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Full Length Research Paper

An eco-epidemiological mathematical model with treatment and disease infection in both prey and predator population

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This paper examines 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.

Key words: Predator-prey system, eco-epidemiology.

INTRODUCTION

Ecological populations suffer from the various infectious diseases and these diseases have a significant role in regulating population size. Thus, it is worthwhile to study the combined effect of epidemiological and demographic features on the real ecological populations. Mathematical study of such eco-epidemiological model has explored various unknown aspects of ecological population (Mukhopadhyay and Bhattacharyya, 2009). However, in ecosystem, the interaction between the predator and prey is a nonlinear 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 development of mathematical models that are essential tools in understanding the interaction mechanisms for persistence or extinction of species in natural systems.

Eco-epidemiology studies the direct and indirect effects that diseases have on interacting populations (Venturino, 2002). Infectious diseases have been known to be an important regulating factor for human and animal population sizes (Hsieh and Hsiao, 2008). 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.

There has been a growing interest in the study of diseases in a prey-predator system. In recent decades, theoretical ecologists as well as epidemiologists became increasingly interested in so-called eco-epidemiology. Eco-epidemic models describe ecosystems of interacting populations among which a disease spreads (Arino et al., 2004; Beltrami and Carroll, 1994; Venturino, 1994). It has been established that invading diseases tend to destabilize the predator-prey communities (Anderson and May, 1986; Dobson, 1988; Hadeler and Freedman, 1989). However, Hilker and Schmitz (2008) 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 (Arino et al., 2004; Chattopadhyay and Arino, 1999). Venturino (2002) considered epidemic aspects in predator-prey models with disease in the predator. Hsieh and Hsiao (2008) considered a predator–prey model with disease infection

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in both populations to account for the possibility of a contagious disease crossing species barrier from prey to predator. A similar idea was also exploited in Venturino (2006) on disease in interacting species models. The work of Venturino (1995) tries to merge the epidemic models with some demographic issues, to take into account the important effects of transmissible diseases in ecological relationships between species.

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 basic eco-epidemiological model which was studied by Mukhopadhyay and Bhattacharyya (2009) by incorporating infected predator group together and treatment.

MODEL FORMULATION

A mathematical model is proposed and analyzed to study the functional response of the predator toward a susceptible as well as infected prey. This dynamics is assumed to follow Michaelis-Menten kinetics Holling type predation function (Mukhopadhyay and Bhattacharyya, 2009). The model consists of three populations: (i) the prey population density denoted by $N_1(t) = \mathrm{S}(t) + \mathrm{I}(t)$, (ii) the predator population density denoted by $N_2(t) = \mathrm{Y}(t) + \mathrm{Z}(t)$ and (iii) population of infected prey and predator under treatment is denoted by $T(t)$.

In formulating the model, the following assumptions are taken into consideration:

(i) In the absence of the disease, the prey population grows logistically with intrinsic growth rate r and environmental carrying capacity k.

(ii) In the presence of the disease, the prey and predator populations consist of two subclasses, namely, the susceptible prey $S(t)$ and the infected prey $I(t)$; susceptible predator $Y(t)$ and infected predator $Z(t)$.

(iii) Only the susceptible prey can reproduce. Logistic law is used to model the birth process with the assumption that births should always be positive. The infected prey is removed with the positive death rate e_2 or by predation before the possibility of reproducing. However, the infected population I contributes with S to population growth towards the carrying capacity k.

(iv) 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. Moreover, the disease is not genetically inherited. The infected population can only recover through treatment.

(v) 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 kinetics with β as the rate of conversion.

The predator populations suffers loss due to the death at constant rates of e_3 and e_4. The predation functional response of the predator towards susceptible as well as infected prey are assumed to follow Michaelis-Menten kinetics and is modelled using a Holling type-II functional form with predation coefficients p_1, p_2, p_3 and half-saturation constant m. Consumed prey is converted into predator with efficiency q. The infected prey and predator are treated at the rates a_1 and a_2 and removed without immunity at the rates λ_1 and λ_2, while e_1 is the death of infected prey and predator under treatment.

Taking into account the aforementioned considerations, we then have the schematic flow diagram shown in Figure 1. From the flow chart (Figure 1), the model will be governed by the following equations:

$$\frac{dS}{dt} = rS\left(1-\frac{S+I}{k}\right) - \beta SI - \frac{p_1 SY}{m+S} - \frac{p_3 SZ}{m+S} + \lambda_1 T,$$

$$\frac{dI}{dt} = \beta SI - (a_1 + e_2)I - \frac{p_2 IY}{m+I},$$

$$\frac{dY}{dt} = q\frac{p_1 SY}{m+S} - e_3 Y + \lambda_2 T, \tag{1}$$

$$\frac{dZ}{dt} = q\frac{p_2 IY}{m+I} + q\frac{p_3 SZ}{m+S} - (a_2 + e_4)Z,$$

$$\frac{dT}{dt} = a_1 I + a_2 Z - (\lambda_1 + \lambda_2 + e_1)T,$$

with the initial conditions

$S(0) = S_0 > 0,\quad I(0) = I_0 > 0,\quad Y(0) = Y_0 > 0,\quad Z(0) = Z_0 > 0,$
$T(0) = T_0 > 0,$ $p_1, p_2, p_3 > 0$ and $0 < q \leq 1$.

MODEL ANALYSIS

The model (Equation 1) will be analyzed qualitatively to get insights into its dynamical features which will give better understanding of the effect of treatment of an infected prey and predator population.

Boundedness 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) by the following theorem (Mukhopadhyay and Bhattacharyya, 2009):

Theorem 1

All solutions of the system (1) are uniformly bounded.

Proof: Let $W = S + I + Y + Z + T$

Then, we have

$$\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dY}{dt} + \frac{dZ}{dt} + \frac{dT}{dt} \tag{2}$$

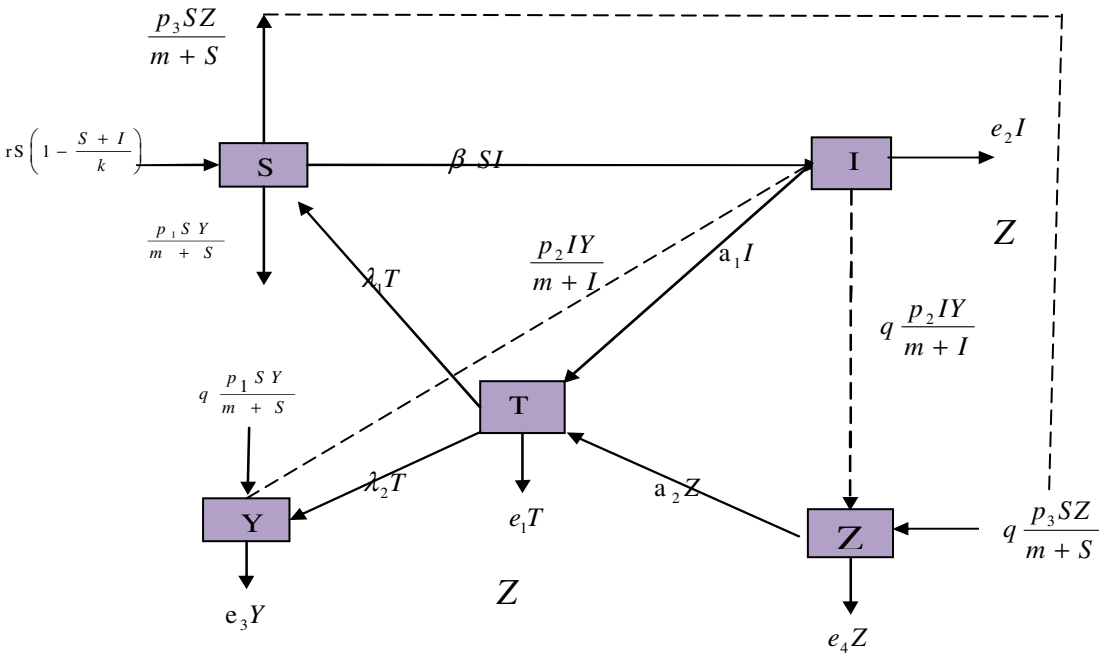

Figure 1. Model flowchart.

By substituting the model equations (1) in (2) one gets:

$$\frac{dW}{dt} \le rS\left(1-\frac{S}{k}\right)+\lambda_1 T-(a_1+e_2)I+\lambda_2 T+e_3 Y-(a_2+e_4)Z+a_1 I+a_2 Z-(\lambda_1+\lambda_2+e_1)T$$

or

$$\frac{dW}{dt} \le rS-e_2 I-e_3 Y-e_4 Z-e_1 T \le (r+1)S-(S+e_2 I+e_3 Y+e_4 Z+e_1 T) \le \hat{k}(r+1)-hW$$

where $\hat{k}=\max\{S(0),\ k\}$ and $h=\min\{1+e_1+e_2+e_3+e_4\}$.

The equation $\frac{dW}{dt}+hW \le \hat{k}(r+1)$

has a solution $W \le \frac{\hat{k}}{h}(r+1)\left(1-e^{-ht}\right)$ (3)

As $t \to \infty$, we have $W \le \frac{\hat{k}}{h}(r+1)$, implying that the solution is bounded for $0 \le W \le \frac{\hat{k}}{h}(r+1)$

Therefore, all solutions of the model (1) in $\mathbb{R}^5_+$ are confined in the region:

$$\Gamma=\left\{(S,\ I,\ Y,\ Z,\ T) \in \mathbb{R}^5_+ :\ W \le \frac{\hat{k}}{h}(r+1)+\varepsilon\right\}$$ for all $\varepsilon > 0$ and $t \to \infty$.

Positivity of solutions

For model (Equation 1) 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 times $t > 0$. This will be established by the following theorem:

Theorem 2

Let $S(0)>0$, $I(0)>0$, $Y(0)>0$, $Z(0)>0$, $T(0)>0$. Then the solutions $S(t)$, $I(t)$, $Y(t)$, $Z(t)$, $T(t)$ of system (1) are positive $\forall t \geq 0$.

Proof: To prove theorem 1, we use all equations of the model (1). From the 1st equation, we obtain the inequality expression

$$\frac{dS}{dt} \leq rS\left(1-\frac{S}{k}\right)$$

which gives

$$S \leq \frac{kS(0)}{e^{-rt}(k-S(0))+S(0)}$$

As $t \to \infty$, we obtain $0 \leq S \leq k$. Hence all feasible solution of system (1) enter region $\Gamma = \{(S, I, Y, Z, T)\}$.

Similar proofs can be established for the positivity of the other solutions.

Disease free equilibrium points

The disease free equilibrium point of the system (1) is obtained by setting

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dY}{dt} = \frac{dZ}{dt} = \frac{dT}{dt} = 0.$$

System (1) possesses the following equilibrium points:

(i) The trivial equilibrium point $E_T(0,0,0,0,0)$

(ii) The axial equilibrium point $E_A(k,0,0,0,0)$

(iii) The boundary equilibrium points $E_{B1} = (\widehat{S},\ 0,\ \widehat{Y},\ 0,\ 0)$ and $E_{B2} = (S^*,\ I^*,\ 0,\ 0,\ T^*)$

where

$$\widehat{S} = \frac{me_3}{qp_1 - e_3}, \quad \widehat{Y} = \frac{r}{k}\left(k - \frac{me_3}{qp_1 - e_3}\right)\left(\frac{mq}{qp_1 - e_3}\right), \quad S^* = \frac{a_1 + e_2}{\beta}$$

$$I^* = \frac{a_1 r\left\{k\beta(a_1+e_2) - (a_1+e_2)^2\right\}(\lambda_1+\lambda_2+e_1)}{\beta\left\{r(a_1+e_2)(\lambda_1+\lambda_2+e_1) + k\beta(a_1\lambda_2 + a_1e_1) + e_2(\lambda_1+\lambda_2+e_1)\right\}},$$

$$T^* = \frac{a_1 r\left\{k\beta(a_1+e_2) - (a_1+e_2)^2\right\}(\lambda_1+\lambda_2+e_1)}{\beta\left\{r(a_1+e_2)(\lambda_1+\lambda_2+e_1) + k\beta(a_1\lambda_2 + a_1e_1) + e_2(\lambda_1+\lambda_2+e_1)\right\}}$$

(iv) The equilibrium point of co-existence $E^* = (S^*,\ I^*,\ Y^*,\ Z^*,\ T^*)$ where

$$Y^* = (\beta S^* - (a_1+e_2))\left(\frac{m+I^*}{p_2}\right) \qquad Z^* = \frac{qI^*(\beta S^* - a_1 - e_2)(m+S^*)}{(m+S^*)(a_2+e_4) - qp_3S^*},$$

$$T^* = \frac{a_1 I^*\left\{(m+S^*)(a_2+e_4) - qp_3S^*\right\} + a_2 qI^*(\beta S^* - a_1 - e_2)(m+S^*)}{(\lambda_1+\lambda_2+e_1)\left((m+S^*)(a_2+e_4) - qp_3S^*\right)}$$

where S^* is the positive root of $q\frac{p_1 S^* Y^*}{m+S^*} - e_3 Y^* + \lambda_2 T^* = 0$.

Stability analysis of the equilibrium points

Here, we study the existence criteria of the different equilibrium points. The trivial equilibrium point $E_T(0,0,0,0,0)$ of the system (1) will always exist and be unstable. The local stability is established by using the Jacobian matrix of the system (1), that is:

$$J=\begin{bmatrix} \frac{\partial f}{\partial S} & \frac{\partial f}{\partial I} & \frac{\partial f}{\partial Y} & \frac{\partial f}{\partial Z} & \frac{\partial f}{\partial T} \\ \frac{\partial g}{\partial S} & \frac{\partial g}{\partial S} & \frac{\partial g}{\partial S} & \frac{\partial g}{\partial S} & \frac{\partial g}{\partial S} \\ \frac{\partial h}{\partial S} & \frac{\partial h}{\partial S} & \frac{\partial h}{\partial S} & \frac{\partial h}{\partial S} & \frac{\partial h}{\partial S} \\ \frac{\partial k}{\partial S} & \frac{\partial k}{\partial S} & \frac{\partial k}{\partial S} & \frac{\partial k}{\partial S} & \frac{\partial k}{\partial S} \\ \frac{\partial j}{\partial S} & \frac{\partial j}{\partial S} & \frac{\partial j}{\partial S} & \frac{\partial j}{\partial S} & \frac{\partial j}{\partial S} \end{bmatrix}$$

This gives

$$J=\begin{bmatrix} r\left(1-\frac{S+I}{k}\right)-\frac{rS}{k}- & -\frac{\left(r+k\beta\right)S}{k} & -\frac{p_1S}{m+S} & -\frac{p_3S}{m+S} & \lambda_1 \\ \beta I-\frac{m\left(p_1Y+p_3Z\right)}{\left(m+S\right)^2} & & & & \\ & \beta S-(a_1+e_2) & & & \\ \beta I & -\frac{mp_2Y}{\left(m+I\right)^2} & -\frac{p_2I}{m+I} & 0 & 0 \\ \frac{mqp_1Y}{\left(m+S\right)^2} & 0 & q\frac{p_1S}{m+S}-e_3 & 0 & \lambda_2 \\ \frac{mqp_3Z}{\left(m+S\right)^2} & q\frac{mp_2Y}{\left(m+I\right)^2} & \frac{qp2I}{m+I} & \frac{qp_3S}{m+S}-(a_2+e_4) & 0 \\ 0 & a_1 & 0 & a_2 & -(\lambda_1+\lambda_2+e_1) \end{bmatrix} \quad (4)$$

By linearizing the system at the point E_T we obtain:

$$J_{ET}=\begin{bmatrix} r & 0 & 0 & 0 & \lambda_1 \\ 0 & -(a_1+e_2) & 0 & 0 & 0 \\ 0 & 0 & -e_3 & 0 & \lambda_2 \\ 0 & 0 & 0 & -(a_2+e_4) & 0 \\ 0 & a1 & 0 & a_2 & -(\lambda_1+\lambda_2+e_1) \end{bmatrix} \quad (5)$$

The corresponding Eigen values are:

$$\begin{bmatrix} r \\ -e_3 \\ -a_1-e_2 \\ -a_2-e_4 \\ -\lambda_1-\lambda_2-e_1 \end{bmatrix}$$

Four of the Eigen values are negative and one is negative. So the trivial equilibrium point E_T is unstable.

With linearization of the system at the point E_A, we obtain the Eigen values:

$$\begin{bmatrix} -r \\ -\lambda_1-\lambda_2-e_1 \\ k\beta-a_1-e_2 \\ \dfrac{qp_1k-e_3(m+k)}{m+k} \\ \dfrac{qp_3k-(a_2+e_4)(m+k)}{m+k} \end{bmatrix} \quad (6)$$

Therefore the axial equilibrium point E_A will have stable manifold in the direction I , if:

(i) $k\beta-(a_1+e_2)<0$,

(ii) $\dfrac{qp_1k-e_3(m+k)}{m+k}<0$,

(iii) $\dfrac{qp_3k-(a_2+e_4)(m+k)}{m+k}$.

Next, we investigate the boundary equilibrium points E_{B1} and E_{B2}.

To determine the stability of E_{B1}, we substitute the point E_{B1} into Equation 4 to obtain

$$JE_{B1}=\begin{bmatrix} -b_1 & -b_2 & -\dfrac{e3}{q} & -b_3 & \lambda_1 \\ 0 & -b_4 & 0 & 0 & 0 \\ b_5 & 0 & 0 & 0 & \lambda_2 \\ 0 & b_6 & 0 & b_7 & 0 \\ 0 & a_1 & 0 & a_2 & -(\lambda_1+\lambda_2+e_1) \end{bmatrix} \quad (7)$$

where

$$b_1=re_3\frac{\left[kqp_1-(m+k)e_3-mqp_1\right]}{qp_1k(qp_1-e_3)},\ b_2=me_3\left(\frac{r+k\beta}{qp_1-e_3}\right),$$

$$b_4=\frac{\beta me_3}{qp_1-e_3}-(a_1+e_2)-\frac{qp_2r}{k(qp_1-e_3)^2}\left(kqp_1-e_3(k+m)\right),$$

$$b_5=\frac{q^2p_2r}{k(qp_1-e_3)}\left(kqp_1-e_3(k+m)\right),$$

$$b_6=\frac{q^2p_2r}{k(qp_1-e_3)}\left(kqp_1-e_3(k+m)\right),\ b_7=\frac{p_3e_3}{p_1}-(a_2+e_4).$$

The corresponding Eigen values are:

$$\begin{bmatrix} b_7 \\ -b_4 \\ -(\lambda_1+\lambda_2+e_1) \\ -\dfrac{1}{2}qb_1-\dfrac{\sqrt{q^2b1^2-4qb5e_3}}{2q} \\ -\dfrac{1}{2}qb_1+\dfrac{\sqrt{q^2b1^2-4qb5e_3}}{2q} \end{bmatrix}$$

where

$$b_4=\frac{\beta me_3}{qp_1-e_3}-(a_1-e_2)-\frac{qp_2r}{k(qp_1-e_3)^2}\left(kqp_1-e_3(k+m)\right)$$

$$b_7=\frac{p_3e_3}{p_1}-(a_2+e_4)$$

E_{B1} will be stable in the direction of Y if $b_7<0$ that is $\frac{e_3p_3}{p_1(a_2+e_4)}<1$ and it will exist if $q^2b1^2>4qb5e_3$. The ratio $\frac{e_3p_3}{p_1(a_2+e_4)}$ can be considered as reproduction number R_0 .

Next, we investigate the disease free equilibrium point E_{B2}. We substitute E_{B2} into Equation 4 and obtain

$$J_{E_{B2}}=\begin{bmatrix} b_1 & -b_2 & -b_3 & -b_4 & \lambda_1 \\ b_5 & b_6 & -b_7 & 0 & 0 \\ 0 & 0 & b_8 & 0 & \lambda_2 \\ 0 & 0 & b_9 & b_{10} & 0 \\ 0 & a_1 & 0 & a_2 & \lambda_1+\lambda_2+e_1 \end{bmatrix} \quad (8)$$

Where $b_1=r\left(1-\frac{S+I}{k}\right)-rS-\beta I$, $b_2=S(r+k\beta)$,

$$b_3=\frac{p_1S}{m+S},\ b_4=\frac{p_3S}{m+S},\ b_5=\beta I,\ b_6=\beta S-(a_1+e_2),$$

$$b_7=\frac{p_1I}{m+I},\ b_8=\frac{qp_1S}{m+S}-e_3,\ b_9=\frac{qp_2I}{m+I},$$

$$b_{10}=\frac{qp_3S}{m+S}-(a_2+e_4)$$

To obtain the Eigen values of Equation 8, we evaluate the determinant:

$$\begin{vmatrix} b_1 - x & -b_2 & -b_3 & -b_4 & \lambda_1 \\ b_5 & b_6 - x & -b_7 & 0 & 0 \\ 0 & 0 & b_8 - x & 0 & \lambda_2 \\ 0 & 0 & b_9 & b_{10} - x & 0 \\ 0 & a_1 & 0 & a_2 & \lambda_1 + \lambda_2 + e_1 - x \end{vmatrix} = 0$$

to obtain $x^5 + Ax^4 + Bx^3 + Cx^2 + Dx + E = 0$

where $A = \lambda_1 + \lambda_2 + e_1 - b_8 - b_{10} - b_6 - b_1$,

$$B = b_8 b_{10} - (b_1 + b_8 + b_6 + b_{10})\lambda_1 + b_1 b_{10} - (b_1 + b_8 + b_6 + b_{10})\lambda_2 - (b_1 + b_8 + b_6 + b_{10})e_1 + b_6 b_{10} + b_1 b_8 + b_2 b_5 + b_1 b_6 + b_6 b_8,$$

$$C = (b_1 b_8 + b_6 b_{10} + b_1 b_{10} + b_6 b_8 + b_2 b_5 + a_1 b_5 + b_8 b_{10} + b_1 b_6)\lambda_1 + (b_1 b_{10} + b_1 b_8 + b_6 b_8 + b_2 b_5 + b_8 b_{10} + b_6 b_{10} + b_1 b_6 - a_2 b_9)\lambda_2 + (b_1 b_{10} + b_1 b_8 + b_6 b_8 + b_6 b_{10} + b_2 b_5 + b_1 b_6 + b_8 b_{10})e_1 - (b_6 b_8 b_{10} + b_1 b_8 b_{10} + b_2 b_5 b_8 b_{10} + b_1 b_6 b_8 + b_1 b_6 b_{10}),$$

$$D = (b_1 b_8 b_{10} + a_1 b_5 b_8 - b_2 b_5 b_{10} - b_6 b_1 b_{10} - b_6 b_8 b_{10} - b_2 b_5 b_8 + a_1 b_5 b_{10} - b_1 b_6 b_8`)\lambda_1 + \begin{pmatrix} a_1(b_3 b_5 - b_1 b_7 - b_7 b_{10}) - b_1 b_6 b_{10} - b_6 b_8 b_{10} - b_1 b_8 b_{10} + a_2(b_6 b_9 + b_1 b_9) - b_1 b_6 b_8 \\ -b_2 b_5 b_{10} - b_2 b_5 b_8 \end{pmatrix}\lambda_2 - (b_2 b_5 b_8 + b_1 b_6 b_{10} b_6 b_8 b_{10} + b_1 b_6 b_8 + b_2 b_5 b_{10} + b_1 b_8 b_{10})e_1 + b_2 b_5 b_8 b_{10} + b_1 b_6 b_{10}$$

$$E = (b_1 b_6 b_8 b_{10} + b_2 b_5 b_8 b_{10} - a_1 b_5 b_8 b_{10})\lambda_1 + (b_2 b_5 b_8 b_{10} + a_1(b_1 b_7 b_{10} + b_4 b_5 b_9) + b_1 b_6 b_8 b_{10} - b_1 b_6 b_9 - a_2 b_2 b_5 b_9)\lambda_2 + (b_2 b_5 b_8 b_{10} + b_1 b_6 b_{10})e_1$$

Using Hurwitz criteria, E_{B2} will be locally asymptotically stable, if:

1. $A > 0$
2. $AB - C > 0$
3. $ABC + AE - A^2 D - C^2 > 0$
4. $(CD - BE)(AB - C) - (AD - E)^2 > 0$
5. $D*\left[(CD - BE)(AB - C) - (AD - E)^2\right] > 0$

and unstable otherwise.

The stability analysis around the coexistence equilibrium point is determined as follows. We consider the Jacobian matrix:

$$J = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22} & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44} & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55} \end{bmatrix} \quad (9)$$

Where

$$a_{11} = r\left(1 - \frac{2S^* + I^*}{k}\right) - \beta I^* - \frac{m(p_1 Y^* + p_3 Z^*)}{(m + S^*)^2},$$

$$a_{12} = -\left(\frac{r + k\beta}{k}\right)S^*, \quad a_{13} = -\frac{p_1 S^*}{m + S^*}, \quad a_{14} = -\frac{p_3 S^*}{m + S^*},$$

$$a_{15} = \lambda_1, \quad a_{21} = \beta I^*, \quad a_{22} = \beta S^* - (a_1 + e_2) - \frac{p_2 Y^*}{(m + I^*)^2},$$

$$a_{23} = -\frac{p_1 I^*}{m + I^*}, \quad a_{24} = 0, \quad a_{25} = 0, \quad a_{31} = \frac{m p_1 Y^*}{(m + S^*)^2},$$

$$a_{32} = 0, \quad a_{33} = \frac{q p_1 S^*}{m + S^*} - e_3, \quad a_{34} = 0, \quad a_{35} = \lambda_2,$$

$$a_{41} = \frac{q p_3 Z^*}{(m + I^*)} - \frac{q p_2 Y^* I^*}{(m + S^*)^2}, \quad a_{42} = \frac{q p_2 Y^*}{(m + S^*)} - \frac{q p_3 S^* Z^*}{(m + I^*)^2},$$

$$a_{43} = \frac{q p_2 I^*}{m + S^*}, \qquad a_{44} = \frac{q p_3 S^*}{(m + I^*)} - (a_2 + e_4),$$

$a_{45}=0$, $a_{51}=0,\ a_{52}=a_1,\ a_{53}=0,\ a_{54}=a_2,$
$a_{55}=-(\lambda_1+\lambda_2+e_1)$

We then evaluate the determinant:

$$\begin{vmatrix} a_{11}-\lambda & a_{12} & a_{13} & a_{14} & a_{15} \\ a_{21} & a_{22}-\lambda & a_{23} & a_{24} & a_{25} \\ a_{31} & a_{32} & a_{33}-\lambda & a_{34} & a_{35} \\ a_{41} & a_{42} & a_{43} & a_{44}-\lambda & a_{45} \\ a_{51} & a_{52} & a_{53} & a_{54} & a_{55}-\lambda \end{vmatrix}=0$$

to get $\lambda^5+A\lambda^4+B\lambda^3+C\lambda^2+D\lambda+E=0$. (10)

Using the Hurwitz criteria, the coexistence equilibrium point will be stable, if:

1. $A>0$,
2. $AB-C>0$,
3. $ABC+AE-A^2D-C^2>0$,
4. $H4=(CD-BE)(AB-C)-(AD-E)^2>0$,
5. $D^*\left[(CD-BE)(AB-C)-(AD-E)^2\right]>0$,

and unstable otherwise.

Global stability analysis

We now perform a global stability analysis of the system (Equation 1) around the positive equilibrium point $E(S^*,\ I^*,\ Y^*,\ Z^*,\ T^*)$ of the coexistence. We consider the following theorem on the Lyapunov function U.

Theorem 3

Let

$$U=\frac{(S-S^*)^2}{2}+\frac{\delta_1}{2}(I-I^*)^2+\frac{\delta_2}{2}(Y-Y^*)^2+\frac{\delta_3}{2}(Z-Z^*)^2+\frac{\delta_4}{2}(T-T^*)^2$$

where $\delta_1,\ \delta_2,\ \delta_3,\ \delta_4>0$ are to be chosen properly such that $U'(E)=0$

where $E(S^*,\ I^*,\ Y^*,\ Z^*,\ T^*)$ and
$U=(S,\ I,\ Y,\ Z,\ T)>0\ \ \forall S,I,Y,\ Z,\ T\,|\{E\}$

The time derivative of U is $\frac{dU}{dt}\le 0$ $\forall S,\ I,\ Y,\ Z,\ T\in\Gamma^+$.

It then follows that $\frac{dU}{dt}=0$, $\forall S^*,\ I^*,\ Y^*,\ Z^*,\ T^*\in\Gamma^+$ implies that E^* of the system is Lyapunov stable and $\frac{dU}{dt}<0,\ \ \forall S,\ I,\ Y,\ Z,\ \mathrm{T}\in\Gamma^+$ near E^* implies that E^* is globally stable.

Proof:

$$\frac{dU}{dt}=(S-S^*)\frac{dS}{dt}+\delta_1(I-I^*)\frac{dI}{dt}+\delta_2(Y-Y^*)\frac{dY}{dt}+\delta_3(Z-Z^*)\frac{dZ}{dt}+\delta_4(T-T^*)\frac{dT}{dt}$$

Now by substituting equations of the model (Equation 1), we get:

$$\frac{dU}{dt}=(S-S^*)\left[rS\left(1-\frac{S+I}{k}\right)-\beta SI-\frac{p_1SY}{m+S}-\frac{p_3SZ}{m+S}+\lambda_1T\right]+\delta_1(I-I^*)\left[\beta SI-(a_1+e_2)I-\frac{p_2IY}{m+I}\right]$$
$$+\delta_2(Y-Y^*)\left[q\frac{p_1SY}{m+S}-e_3Y+\lambda_2T\right]+\delta_3(Z-Z^*)\left[q\frac{p_2IY}{m+I}+q\frac{p_3SZ}{m+S}-(a_2+e_4)Z\right]$$
$$+\delta_4(T-T^*)[a_1I+a_2Z-(\lambda_1+\lambda_2+e_1)T] \quad (11)$$

Then Equation 11 becomes:

$$\frac{dU}{dt}=\left(S-S^*\right)\left[\left\{r\left(1-\frac{S+I}{k}\right)-\beta I-\frac{p_1Y}{m+S}-\frac{p_3Z}{m+S}+\frac{\lambda_1T}{S}\right\}\left\{S-S^*\right\}\right]+\delta_1\left(I-I^*\right)\left[\left\{\beta S-(a_1+e_2)-\frac{p_2Y}{m+I}\right\}\left\{I-I^*\right\}\right]$$

$$+\delta_2\left(Y-Y^*\right)\left[\left\{q\frac{p_1S}{m+S}-e_3+\frac{\lambda_2T}{Y}\right\}\left\{Y-Y^*\right\}\right]+\delta_3\left(Z-Z^*\right)\left[\left\{q\frac{p_2IY}{Z(m+I)}+q\frac{p_3S}{m+S}-(a_2+e_4)\right\}\left\{Z-Z^*\right\}\right]$$

$$+\delta_4\left(T-T^*\right)\left[\left\{\frac{a_1I+a_2Z}{T}-(\lambda_1+\lambda_2+e_1)\right\}\left\{T-T^*\right\}\right]$$

By rearranging, we obtain:

$$\frac{dU}{dt}=-\left(S-S^*\right)^2\left[r\left(-1+\frac{S+I}{k}\right)+\beta I+\frac{p_1Y}{m+S}+\frac{p_3Z}{m+S}-\frac{\lambda_1T}{S}\right]-\delta_1\left(I-I^*\right)^2\left[-\beta S+(a_1+e_2)+\frac{p_2Y}{m+I}\right]$$

$$-\delta_2\left(Y-Y^*\right)^2\left[-q\frac{p_1S}{m+S}+e_3-\frac{\lambda_2T}{Y}\right]-\delta_3\left(Z-Z^*\right)^2\left[-q\frac{p_2IY}{Z(m+I)}-q\frac{p_3S}{m+S}+(a_2+e_4)\right]$$

$$-\delta_4\left(T-T^*\right)^2\left[\frac{-a_1I-a_2Z}{T}+(\lambda_1+\lambda_2+e_1)\right]$$

Thus, it is possible to set δ_1, δ_2, δ_3, δ_4 such that $U'\le 0$ and endemic equilibrium point is globally stable. It is noted that the parameters k, m and q play important roles in controlling the stability aspects of the system.

Numerical simulation

In order to illustrate some of the analytical results of the study, numerical simulations of the model (1) are carried out using Rung-Kutta iteration scheme with a set of reasonable parameter values given in Table 1. These parameter values are mainly hypothetical. They are chosen following realistic ecological observations.

In order to verify the theoretical predictions of the model, we present numerical simulation of some solutions of the systems by comparing the numerical simulation figures of the model with infected predator and treatment group (left panel) and without infected predator and treatment. The basic eco-epidemiological model is achieved by setting the following parameter values of the present model to zero: $a_1=a_2=\lambda_1=\lambda_2=p_3=e_1=e_4=0$.

Figure 2a shows that the variation of the susceptible prey population oscillates at high to low amplitude due to the treatment until a steady state is attained, while Figure 2b oscillates with high amplitude then changes to low amplitude, and abruptly oscillations increase with high amplitude until the steady state reached. Figure 3a shows that the infected prey population increases and then decreases due to the effect of treatment. The population oscillates at high amplitude at

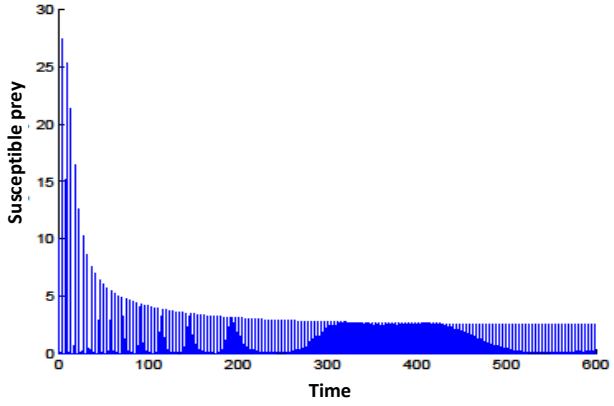

Figure 2a. Prey population around disease free parameter values in Table 1.

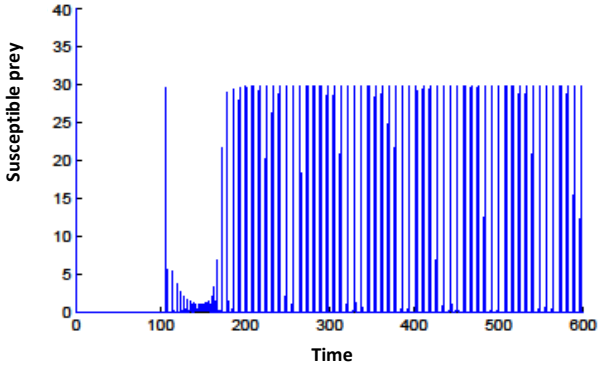

Figure 2b. Prey population around disease free parameter values in Table 1 except $a_1=a_2=\lambda_1=\lambda_2=p_3=e_1=e_4=0$.

Table 1. Parameter value used in numerical simulation.

Symbol	Value	Source	Symbol	Value	Source
r	11.2	Mukhopadhyay and Bhattacharyya (2009)	Λ_2	0.002	Estimated
k	30	Mukhopadhyay and Bhattacharyya (2009)	a_1	0.01	Estimated
β	1.2	Mukhopadhyay and Bhattacharyya (2009)	a_2	0.03	Estimated
p_1	0.4	Mukhopadhyay and Bhattacharyya (2009)	e_1	0.05	Estimated
p_2	0.6	Mukhopadhyay and Bhattacharyya (2009)	e_2	0.4	Mukhopadhyay and Bhattacharyya (2009)
p_3	0.2	Estimated	e_3	0.08	Mukhopadhyay and Bhattacharyya (2009)
m	0.5	Mukhopadhyay and Bhattacharyya (2009)	e_4	0.01	Estimated
λ_1	0.001	Estimated	q	0.25	Mukhopadhyay and Bhattacharyya (2009)

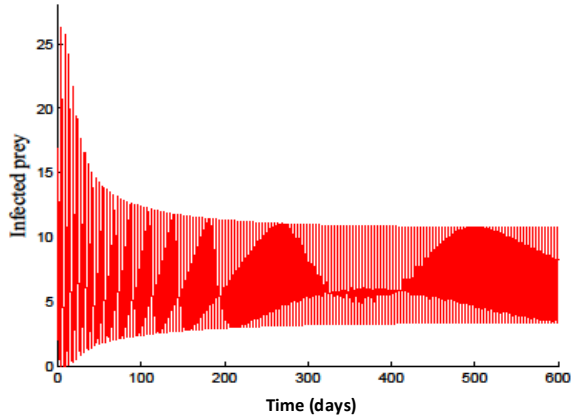

Figure 3a. Infected prey population around disease free, parameter values in Table 1.

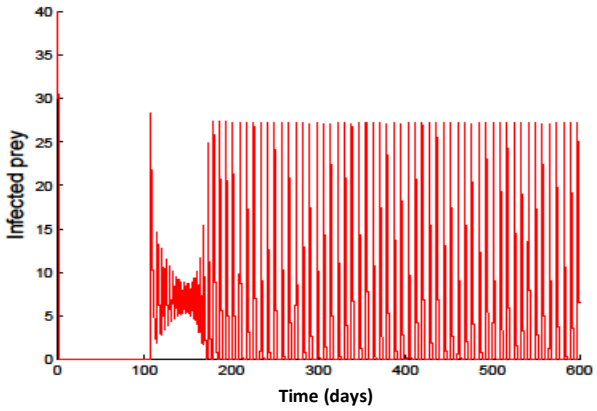

Figure 3b. Infected prey population around disease free, parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$.

the beginning and then oscillates at low amplitude until the steady state is attained. The recovery rates λ_1 and λ_2 increase the up oscillations of the susceptible as the result of the treatment. Figure 3b shows high variations of the oscillations and then the oscillations are highly increased with high amplitude until the steady state is attained.

Figure 4a shows how the predator population varying with time depend on the interaction with prey population.

The sharply decrease of the population in Figure 4a occurs as the result of high number of infected prey that are easily captured by the predator. The high death rate of the predator also contributes in the decline of the population, although the recovery rate λ_2 leads the population to reach its steady state. Figure 4b shows that untreated population decreases to its optimal point then slowly oscillates with low amplitude to its steady state.

Figure 5a 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 5b 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. Thereafter, it decline by low oscillation amplitude and eventually reached the steady state.

Figure 6a shows the sharp decrease of population as the death rate of the predator increases. The population oscillates from high to low amplitudes reaching its steady state, while Figure 6b shows the decline of the oscillations to the equilibrium point and then slowly oscillates up and down with high and low amplitudes, respectively.

Figure 7a shows how the infected prey population start to oscillate from high amplitude to low amplitude attaining its steady state, while in Figure 7b the population declines with the oscillation from high to low amplitudes and reaches a temporary steady state, then increases and starts to oscillate with high and low amplitudes due to the disease.

Figure 8a shows low oscillations amplitude compared to high amplitudes in Figure 8b of the population, as the result of the interaction with the infected prey population becomes high. Figure 8a did not give good results, since the purpose is to control the disease. Then modified parameters values are used to obtain susceptible predator population above the infected predator population as seen in Figure 8c.

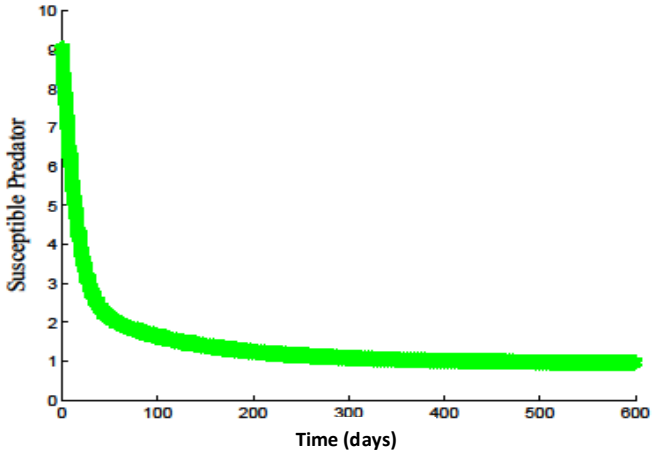

Figure 4a. Predator population around disease free, parameter values in Table 1.

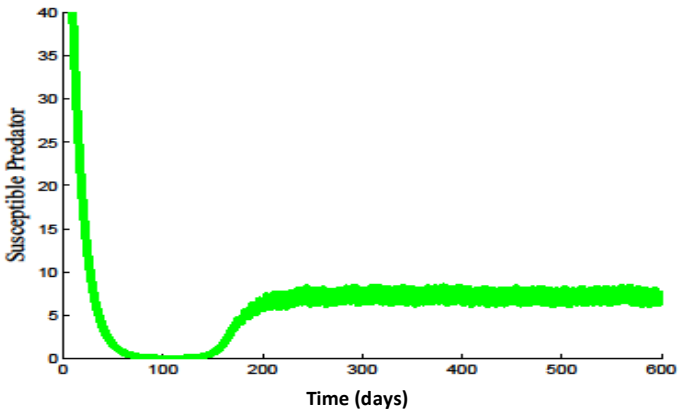

Figure 4b. Predator population around disease free, parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$.

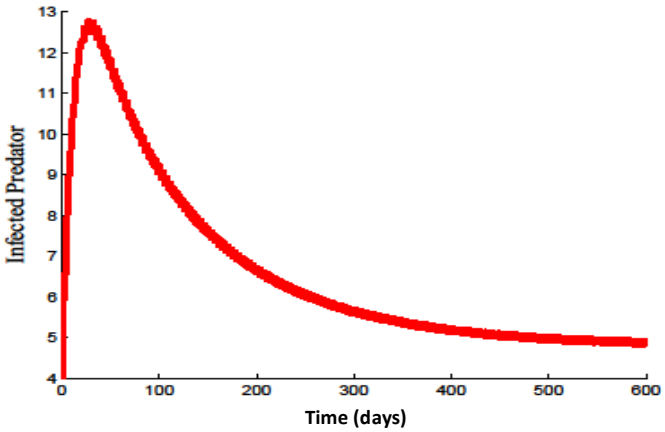

Figure 5a. Infected predator population around disease free, parameter values in Table 1.

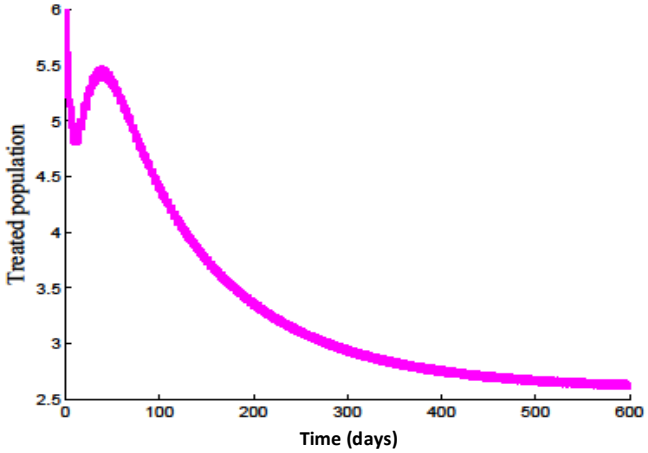

Figure 5b. Treated population for the model (1) with the parameter values in Table 1.

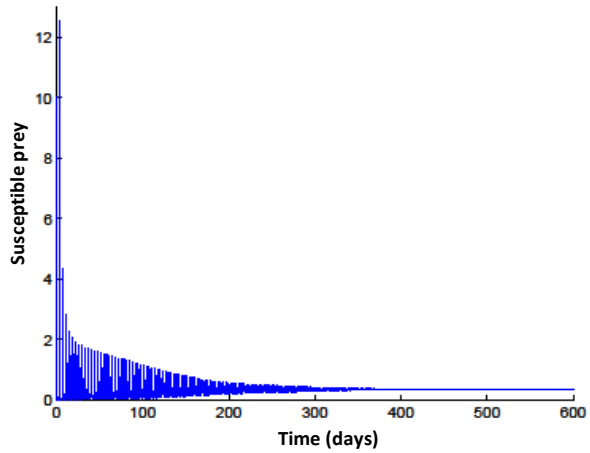

Figure 6a. Prey population parameter values in Table 1 with for $e_3 \geq 0.2$.

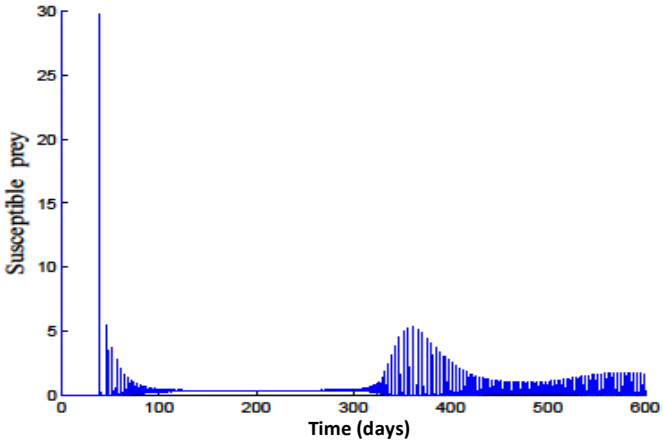

Figure 6b. Infected prey population parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$ and e_3=0.17.

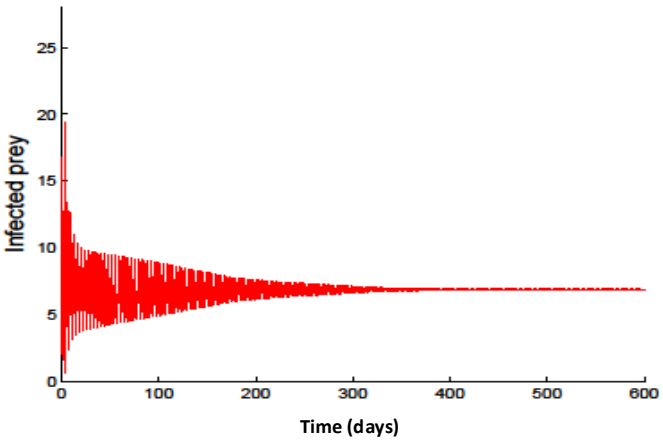

Figure 7a. Variation in the infected prey population for the model (1) with the parameter values in the Table 1 with $e_3 \geq 0.2$.

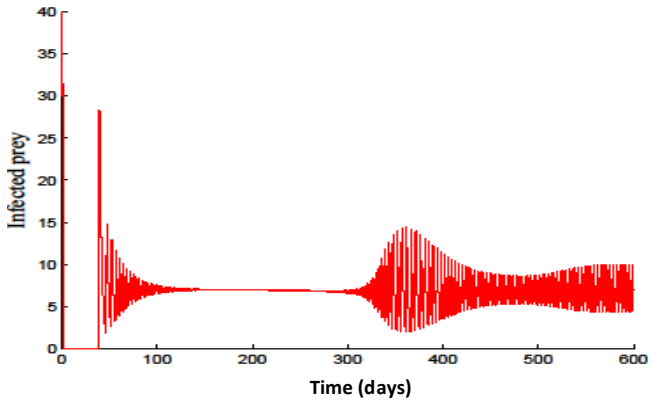

Figure 7b. Variation in the infected prey population for the model (1) with the parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$ and $e_3 = 0.17$.

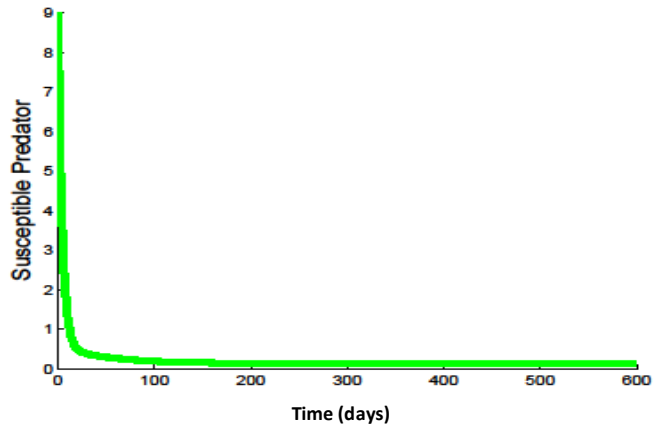

Figure 8a. Variation in the susceptible predator population for the model (1) with the parameter values in Table 1 except $e_3 \geq 0.2$.

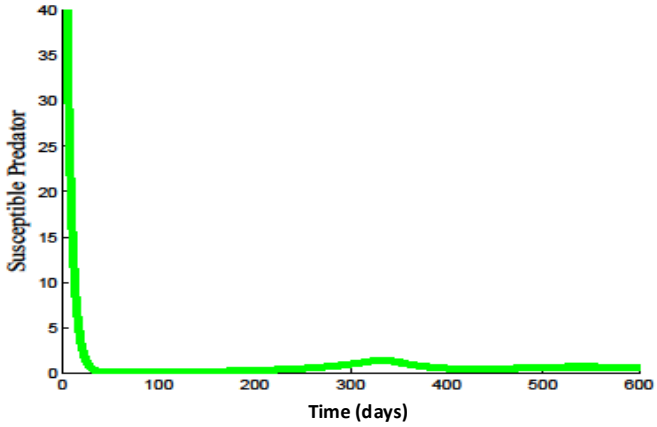

Figure 8b. Variation in the susceptible predator population for the model (1) with the parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$ and $e_3 = 0.17$.

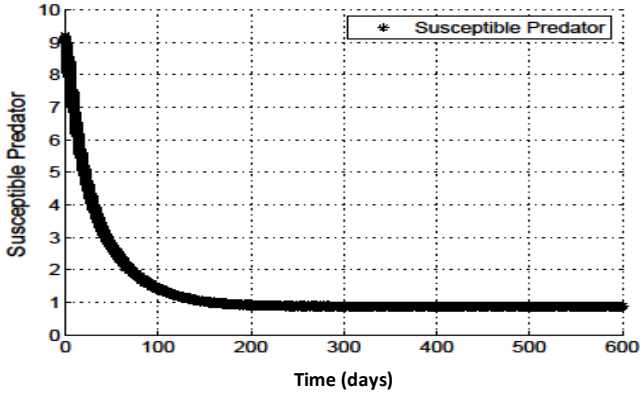

Figure 8c. Susceptible predator population for the model (1) with the parameter values in the Table 1 except $e_4 = 0.9,\ a_2 = 0.5,\ p_1 = 0.6$.

Phase portrait of the model

A numerical study with the predator efficiency q reveals an interesting result, namely that as the predator conversion efficiency q decreases; the system dynamics changes the behaviour as the result of the incorporated treatment parameters. This is shown in Figures 9 to 12.

Conclusions

This 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

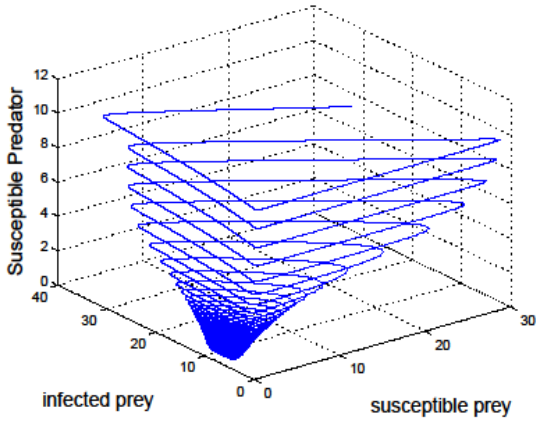

Figure 9a. Variation of prey, infected prey and predator for the model (1) with the parameter values in Table 1.

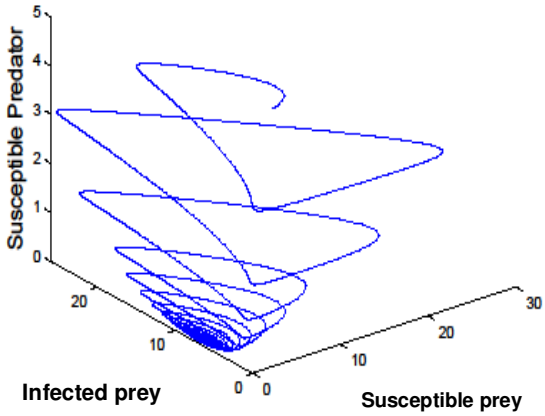

Figure 9b. Variation of prey, infected prey and predator the model (1) with the parameter values in Table 1 except $a_1 = a_2 = \lambda_1 = \lambda_2 = p_3 = e_1 = e_4 = 0$.

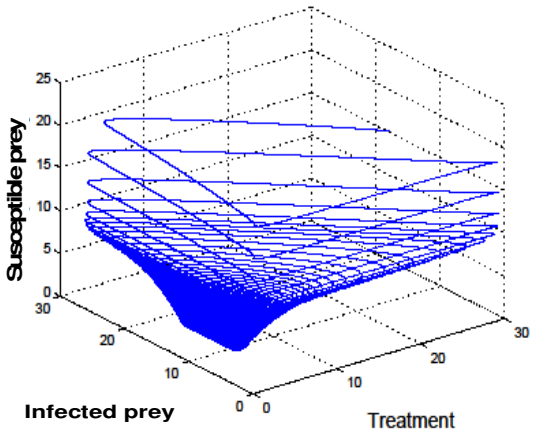

Figure 10a. Variation of prey, infected prey and treatment for the model (1) with the parameter values in the Table 1.

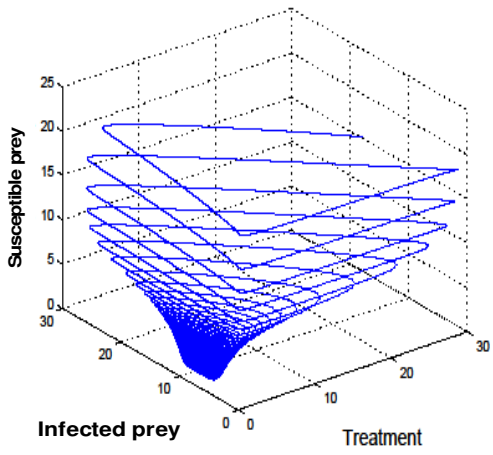

Figure 10b. Variation of prey, infected prey and treatment for the model (1) with the parameter values in the Table 1 except $q = 0.15$.

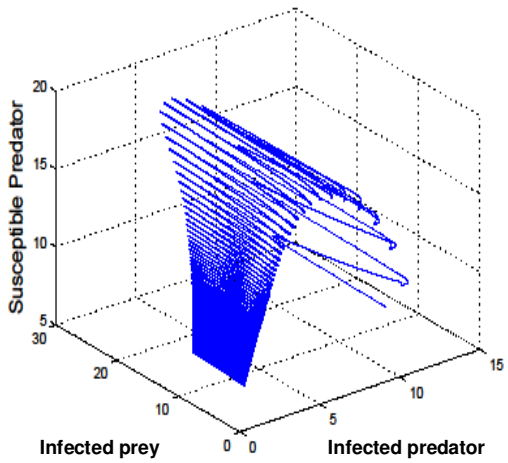

Figure 11a. Variation of infected predator, infected prey and susceptible predator for the model (1) with the parameter values in Table 1.

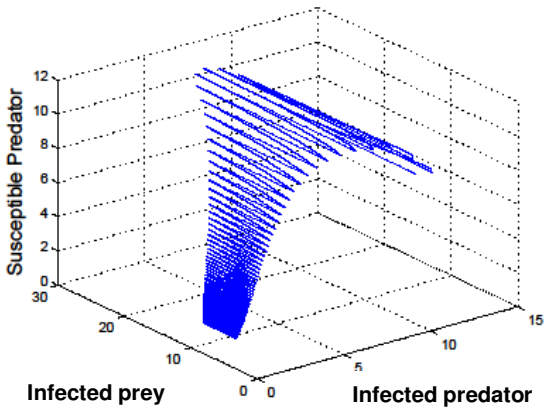

Figure 11b. Variation of infected predator, infected prey and susceptible predator for the model (1) with the parameter values in Table 1 except $q = 0.15$

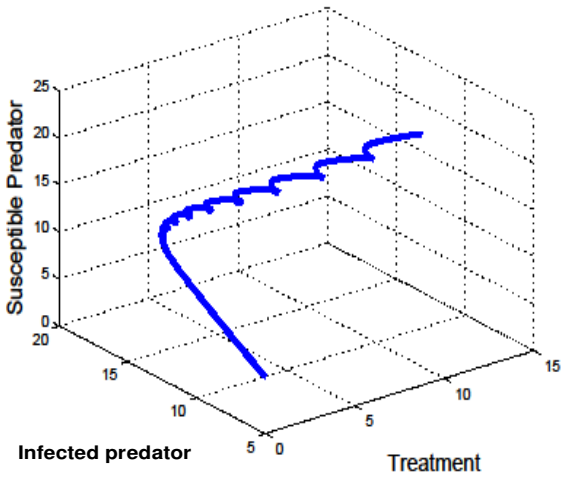

Figure 12a. Variation of susceptible predator, infected predator and treatment for the model (1) with the parameter values in Table 1.

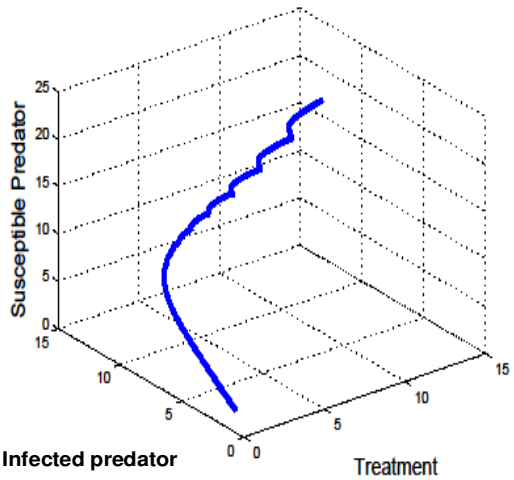

Figure 12b. Variation of susceptible predator, infected predator and treatment for the model (1) with the parameter values in Table 1 except $q = 0.15$.

population. The boundedness and positivity of the system seem to hold which implies that the system is biologically well behaved. Disease free equilibrium points were obtained and their stability analysis investigated. The existence of an interior equilibrium with predator and prey coexisting and both endemic is interesting biologically. 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 can be concluded that the disease can be eradicated in a population through treatment.

REFERENCES

Anderson R, May R (1986). The Invasion, Persistence and Spread of Infectious-Diseases within Animal and Plant-Communities. Philos. Trans. Royal Soc. London Ser. B-Biol. Sci. 314:533-570.

Arino O, El Abdllaoui A, Mikram J, Chattopadhyay J (2004). Infection in prey population may act as a biological control in ratio-dependent predator-prey models. Nonlinearity 17: 1101-1116.

Beltrami E, Carroll TO (1994). Modelling the role of viral disease in recurrent phytoplankton blooms. J. Math. Biol. 32:857-863.

Chattopadhyay J, Arino O (1999). A predator-prey model with disease in the prey. Nonlinear Anal.- Theory Method Appl. 36:747-766.

Dobson AP (1988). The population biology of parasite-induced changes in host behaviour. Q. Rev. Biol. 63:139-165.

Hadeler KP, Freedman HI (1989). Predator-prey populations with parasitic infection. J. Math. Biol. 27: 609-631.

Hilker FM, Schmitz K (2008). Disease-induced stabilization of predator-prey oscillations. J. Theor. Biol. 255:299-306.

Hsieh Y-H, Hsiao C-K (2008). Predator-prey model with disease infection in both populations. Math. Med. Biol. 25:247-266.

Mukhopadhyay B, Bhattacharyya R (2009). Role of predator switching in an eco-epidemiological model with disease in the prey. Elsevier-Ecol. Model. 220:931-939.

Venturino E (1994). The influence of diseases on Lotka-Volterra systems. Rocky Mount. J. Math. 24:381-402.

Venturino E (1995). Epidemics in predator–prey models: Disease among the prey. In: Arino O, Axelrod D, Kimmel M, Langlais M (Eds.), Mathematical Population Dynamics: Analysis of Heterogeneity, Vol. I: Theory of Epidemics. Wuertz Publishing Ltd, Winnipeg, Canada, pp. 381-393.

Venturino E (2002). Epidemics in predator-prey model with disease in the predators. IMA J. Math. Appl. Med. Biol. 19:185.

Venturino E (2006) On epidemics crossing the species barriers in interacting population models. Varahmihir J. Math. Sci. 6:247-263.