beta k - \delta_1)]/[\beta(r + \beta k)], 0, 0)$

- v. Infected predator-free equilibrium point (IPFEP)  $E_4(x, w, y, 0)$

$$\begin{cases} rx\{1 - [(x + w)/k]\} - \beta xw - p_1 xy = 0 \\ \beta xw - p_2 wy - \delta_1 w = 0 \\ qp_1 xy + qp_2 wy - \alpha yz - \mu y = 0 \\ rx\{1 - [(x + w)/k]\} - \beta xw - p_1 xy = 0 \\ (\beta x - p_2 y - \delta_1)w = 0 \\ (qp_1 x + qp_2 w - \alpha z - \mu)y = 0 \end{cases}$$

since  $x \neq 0, w \neq 0, y \neq 0$ , similar procedures it is obtained as  $x = [qk(\beta \delta_1 + rp_2)]/[\beta^2 qk + 2rqp_2]$

$$w = \{\mu(\beta^2 k + 2rp_2) - kqp_1(\beta\delta_1 + rp_2)\} / \{p_2(\beta^2 qk + 2qrp_2)\}$$

$$y = \{\beta kq(\beta\delta_1 + rp_2) + \beta r\mu - q\delta_1(\beta^2 k + 2rp_2)\} / \{qp_2(\beta^2 k + 2rp_2)\}$$

vi. Endemic equilibrium points (EEP)  $E_5(x^*, w^*, y^*, z^*)$

$$\begin{cases} rx\{1 - [(x+w)/k]\} - \beta xw - p_1 xy = 0 \\ \beta xw - p_2 wy - \delta_1 w = 0 \\ qp_1 xy + qp_2 wy - \alpha yz - \mu y = 0 \\ \alpha yz - \delta_2 z = 0 \end{cases}$$

Solving this non-linear systems will result

$$x^* = [k\delta_2 + \alpha\delta_1] / [\alpha\beta]$$

$$w^* = \{r[\alpha\beta k - \alpha\delta_1 - p_2\delta_2] - \beta p_1\delta_2\} / \{\alpha\beta(r + \beta)\}$$

$$y^* = \delta_2 / \alpha$$

$z^* = \{qp_1(\alpha\delta_1 + \alpha\beta\delta_1 + rp_2\delta_2) + rqp_2(\alpha\beta k - \alpha\delta_1 - p_2\delta_2)\} / \{\beta^3(r + \beta)\}$ , the equilibrium point is non-negative for  $r[\alpha\beta k - \alpha\delta_1 - p_2\delta_2] - \beta p_1\delta_2 > 0$

### 1.3.1 Local Stability Analysis of Equilibrium Points

The local stability can be established by linearization of the model equations using Jacobian matrix. Let the formulated model (1)-(4) can be written as function Variables  $x, w, y, z$  as follows.

$$dx/dt = rx\{1 - [(x+w)/k]\} - \beta xw - p_1 xy \equiv F(x, w, y, z)$$

$$dw/dt = \beta xw - p_2 wy - \delta_1 w \equiv G(x, w, y, z)$$

$$dy/dt = qp_1 xy + qp_2 wy - \alpha yz - \mu y \equiv H(x, w, y, z)$$

$$dz/dt = \alpha yz - \delta_2 z \equiv I(x, w, y, z)$$

The Next generation matrix of the foregoing functions is given by

$$J(x, w, y, z) = \begin{pmatrix} F_x & F_w & F_y & F_z \\ G_x & G_w & G_y & G_z \\ H_x & H_y & H_y & H_z \\ I_x & I_w & I_y & I_z \end{pmatrix}$$

Here the components of the matrix  $J(x, w, y, z)$  are partial derivatives represented by the parametric expression placed in respective positions of the following matrix. Then

$$J(x, w, y, z) = \begin{pmatrix} r[1 - (x+w)/k] - [(rx)/k] - \beta w - p_1 y & -(rx)/k & -p_1 x & 0 \\ \beta w & \beta x - p_2 y - \delta_1 & -p_2 w & 0 \\ qp_1 y & qp_2 y & qp_1 x + qp_2 w - \mu & -\alpha y \\ 0 & 0 & \alpha z & \alpha y - \delta_2 \end{pmatrix}$$

Theorem 4 [ Stability of Trivial Equilibrium Point (STEP)] The trivial equilibrium point  $E_0(0, 0, 0, 0)$  is a saddle point which is unstable.

Proof: Consider the Next generation matrix at  $E_0$  and it takes the form as

$$J(E_0) = \begin{pmatrix} r & 0 & 0 & 0 \\ 0 & -\delta_1 & 0 & 0 \\ 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & -\delta_2 \end{pmatrix}$$

Now, the eigenvalues of  $J(E_0)$  are found by solving the corresponding characteristic equation  $\det[J(E_0) - \lambda I] = 0$  as follows. The characteristic equation for the given model at trivial equilibrium point takes the form as  $(r - \lambda)(-\delta_1 - \lambda)(-\mu - \lambda)(-\delta_2 - \lambda) = 0$ .

The eigenvalues are then obtained to be  $\lambda_1 = r, \lambda_2 = -\delta_1, \lambda_3 = -\mu, \lambda_4 = -\delta_2$

Here three eigenvalues are negative and one eigenvalue is positive so the trivial equilibrium point is a saddle point which is unstable manifold in the direction of  $X$  and stable manifold in the direction of  $W, Y$ , and  $Z$ .

Theorem 5 [ Stability of Axial Equilibrium Point (SAEP)] Axial equilibrium point  $E_1(k, 0, 0, 0)$  is stable if the following two conditions hold (i)  $\beta k - \delta_1 < 0$  (ii)  $qkp_1 - \mu < 0$ . Otherwise unstable

Proof: Consider the Next generation matrix at axial equilibrium point  $J(E_1)$

$$J(E_1) = \begin{pmatrix} -r & -r & -p_1 k & 0 \\ 0 & \beta k - \delta_1 & 0 & 0 \\ 0 & 0 & qkp_1 - \mu & 0 \\ 0 & 0 & 0 & -\delta_2 \end{pmatrix}$$

To find eigenvalues of  $J(E_A)$  take  $\det[J(E_1) - \lambda I_4] = 0$  and solve as follows:

$$\begin{vmatrix} -r - \lambda & -r & -p_1 k & 0 \\ 0 & \beta k - \delta_1 - \lambda & 0 & 0 \\ 0 & 0 & qkp_1 - \mu - \lambda & 0 \\ 0 & 0 & 0 & -\delta_2 - \lambda \end{vmatrix} = 0$$

Thus,  $(-r - \lambda)(\beta k - \delta_1 - \lambda)(qkp_1 - \mu - \lambda)(-\delta_2 - \lambda) = 0$  is characteristic equation of the model at axial equilibrium point and the eigenvalues are obtained as

$$\lambda_1 = -r, \quad \lambda_2 = \beta k - \delta_1, \quad \lambda_3 = qkp_1 - \mu, \quad \lambda_4 = -\delta_2$$

Therefore the axial equilibrium point  $E_0$  will be stable, if (i)  $\beta k - \delta_1 < 0$  (ii)  $qkp_1 - \mu < 0$ , otherwise it is unstable.

**Theorem 6 [Stability of Disease-Free Equilibrium Point (SDFEP)]** The Disease-free equilibrium point  $E_2(x, 0, y, 0)$  is stable if the following four conditions are satisfied. (i)  $\alpha y - \delta_2 < 0$  (ii)  $\beta x - p_2 y - \delta_1 < 0$ , (iii)  $[(2rx - rk)/k] + p_1 y + [p_2 y + \delta_1 - \beta x] > 0$ , and (iv)  $[(2rx - rk)/k] + p_1 y * [p_2 y + \delta_1 - \beta x] + qp_1^2 xy > 0$ . otherwise unstable.

Proof: Consider the next generation matrix

$$J(x, w, y, z) = \begin{pmatrix} r[1 - (x+w)/k] - [(rx)/k] - p_1 y & -(rx)/k & -p_1 x & 0 \\ 0 & \beta x - p_2 y - \delta_1 & 0 & 0 \\ qp_1 y & qp_2 y & qp_1 x - \mu & -\alpha y \\ 0 & 0 & 0 & \alpha y - \delta_2 \end{pmatrix}$$

Here Evaluate the Next generation matrix at disease free equilibrium point  $E_2(x, 0, y, 0)$  as follows:

$$J(x, 0, y, 0) = \begin{pmatrix} r - [(2rx)/k] - p_1 y & -[(rx)/k] & -p_1 x & 0 \\ 0 & (\beta x - p_2 y - \delta_1) & 0 & 0 \\ qp_1 y & qp_2 y & (qp_1 x - \mu) & -\alpha y \\ 0 & 0 & 0 & (\alpha y - \delta_2) \end{pmatrix}$$

To find Eigen values of such matrix, compute  $\det(J(E_2) - \lambda I_4) = 0$  using fourth row

$$\begin{vmatrix} r - [(2rx)/k] - p_1 y - \lambda & -[(rx)/k] & -p_1 x & 0 \\ 0 & (\beta x - p_2 y - \delta_1) - \lambda & 0 & 0 \\ qp_1 y & qp_2 y & (qp_1 x - \mu) - \lambda & -\alpha y \\ 0 & 0 & 0 & (\alpha y - \delta_2) - \lambda \end{vmatrix} = 0$$

Now again use second row to compute determinant

$$[(\alpha y - \delta_2) - \lambda] * \begin{vmatrix} r - [(2rx)/k] - p_1 y - \lambda & -[(rx)/k] & -p_1 x \\ 0 & (\beta x - p_2 y - \delta_1) - \lambda & 0 \\ qp_1 y & qp_2 y & (qp_1 x - \mu) - \lambda \end{vmatrix} = 0$$

Then use second row to find determinant

$$[(\alpha y - \delta_2) - \lambda] * [(\beta x - p_2 y - \delta_1) - \lambda] * \begin{vmatrix} r - [(2rx)/k] - p_1 y - \lambda & -p_1 x \\ qp_1 y & (\beta x - p_2 y - \delta_1) - \lambda \end{vmatrix} = 0$$

Finally, the characteristic equation is given by

$[(\alpha y - \delta_2) - \lambda] * [(\beta x - p_2 y - \delta_1) - \lambda] * \{[\lambda + ((2rx - rk)/k) + p_1 y][\lambda + p_2 y + \delta_1 - \beta x] + qp_1^2 xy\} = 0$   
Then the Eigen values are  $\lambda_1 = \alpha y - \delta_2$ ,  $\lambda_2 = \beta x - p_2 y - \delta_1$  and Remaining two roots are the solutions of the quadratic equation

$$\left( \lambda + \overbrace{[(2rx - rk)/k] + p_1 y}^a \right) \left( \lambda + \overbrace{[p_2 y + \delta_1 - \beta x]}^b \right) + \overbrace{qp_1^2 xy}^c = 0.$$

Using Routh Hurwitz criterion stability [2], the disease free equilibrium point  $E_2(x, 0, y, 0)$  will be asymptotically stable if  $(a + b) > 0$ ,  $(a * b) + c > 0$ , and Hence DFEP is stable if (i)  $\alpha y - \delta_2 < 0$ , (ii)  $\beta x - p_2 y - \delta_1 < 0$ , (iii)  $[(2rx - rk)/k] + p_1 y + [p_2 y + \delta_1 - \beta x] > 0$ , and (iv)  $[(2rx - rk)/k] + p_1 y * [p_2 y + \delta_1 - \beta x] + qp_1^2 xy > 0$

**Theorem 6 [Stability of Predator-Free Equilibrium Point (SOPFEP)]** The predator-free equilibrium point  $E_2(x, w, 0, 0)$  is stable if the following four conditions are satisfied. (i)  $\alpha y - \delta_2 < 0$  (ii)  $\beta x - p_2 y - \delta_1 < 0$ , (iii)  $[(2rx - rk)/k] + p_1 y + [p_2 y + \delta_1 - \beta x] > 0$ , and (iv)  $[(2rx - rk)/k] + p_1 y * [p_2 y + \delta_1 - \beta x] + qp_1^2 xy > 0$ . otherwise unstable.

Proof: Consider the Next generation matrix

$$J(x, w, y, z) = \begin{pmatrix} r[1 - (x+w)/k] - [(rx)/k] - p_1 y & -(rx)/k & -p_1 x & 0 \\ 0 & \beta x - p_2 y - \delta_1 & 0 & 0 \\ qp_1 y & qp_2 y & qp_1 x - \mu & -\alpha y \\ 0 & 0 & 0 & \alpha y - \delta_2 \end{pmatrix}$$

Here Evaluate the Next generation matrix at disease free equilibrium point  $E_2(x, w, 0, 0)$  as follows:

$$J(x, w, 0, 0) = \begin{bmatrix} (rk - 2rx - rw)/k & -(rx)/k & -p_1x & 0 \\ \beta w & \beta x - \delta_1 & -p_2x & 0 \\ 0 & 0 & qp_1x + qp_1w - \mu & 0 \\ 0 & 0 & 0 & -\delta_2 \end{bmatrix}$$

To find Eigen values of such matrix, compute  $det(J(E_2) - \lambda I_4) = 0$

$$\begin{vmatrix} (rk - 2rx - rw)/k - \lambda & -(rx)/k & -p_1x & 0 \\ \beta w & \beta x - \delta_1 - \lambda & -p_2x & 0 \\ 0 & 0 & qp_1x + qp_1w - \mu - \lambda & 0 \\ 0 & 0 & 0 & -\delta_2 - \lambda \end{vmatrix} = 0$$

Now using fourth row to find determinant

$$[-\delta_2 - \lambda] * \begin{vmatrix} (rk - 2rx - rw)/k - \lambda & -(rx)/k & -p_1x \\ \beta w & \beta x - \delta_1 - \lambda & -p_2x \\ 0 & 0 & qp_1x + qp_1w - \mu - \lambda \end{vmatrix} = 0$$

Then use third row to compute determinant

$$[-\delta_2 - \lambda] * [qp_1x + qp_1w - \mu - \lambda] * \begin{vmatrix} (rk - 2rx - rw)/k - \lambda & -(rx)/k \\ \beta w & \beta x - \delta_1 - \lambda \end{vmatrix} = 0$$

Finally, the characteristic equation is given by

$$[-\delta_2 - \lambda] * [qp_1x + qp_1w - \mu - \lambda] * \{[(rk - 2rx - rw)/k - \lambda][\beta x - \delta_1 - \lambda] + [(r\beta xw)/k]\} = 0$$

Then the Eigen values are  $\lambda_1 = -\delta_2 < 0$ ,  $\lambda_2 = qp_1x + qp_1w - \mu$  and Remaining two roots are the solutions of the quadratic equation

$$\left( \lambda + \frac{a}{(2rx + rw - rk)/k} \right) \left( \lambda + \frac{b}{[\delta_1 - \beta x]} \right) + \frac{c}{(r\beta xw)/k} = 0.$$

Using Routh Hurwitz criterion stability[2], the disease free equilibrium point  $E_2(x, 0, y, 0)$  will be asymptotically stable if  $(a + b) > 0$ ,  $(a * b) + c > 0$

hence DFEP is stable if (i)  $qp_1x + qp_1w - \mu < 0$ , (ii)  $[(2rx + rw - rk)/k] + [\delta_1 - \beta x] > 0$ , and (iii)  $[(2rx + rw - rk)/k] * [\delta_1 - \beta x] + (r\beta xw)/k > 0$

### 1.3.2 Global Stability Analysis

Here, the global stability analysis of the system of model equations (1) - (4) around the positive equilibrium point  $E^*(x^*, w^*, y^*, z^*)$  or the coexistence equilibrium is performed by stating the following theorem.

**Theorem 7 [Global Stability of Endemic Equilibrium Point (GSOEEP)]** The coexistence equilibrium point/endemic equilibrium point/positive equilibrium point  $E^*(x^*, w^*, y^*, z^*)$  of system (1) - (4) is globally asymptotically stable.

**Proof:** Consider the following on Liapunove function  $L(x, w, y, z)$  [1, 19]

$$L = m_1(x - x^*)^2/2 + m_2(w - w^*)^2/2 + m_3(y - y^*)^2/2 + m_4(z - z^*)^2/2$$

$$dL/dt = m_1(x - x^*) dx/dt + m_2(w - w^*) dw/dt + m_3(y - y^*) dy/dt + m_4(z - z^*) dz/dt \quad (10)$$

Now substitute the model equation (1) - (4) into equation (10)

$$\begin{aligned} dL/dt = m_1(x - x^*) \{rx[1 - [(x + w)/k]] - \beta xw - p_1xy\} \\ + m_2(w - w^*) \{\beta xw - p_2wy - \delta_1w\} \\ + m_3(y - y^*) \{qp_1xy + qp_2wy - \alpha yz - \mu y\} \\ + m_4(z - z^*) \{\alpha yz - \delta_2z\} \end{aligned}$$

Take out x, w, y, z and put as change

$$\begin{aligned} dL/dt = m_1(x - x^*)(x - x^*) \{r[1 - [(x + w)/k]] - \beta w - p_1y\} \\ + m_2(w - w^*)(w - w^*) \{\beta x - p_2y - (\delta_1/w)\} \\ + m_3(y - y^*)(y - y^*) \{qp_1x + qp_2w - \alpha z - \mu\} \\ + m_4(z - z^*)(z - z^*) \{\alpha y - \delta_2\} \end{aligned}$$

By rearranging, it could be obtained

$$\begin{aligned} dL/dt = -m_1(x - x^*)^2 \{-r[1 - [(x + w)/k]] + \beta w + p_1y\} \\ - m_2(w - w^*)^2 \{-\beta x + p_2y + (\delta_1/w)\} \\ - m_3(y - y^*)^2 \{-qp_1x - qp_2w + \alpha z + \mu\} \\ - m_4(z - z^*)^2 \{-\alpha y + \delta_2\} \end{aligned}$$

Thus it is possible to set  $m_1, m_2, m_3, m_4$  such that  $dL/dt \leq 0$  and endemic equilibrium point is globally stable.

Alternative proof: Take a proper Liapunove function [20],  $V(x, w, y, z): \mathbb{R}_+^4 \rightarrow \mathbb{R}$  such that

$$\begin{aligned} V(t) = m_1(x - x^* - x^* \ln(x/x^*)) + m_2(w - w^* - w^* \ln(w/w^*)) + m_3(y - y^* - y^* \ln(y/y^*)) \\ + m_4(z - z^* - z^* \ln(z/z^*)) \end{aligned}$$

For  $V(t)$  derivation along the system gives



$$dV(t)/dt = m_1([x - x^*]/x)[dx/dt] + m_2([w - w^*]/w)[dw/dt] + m_3([y - y^*]/y)[dy/dt] + m_4(z)[dz/dt]$$

$$dV(t)/dt = m_1([x - x^*]/x)[rx\{1 - [(x + w)/k]\} - \beta xw - p_1xy] + m_2([w - w^*]/w)[\beta xw - p_2wy - \delta_1w] + m_3([y - y^*]/y)[qp_1xy + qp_2wy - \alpha yz - \mu y] + m_4([z - z^*]/z)[\alpha yz - \delta_2z]$$

Take out x, w, y, z and put as change, the take out negative from all brackets

$$dV(t)/dt = -(m_1/x)(x - x^*)^2\{-r\{1 - [(x + w)/k]\} + \beta w + p_1y\} - (m_2/x)(w - w^*)^2\{-\beta x + p_2y + (\delta_1/w)\} - (m_3/x)(y - y^*)^2\{-qp_1x - qp_2w + \alpha z + \mu\} - (m_4/x)(z - z^*)^2\{-\alpha y + \delta_2\}$$

Thus it is possible to set  $m_1, m_2, m_3$  and  $m_4$  such that  $dL/dt \leq 0$  and endemic equilibrium point is globally stable.

### 1.3.3 Reproduction number or Threshold number $R_0$

If  $R_0 < 1$  then each infected individual produces on average less than one new infected individual so it is expected that the disease would die out. On the other hand if  $R_0 > 1$  then each individual produces more than one new infected individual so it is expected that the disease would continue spreading in the population.

Theorem 8 [Infected Prey Threshold] The Reproduction number for infected prey at Disease free equilibrium point is given by  $R_{01} = [\beta\mu k]/[rp_2(kqp_1 - \mu)]$

Proof: Consider infected prey equation (2)  $dw/dt = \beta xw - p_2wy - \delta_1w = \{\beta x - p_2y - \delta_1\}w = \{\beta x - (p_2y + \delta_1)\}w$

Now Let  $F = \beta x$  and  $V = p_2y + \delta_1$ , Evaluate  $F$  and  $V$  at Disease equilibrium point  $(\bar{x}, 0, \bar{y}, 0)$

Then  $F = \beta\bar{x}$  and  $V = p_2\bar{y} + \delta_1$

It is known that  $R_{01} = FV^{-1} = [\beta\bar{x}]/[p_2\bar{y} + \delta_1]$  and hence  $R_{01} = [\beta\mu k]/[rp_2(kqp_1 - \mu)]$  proved.

Theorem 9 [Infected Predator Threshold] The Reproduction number for infected predators at disease free equilibrium point takes the form as  $R_{02} = [\alpha r(kqp_1 - \mu)]/[kq\delta_2(p_1)^2]$

Proof: Consider the infected predator model equation (4)  $dz/dt = \{\alpha yz - \delta_2z\} = \{\alpha y - \delta_2\}z$

Now Let  $F = \alpha y$  and  $V = \delta_2$  Evaluate  $F$  and  $V$  at disease free equilibrium point  $(\bar{x}, 0, \bar{y}, 0)$ ,

Then  $F = \alpha\bar{y}$  and  $V = \delta_2$

It is known that  $R_{02} = FV^{-1} = \alpha\bar{y}/\delta_2$  and hence  $R_{02} = [\alpha r(kqp_1 - \mu)]/[kq\delta_2(p_1)^2]$  hence proved.

## IV. Result And Discussion

In this section, Numerical simulation of model equations (1) - (4) is carried out using the software DEDiscover version: 2.6.4. Model equations and parameters were arranged for DEDiscover software in this way for simulation purpose.

$dx/dt = r*x*(1 - (x+w)/k) - \text{Beta}*x*w - p_1*x*y$  // Susceptible prey

$dw/dt = \text{Beta}*x*w - p_2*w*y - \text{Delta}_1*w$  // Infected prey

$dy/dt = q*p_1*x*y + q*p_2*w*y - \text{Alpha}*y*z - \text{Mu}*y$  // Susceptible predator

$dz/dt = \text{Alpha}*y*z - \text{Delta}_2*z$  // Infected predator

Table 4 Parameter values used for Simulations

Parameter	Values	Source
r	11.2000	[1]
k	30.0000	[1]
Beta	1.2000	[1]
p_1	0.4000	[1]
p_2	0.6000	[1]
Delta_1	0.0100	Assumed
q	0.2500	[1]
Alpha	1.3000	[19]
Mu	0.0010	Assumed
Delta_2	0.4000	Assumed

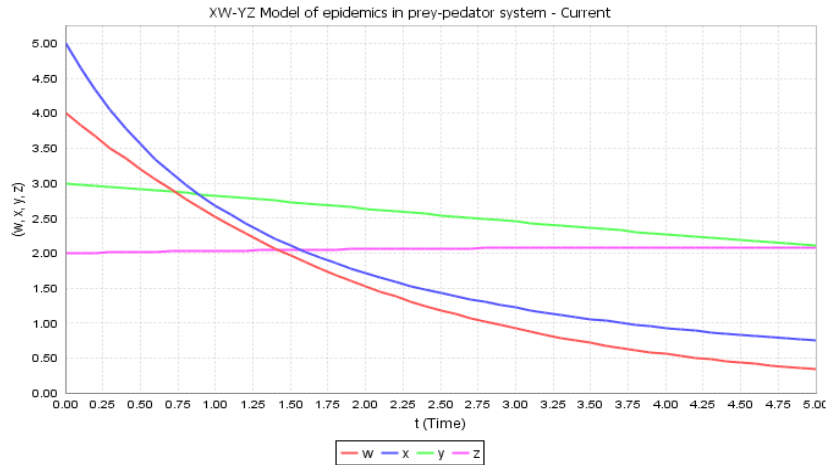


Figure 1 Time series plot for model with initial conditions

In fig.1 it can be observed that Susceptible and infected prey populations are continuously decreasing which implies that preys are under the influence of Susceptible predators. Then after some time there is relative decrease on the susceptible predators due to scarcity of prey. Infected predators are constant which confirms that they have no influence on prey. Infected preys are more exposed to predators than susceptible preys.

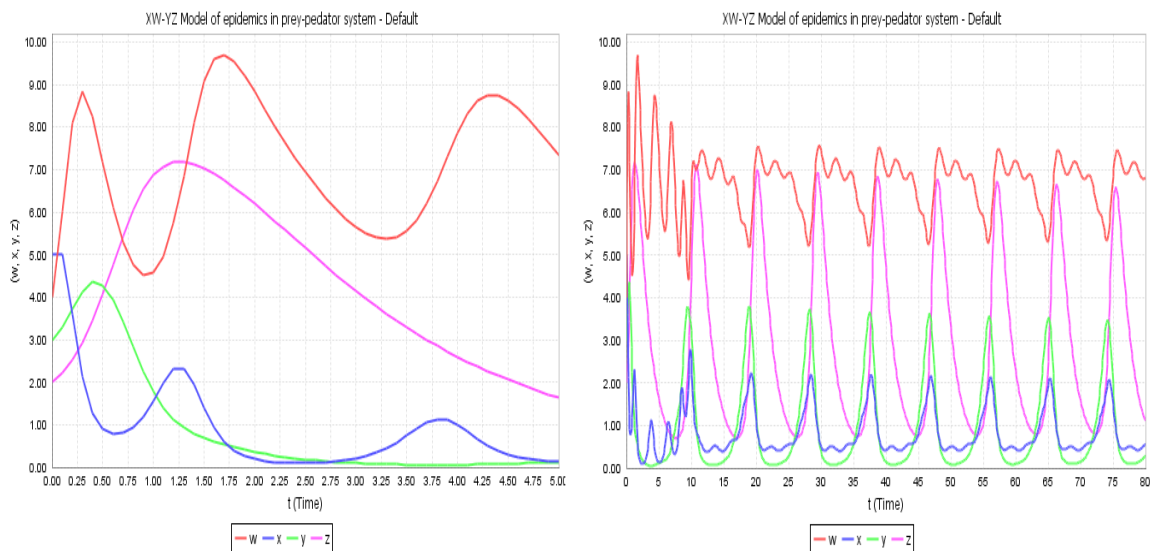


Figure 2a Time Series plot Figure 2b Time Series plot

From Fig 2a and Fig.2b, shows that continuous change of population occurs in prey-predator system which are described by oscillation of the graph with different amplitude, except infected predators with constant Amplitude. In the long run we have the following prey- predator graphs.

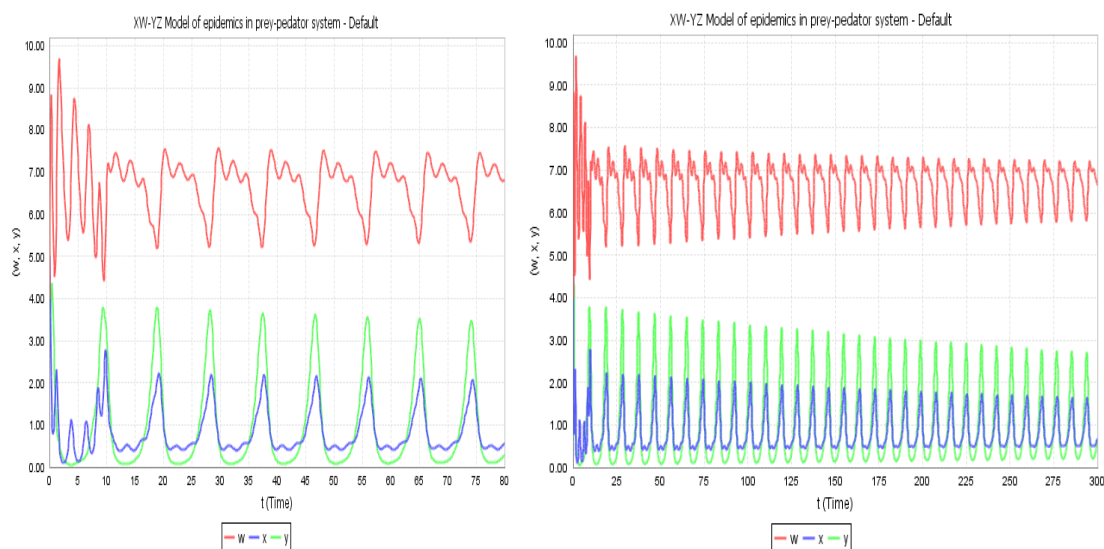


Figure 3c Time Series plot Figure 3d Time Series plot

From Fig.3c, and Fig.3d, shows that the influence of susceptible predator on preys. This can be explained as follows. If the population of susceptible predator increase, then infected prey population decrease which mean all the Susceptible predator populations size increase as a result of prey population and on the long run both prey and predator graph shows fast oscillation ,decrease in amplitude and total population decrease due to due to predation or infectious disease.

### V. Conclusion

The positivity, boundedness, and existence of solutions of the system are shown to hold implying that the system is meaningful and biologically well behaved. Disease free equilibrium points and endemic equilibrium points are Computed. Local stability analysis has been done using the Concept of next Generation matrix and Routh Hurwitz criterion. Global Stability analysis of endemic equilibrium point is proved by taking appropriate Liapunov function.

A continuous change of population occurs in prey-predator system which are described by oscillation of the graph with different amplitude, except infected predators with constant Amplitude due to our assumption infected predators are not involved in predation. Susceptible predator populations size increase as a result of prey population and on the long run both prey and predator graph shows fast oscillation , decrease in amplitude and total population decrease due to predation and infectious disease.

one can extend this work by including other assumptions like the predator grows logistically, or infected predator-prey recover from disease, or adding other parameters like vaccination, immigration, migration on prey-predator interaction.

### Acknowledgements

The authors would like to thank the editor and the anonymous reviewers of IOSR Journal of Mathematics for their helpful suggestions and remarks.

### References

- [1] Raid Kamel Naji, Rasha Ali Hamodi, The dynamics of an ecological model with infectious disease, Global Journal of Engineering Science and Researches. Raji, 3(8): August 2016 DOI: 10.5281/zenodo.61221
- [2] Karrar Qahtan Khalaf, Azhar Abbas Majeed, and Raid Kamel Naji. The Local Bifurcation and the Hopf Bifurcation for Eco-Epidemiological System with One Infectious Disease, Gen. Math. Notes, Vol. 31, No. 1, November 2015, pp. 18-41. Available free online at <http://www.geman.in>
- [3] Raid Kamel Naji, Kawa Ahmed Hasan. The Dynamics of Prey-Predator Model With Disease In Prey, J. Math. Comput. Sci. 2 (2012), No. 4, 1052-1072, Available online at <http://scik.org>
- [4] Alfred Hugo, Estomih S. Massawe, and Oluwole Daniel Makinde. An Eco-Epidemiological Mathematical Model with Treatment and Disease Infection in both Prey and Predator Population. Journal of Ecology and natural environment Vol.4(10), pp.266-273, July 2012.
- [5] Sachin Kumar and Harsha Kharbanda. Stability Analysis Of Prey-Predator Model With Infection, Migration and Vaccination In Prey, arXiv:1709.10319v1 [math.DS], 29 Sep 2017.
- [6] Paritosh Bhattacharya, Susmita Paul and K.S.Choudhury (2015). Mathematical Modeling of Ecological Networks, Structure and Interaction Of Prey and Predator, Palestine Journal of Mathematics Vol. 4(2), 335-347.
- [7] C. M. Silva (2017). Existence of periodic solutions for periodic eco-epidemic models with disease in the prey, J. Math. Anal. Appl. 453(1), 383-397.
- [8] J. Chattopadhyay and O. Arino. A predator-prey model with disease in the prey, Nonlinear Analysis, 36 (1999), 747-766.

- [9] J. A. Collera. Stability and bifurcations in delayed three-species model, *Advanced Studies in Biology*, 5(11), (2013), 455–464.
- [10] S.P.Bera, A.Maiti, G.Samanta. A Prey-predator Model with Infection in both prey and predator, *Filomat* 29:8 (2015), 1753-1767.
- [11] Asrul Sani, Edi Cahyono, Mukhsar, Gusti Arviana Rahman (2014). Dynamics of Disease Spread in a Predator-Prey System, Indonesia, *Advanced Studies in Biology*, Vol. 6, 2014, No. 4, 169 - 179
- [12] M. Y. Dawed, P. R. Koya, and T. Tibebe. Analysis of Prey – Predator System with Prey Population Experiencing Critical Depensation Growth Function. *American Journal of Applied Mathematics*. Vol. 3, No. 6, 2015, Pp. 327-334
- [13] A.M.Tulu and P. R. Koya. The Impact of Infective Immigrants on the Spread of Dog Rabies. *American Journal of Applied Mathematics*. Vol. 5, No. 3, 2017, Pp. 68-77.
- [14] G.K. Edessa, Boka Kumsa, P. R. Koya. Modeling and Simulation Study of the Population Dynamics of Commensal-Host-Parasite System. *American Journal of Applied Mathematics*. Vol. 6, No. 3, 2018, Pp. 97-108.
- [15] M. Haque and D. Greenhalgh. When a predator avoids infected prey: A model-based theoretical study, *Math. Med. Biol.*, 27(1) (2010), 75–94.
- [16] O. Diekmann, J.A.P Heesterbeek and J.A. J. Metz. *Mathematical Epidemiology of Infectious diseases: Model Building, Analysis and Interpretation*. John Wiley: New York, 2000.
- [17] M. Haque. A predator–prey model with disease in the predator species only, *Nonlinear Anal., Real World Appl.*, 11(4) (2010), 2224–2236.
- [18] M. Liu, Z. Jin and M. Haque, An impulsive predator-prey model with communicable disease in the prey species only, *Nonlinear Anal. Real World Appl.* 10 (2009), no. 5, 3098–3111.
- [19] A. J. Lotka. *Elements of mathematical biology*, Dover Publications, Inc., New York, NY, 1958.
- [20] K. P. Hadeler and H. I. Freedman. Predator-prey populations with parasitic infection, *J. Math. Biol.*, 27 (1989), 609–631.
- [21] M. S. Rahman and S. Chakravarty. A predator-prey model with disease in prey. *Nonlinear Analysis: Modeling and Control*, 18(2) (2013), 191–209.
- [22] A. F. Bezabih, G. K. Edessa, K. P. Rao, *Mathematical Eco-Epidemic Model on Prey-Predator System*. *IOSR Journal of Mathematics (IOSR-JM)*, 16(1), (2020): pp. 22-34.
- [23] G. K. Edessa, B. Kumsa, P. R. Koya. Dynamical behavior of Susceptible prey – Infected prey – Predator Populations. *IOSR Journal of Mathematics (IOSR-JM)* 14.4 (2018) PP: 31-41.
- [24] Raid Kamel Naji and Arkan N. Mustafa. The dynamics of an Eco-Epidemiological model with nonlinear incidence rate. *Hindawi journal of Applied mathematics*, volume 2012, Article ID:852631, 24 pages
- [25] Krishna pada Das. A Mathematical Study of a Predator-Prey Dynamics with Disease in Predator. *International Scholarly Research Network ISRN Applied Mathematics* Volume 2011, Article ID 807486, 16 pages doi:10.5402/2011/807486
- [26] A. F. Bezabih, G. K. Edessa, P. R. Koya. Mathematical Eco-Epidemiological Model on Prey-Predator System. *Mathematical Modeling and Applications*. Vol. 5, No. 3, 2020, pp. 183-190. doi: 10.11648/j.mma.20200503.17
- [27] A. F. Bezabih, G.K. Edessa, P. R. Koya. Mathematical Epidemiology Model Analysis on the Dynamics of COVID-19 Pandemic. *American Journal of Applied Mathematics*. Vol. 8, No. 5, 2020, pp. 247-256. doi: 10.11648/j.ajam.20200805.12.

Abayneh Fentie Bezabih, et. al. "Mathematical Modeling the Impact of Infectious Diseases in Prey-predator Interactions." *IOSR Journal of Mathematics (IOSR-JM)*, 17(2), (2021): pp. 45-56.