An Eco-Epidemiological Model with Nonlinear Incidence and Infective Prey Treatment Class

Peter Tengaa\textsuperscript{1}, Daniel O. Makinde\textsuperscript{2}, Estomih S. Massawe\textsuperscript{1}

\textsuperscript{1}Department of Mathematics, University of Dar es Salaam, P. O. Box 35062, Dar es Salaam, Tanzania
\textsuperscript{2}Faculty of Military Science, Stellenbosch University, Stellenbosch University, Private Bag X2, Saldanha Bay, 7395, South Africa

Abstract—In this work, an eco-epidemiological model with nonlinear incidence and infective prey treatment class has been proposed and analysed. The model is found to be well posed and solutions exist in a feasible region where equilibrium points are obtained and their stabilities are investigated. It integrates the effect of treatment of infective prey population. The equilibrium points are found to be locally stable and globally asymptotically stable under certain conditions. The model analysis shows that treatment of infective prey has a potential positive impact to the population which saves population from extinction. Finally, numerical simulations are performed. It is observed that the increase of treatment rate tends to increase both susceptible prey as well as predator population.

Keywords—Eco-Epidemiological, Nonlinear Incidence, Infective Prey, Treatment Class

I. INTRODUCTION

In recent years there have emerged studies on infectious diseases dealing with four predator prey models with infectious diseases which focus on the influence of infectious diseases on predator-prey ecological interactions \cite{3}. They include the possibility that the infectious diseases can persist in the predator population and can be acquired by predators during the predation process. Some studies have been undertaken to develop an eco-epidemiological mathematical model with nonlinear incidence rate. The studies have focused on issues like stability analysis of an eco-epidemiological model incorporating a prey refuge with disease in the prey population for which it is very important for the biological control of a prey. However, increasing the amount of refuge can increase prey densities and lead to population outbreaks \cite{1}. Also some studies have focused on the role of predator switching in an eco-epidemiological model with disease in the prey, for which they investigated the role of switching on disease dynamics and incorporate the switching behaviour of the predator. However, among the few studies that have been done on eco-epidemiological models, more effort has been put on eco-epidemiological models with nonlinear incidence rate combining a prey-predator model with an eco-epidemiological model whereby the disease in prey is modelled by a susceptible-infected epidemic system \cite{3}. The results revealed that the infective rate has vital role on the dynamics of the system upon which decrease in the rate of infection leads to the decrease in infective population, hence, leading to the extinction of infective population. In this paper, it is intended to extend the work by \cite{3} by incorporating the aspect of infective prey treatment class in the deterministic model of prey-predator population.

II. MODEL FORMULATION AND ANALYSIS

A nonlinear mathematical model is proposed and analysed to study the susceptible and infective prey interacting with susceptible predator for which the treatment is imposed to the
infective prey class. The basic eco-epidemiological model consists of two populations: the susceptible prey population and infected prey population interacting with predator population.

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

i. In the absence of disease, the prey population grows logistically with the carrying capacity and intrinsic growth rate constant,

ii. The disease is transmitted from infected prey to susceptible prey by contact according to nonlinear incidence rate of the form [3],

iii. Susceptible prey can reproduce reaching to its carrying capacity and the infected prey can recover through treatment,

iv. The predator species consumes the susceptible prey species as well as infected according to the modified Holling type II functional response. However, in the absence of prey population, the predator population decays exponentially.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Definitions of Symbols used frequently</th>
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<tbody>
<tr>
<td>Parameter /Variable</td>
<td>Description</td>
</tr>
<tr>
<td>$r$</td>
<td>Intrinsic growth rate constant $r &gt; 0$</td>
</tr>
<tr>
<td>$K$</td>
<td>$K$-carrying capacity of the environment</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>$\lambda$ - Represent the infected rate,</td>
</tr>
<tr>
<td>$\alpha_1$</td>
<td>Represent maximum predation rate of $S$</td>
</tr>
<tr>
<td>$\alpha_2$</td>
<td>Represent maximum predation rate of $I$</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Is the half saturation constant</td>
</tr>
<tr>
<td>$m$</td>
<td>Represent the predator preference rate between $S$ and $I$</td>
</tr>
<tr>
<td>$\theta_1$</td>
<td>are the conversion rates of $S$</td>
</tr>
<tr>
<td>$\theta_2$</td>
<td>are the conversion rates of $I$</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>Represent the death rate of $I$ due to the diseases</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>Represent the natural death rate of $Y$ due to diseases</td>
</tr>
<tr>
<td>$a$</td>
<td>The treatment rate of the infected prey population</td>
</tr>
<tr>
<td>$\rho$</td>
<td>Rate at which the infected prey get immune (recovered after treatment)</td>
</tr>
<tr>
<td>$\mu_3$</td>
<td>Represent the natural death rate of $T$ due to diseases</td>
</tr>
<tr>
<td>$S$</td>
<td>Susceptible Prey</td>
</tr>
<tr>
<td>$I$</td>
<td>Infective Prey</td>
</tr>
<tr>
<td>$Y$</td>
<td>Susceptible Predator</td>
</tr>
<tr>
<td>$T$</td>
<td>Treatment class</td>
</tr>
</tbody>
</table>

In view of the above considerations and assumptions, the dynamics of such a model will be governed by the following system of equations:

$$
\frac{dS}{dt} = rS \left( 1 - \frac{S}{K} \right) - \frac{\lambda IS}{1 + I} - \frac{\alpha_1 SY}{\beta + S + mI} + \rho T ,
$$

$$
\frac{dI}{dt} = \frac{\lambda IS}{1 + I} - \frac{\alpha_2 IY}{\beta + S + mI} - \mu_1 I - aI ,
$$

$$
\frac{dY}{dt} = \frac{\theta_1 S + \theta_2 I}{\beta + S + mI} Y - \mu_3 Y ,
$$

(1)
\[
\frac{dT}{dt} = aI - (\mu_1 + \rho)T .
\]

where

\[
S_0 > 0, \ I_0 > 0, \ Y_0 > 0, \ T_0 > 0 .
\]

### III. MODEL ANALYSIS

The model (1) will be analysed qualitatively to get insight into its dynamical features which might give better understanding of the effect of infective prey treatment in an eco-epidemiological model.

#### 3.1 Boundedness of the Solutions

In this section we show that the system (1) with initial conditions is bounded. This will be established by considering the following Theorem.

**Theorem 1**

All the solutions of the system (1) are uniformly bounded [4].

**Proof**

Let

\[ W = S + I + Y + T . \]

Then the time derivative of \( W \) is given by

\[
\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dY}{dt} + \frac{dT}{dt} .
\]  

(2)

This gives

\[
\frac{dW}{dt} = rS \left(1 - \frac{S}{K}\right) - \frac{\lambda IS}{1 + I} - \frac{\alpha IS}{\beta + S + ml} + \frac{\lambda IS}{1 + I} - \frac{\alpha IS Y}{\beta + S + ml} - \mu_1 I - aI
\]

\[+ \frac{\alpha S Y}{\beta + S + ml} - \mu_2 Y + aI - (\mu_3 + \rho)T .\]

Thus

\[
\frac{dW}{dt} \leq rS \left(1 - \frac{S}{K}\right) + \rho T - \mu_1 I - aI - \mu_2 Y + aI - (\mu_3 + \rho)T .
\]

Then

\[
\frac{dW}{dt} \leq rS - \mu_1 I - \mu_2 Y - \mu_3 T .
\]  

(3)

or

\[
\frac{dW}{dt} \leq (r + 1)S - (S + \mu_1 I + \mu_2 Y + \mu_3 T) .
\]
Knowing that \( W = S + I + Y + T \), then
\[
\frac{dW}{dt} \leq \bar{k}(r+1) - hW
\]
where
\[
\bar{k} = \max \{S_b, K\}, \quad h = \min\{1 + \mu_1 + \mu_2 + \mu_3\}
\]

Equation (3) can be written as
\[
\frac{dW}{dt} + hW \leq \bar{k}(1 + r)
\]
Equation (4) has a solution
\[
We^{hr} = \int \bar{k}(1 + r)e^{hr}dt
\]
or
\[
We^{hr} = \bar{k}\left(\frac{1 + r}{h}\right)e^{hr} + c
\]
Applying initial conditions \( W' = W_0 \) and \( t = 0 \), then
\[
W_0 = \bar{k}\left(\frac{1 + r}{h}\right) + c
\]
This gives
\[
c = W_0 - \bar{k}\left(\frac{1 + r}{h}\right).
\]
Thus
\[
W = \bar{k}\left(\frac{1 + r}{h}\right)(1 - e^{-ht}) + W_0e^{-ht},
\]
As \( t \to \infty \Rightarrow e^{-ht} \to 0 \)
This gives
\[
W \leq \frac{\bar{k}(1 + r)}{h}.
\]
Thus, the solution is bounded i.e.
\[
0 \leq W \leq \frac{\bar{k}(1 + r)}{h}.
\]
Therefore the feasible set for the model system (1) is contained in the domain
\[
\Omega = \left\{(S, I, Y, T) \in R^4 : W \leq \frac{\bar{k}(1 + r)}{h} + \varepsilon \right\} \text{ for } \varepsilon > 0 \text{ and } t \to \infty.
\]
Hence it is verified that \( \Omega \) is positively invariant set with respect to model (1)
3.2 Positivity of the solution

For model equations 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 \( t \geq 0 \). This will be established by the following theorem:

**Theorem 3.2**

Let \( S_0 > 0, I_0 > 0, Y_0 > 0, T_0 > 0 \). Then the solutions of the equation above are positive \( \forall t \geq 0 \) [5] (Hugo et al., 2012).

**Proof**

To prove the theorem, we consider the 1st equation of system (1):

\[
\frac{dS}{dt} = rS \left( 1 - \frac{S}{K} \right) - \frac{\lambda IS}{1 + I} - \frac{\alpha SY}{\beta + S + mI} + \rho T,
\]

or

\[
\frac{dS}{dt} \leq rS \left( 1 - \frac{S}{K} \right).
\]

This is a first order nonlinear differential inequality which has a solution

\[
\ln S - \ln (K - S) \leq \frac{r}{K} t + c.
\]

This can be written as

\[
\ln \left( \frac{S}{K - S} \right) \leq \frac{rt}{K} + c.
\]

or

\[
S \leq \frac{cK}{e^{-rt} + c}.
\]

Applying the initial conditions to equation (6) that is when \( t = 0 \) we have \( S_t = S_0 \) leads to

\[
S_0 = \frac{Kc}{e^{-rt} + c}.
\]

This gives

\[
c = \frac{S_0}{K - S_0}.
\]

Thus

\[
S \leq \frac{KS_0}{e^{-rt} (K - S_0) + S_0},
\]

As \( t \to \infty \) then \( e^{-ht} \to 0 \)

This leads to
0 ≤ S ≤ K.

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

Therefore it is true that

\[ S_0 ≥ 0, \quad I_0 ≥ 0, \quad Y_0 ≥ 0 \quad \& \quad T_0 ≥ 0 \quad \forall t ≥ 0. \]

### 3.3 Equilibrium points of the system

The equilibrium points of the system (1) is obtained by setting

\[
\frac{dS}{dt} = \frac{dI}{dt} = \frac{dY}{dt} = \frac{dT}{dt} = 0.
\]

Thus

\[
rS \left(1 - \frac{S}{K}\right) - \frac{\lambda IS}{1 + I} - \frac{a_s SY}{\beta + S + mI} + \rho T = 0,
\]

\[
\frac{\lambda IS}{1 + I} - \frac{\alpha_i Y}{\beta + S + mI} - \mu I - aI = 0,
\]

\[
\frac{\theta S + \theta I}{\beta + S + mI} - \mu Y = 0,
\]

\[
aI - (\mu + \rho)T = 0.
\]

#### 3.3.1 Trivial equilibrium point \( E_0 = (0,0,0,0) \)

The trivial equilibrium point is obtained by setting \( S = 0, I = 0, Y = 0 \) and \( T = 0 \) into the system (1) to obtain \( E_0 (S,I,Y,T) = (0,0,0,0) \).

#### 3.3.2 Axial equilibrium point \( E_1 = (K,0,0,0) \)

The axial equilibrium point is obtained by setting \( I = 0, Y = 0 \) and \( T = 0 \) into (1)

This gives \( rS \left(1 - \frac{S}{K}\right) = 0 \). It follows that either \( rS = 0 \) resulting to \( S = 0 \) or \( 1 - \frac{S}{K} = 0 \) giving \( S = K \).

Thus the axial equilibrium point is \( E_1 (S,0,0,0) = (K,0,0,0) \).

#### 3.3.3 Disease free equilibrium point of the system \( E_2 = (S,0,Y,0) \)

In the absence of the disease, the prey population grows logistically with intrinsic carrying capacity \( r \) and the environmental carrying capacity \( K \). Setting \( I = 0 \) to the system (1) yields

\[
rS \left(1 - \frac{S}{K}\right) - \frac{\alpha_i SY}{\beta + S} + \rho T = 0, \tag{7}
\]

\[
\frac{\theta SY}{\beta + S} - \mu Y = 0, \tag{8}
\]
\[-(\mu_i + \rho)T = 0, \quad \text{(9)}\]
\[
\left( \frac{\partial_i S}{\beta + S} - \mu_i \right) Y = 0, \quad \text{(10)}
\]

Equation (10) has solutions
\[
Y = 0 \quad \text{or} \quad Y = \frac{r \beta \theta_i \left[ K \theta_i - (K + \beta) \mu_i \right]}{\alpha_i K (\theta_i - \mu_i)^2}, \quad \text{(11)}
\]

Thus the disease free equilibrium point becomes
\[
E_2 = \begin{bmatrix} S \\ I \\ Y \\ T \end{bmatrix} = \begin{bmatrix} \frac{\mu_i \beta}{\theta_i - \mu_i} \\ 0 \\ \frac{r \beta \theta_i \left[ K \theta_i - (K + \beta) \mu_i \right]}{\alpha_i K (\theta_i - \mu_i)^2} \\ 0 \end{bmatrix}. \quad \text{(12)}
\]

### 3.3.4 The Effective Reproduction Number $R_e$

The effective reproduction number of the model system (1) is obtained by using the next generation operator method. The basic reproduction number $R_0$ is defined as the effective number of secondary infections caused by typical infected individual during his/her entire period of infectiousness [6]. This definition is given to a model that represents the spread of infection in a population. The basic reproduction number is obtained by taking the largest (dominant) eigenvalue (spectral radius) of
\[
\frac{\partial f(E_2)}{\partial t} \left[ \frac{\partial V_i(E_2)}{\partial t} \right]^{-1}. \quad \text{(13)}
\]

$r_i$ is the rate of appearance of new infection in compartment $i$, $V_i$ is the transfer of individuals out of the compartment $i$ by all other means and $E_2$ is the disease free equilibrium.

Consequently
\[
V_{i \rightarrow i} = \frac{\alpha_i IY}{\beta + S + mI} + \mu_i I + aI \quad \text{(14)}
\]

Using the linearization method, the associated matrix at disease free equilibrium is given by
\[
V_{i \rightarrow i} = \frac{\partial V_i}{\partial I} (E_2), \quad \text{(15)}
\]
or
\[
V = \frac{\partial \left( \frac{\alpha_i IY}{\beta + S + mI} \right)}{\partial I} + \mu_i + a. \quad \text{(16)}
\]

This gives
\[ V = \frac{\alpha Y (\beta + S)}{(\beta + S + mI)^2} + \mu_i + a, \]  
\((16)\)

For \( I = 0 \) we have
\[ V = \frac{\alpha Y (\beta + S) + (\mu_i + a)(\beta + S)}{(\beta + S)^2} \]  
\((17)\)

with
\[ V^{-1} = \frac{(\beta + S)^2}{\alpha Y (\beta + S) + (\mu_i + a)(\beta + S)} \]  
\((18)\)

or
\[ V^{-1} = \frac{\beta^2 \theta_i^2}{(\theta_i - \mu_i) \left( \beta \theta_i (a + \mu_i) + \alpha_i (\theta_i - \mu_i) \right) + \frac{r \beta \theta_i \left( K \theta_i - (K + \beta) \mu_i \right)}{K \theta_i^2 (\theta_i - \mu_i)^2}} \]  
\((19)\)

Next generation operator gives
\[ G = FV^{-1} = \frac{\beta^2 \theta_i^2 \lambda_\mu_i}{(\theta_i - \mu_i)^3 \left( \beta \theta_i (a + \mu_i) + \alpha_i (\theta_i - \mu_i) \right) + \frac{r \beta^2 \theta_i^2 \left( K \theta_i - (K + \beta) \mu_i \right)}{K \theta_i^2 (\theta_i - \mu_i)^3}} \]

Thus the effective reproduction number is
\[ R_e = \frac{\beta^2 \theta_i^2 \lambda_\mu_i}{(\theta_i - \mu_i)^3 \left( \beta \theta_i (a + \mu_i) + \alpha_i (\theta_i - \mu_i) \right) + \frac{r \beta^2 \theta_i^2 \left( K \theta_i - (K + \beta) \mu_i \right)}{K \theta_i^2 (\theta_i - \mu_i)^3}}. \]

3.3.5 The predator free equilibrium

Setting \( E_0(S, I, 0, T) \) in the absence of predator the system we have
\[ rS \left( 1 - \frac{S}{K} \right) - \frac{\lambda IS}{1 + I} + \rho T = 0 \]  
\((20)\)

\[ \frac{\lambda IS}{1 + I} - \mu_i I - aI = 0 \]  
\((21)\)

\[ aI - (\mu_i + \rho) T = 0 \]  
\((22)\)

Consequently
\[ R_1 T^3 + R_2 T^2 + R_3 T + R_4 = 0 \]  
\((23)\)

Equation (23) has roots of
\[ T_1 = -F_1 - F_2 + F_3, T_2 = -F_4 + F_5 - F_6, T_3 = -F_7 - F_8 - F_9. \]

where

\[ F_1 = -\frac{R_2}{3R_i}, \]

\[ F_2 = \frac{2^{1/3} (-R_2^2 + 3R_iR_1)}{3R_i \left[ -2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 + \sqrt{4(-R_2^2 + 3R_iR_1)^3 + (-2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4)^2} \right]^{1/3}}, \]

\[ F_3 = \frac{3 \times 2^{2/3} R_i}{(1 + i\sqrt{3})(-R_2^2 + 3R_iR_1)}, \]

\[ F_4 = \frac{3 \times 2^{2/3} R_i}{-2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 + \frac{4(-R_2^2 + 3R_iR_1)^3 + (-2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4)^2}{\left( -2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 \right)^2}}^{1/3}, \]

\[ F_5 = \frac{3 \times 2^{2/3} R_i}{6 \times 2^{2/3} R_i} \left[ 1 - \frac{1 + i\sqrt{3}}{1 + \sqrt{3}} \frac{-2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 + \frac{4(-R_2^2 + 3R_iR_1)^3 + (-2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4)^2}{\left( -2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 \right)^2}}{\left( -2R_2^2 + 9R_1R_iR_3 - 27R_i^2R_4 \right)^2} \right]^{1/3}. \]

Therefore the equilibrium points of the predator free will be

\[
\begin{bmatrix}
A_2 + A_1T_s \\
A_1T_s \\
0 \\
R_1T_s^3 + R_2T_s^2 + R_3 + R_4
\end{bmatrix}
\]

(24)

3.3.6 Endemic Equilibrium Points

The endemic equilibrium point of the model is obtained by setting each equation of the model (1) equal to zero. Then by solving the system we get each equilibrium point. Let \( S^*, I^*, Y^*, T^* \) be the endemic equilibrium point for \( S, I, Y, T \) respectively. Then

\[
s \left( 1 - \frac{S}{K} \right) - \frac{\lambda IS}{1 + I} - \frac{\alpha SY}{\beta + S + mI} + \rho T = 0 \quad (25)
\]

\[
\frac{\lambda IS}{1 + I} - \frac{\alpha SY}{\beta + S + mI} - \mu I - aI = 0 \quad (26)
\]

\[
\frac{\theta S + \theta I}{\beta + S + mI} - \mu Y = 0, \quad (27)
\]

\[
aI - (\mu_I + \rho)T = 0. \quad (28)
\]

Solving the resulting system we get

\[
S^* = A_1 - A_2I
\]
where
\[ A_1 = \frac{\mu \beta \theta}{\theta_1 - \mu}, \quad A_2 = \frac{(\mu \gamma - \theta_1)}{\theta_1 - \mu}, \]
\[ S' = A_1 - A_2 T, \]
with
\[ A_4 = A_2 A_3, \quad (29) \]
\[ I^* = \left( \frac{\mu + \rho}{a} \right) T, \quad (30) \]
or
\[ I^* = A_2 T, \quad (31) \]
where
\[ A_3 = \left( \frac{\mu + \rho}{a} \right), \]
\[ T^* = \left( \frac{G_1}{1 + I^*} + G_2 \right) (S')^2 - G_1 S' \]
where
\[ G_1 = \frac{\alpha \mu + \alpha \beta}{\rho \alpha_2}, \quad G_2 = \frac{r}{\rho K} \quad \text{and} \quad G_3 = \left( \frac{\alpha \mu + \alpha \beta + \alpha a}{\rho \alpha_2} \right), \]
Consequently
\[ H_1 T^3 + H_2 T^2 + H_3 T + H_4 = 0, \]
where
\[ T_1 = -Q_1 - Q_2 + Q_3, \quad T_2 = -Q_1 + Q_4 - Q_5, \quad T_3 = -Q_1 + Q_4 - Q_5. \]
with
\[ Q_1 = \frac{H_4}{3 H_1}, \]
\[ Q_2 = \left( \frac{2^{1/3}(-H_2^2 + 3 H_1 H_3)}{3 H_1 \left(-2 H_2^3 + 9 H_1 H_2 H_3 - 27 H_1^2 H_4 + \sqrt{4(-H_2^2 + 3 H_1 H_3)^2 + \left(2 H_2^3 + 9 H_1 H_2 H_3 - 27 H_1^2 H_4\right)^2}} \right)^{1/3}, \]
\[
Q_3 = \frac{1}{3 \times 2^{1/3}} H_1 \left[ -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y + \sqrt{4 \left( -H_x^2 + 3H_xH_y \right)^3 + \left( -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y \right)^2} \right]^{1/3},
\]

\[
Q_4 = \frac{1}{3 \times 2^{1/3}} H_1 \left( 1 + i \sqrt{3} \right) \left( -H_x^2 + 3H_xH_y \right) \left[ -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y + \sqrt{4 \left( -H_x^2 + 3H_xH_y \right)^3 + \left( -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y \right)^2} \right]^{1/3},
\]

\[
Q_5 = \frac{1}{6 \times 2^{1/3}} H_1 \left[ (1 - i \sqrt{3}) \left( -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y + \sqrt{4 \left( -H_x^2 + 3H_xH_y \right)^3 + \left( -2H_x^2 + 9H_xH_yH_z - 27H_x^3H_y \right)^2} \right) \right]^{1/3}.
\]

\[
Y^* = \frac{N_1T^3 - N_2T^2 + N_3T + N_4}{N_5 + N_6T},
\]

where

\[
N_1 = \left( -rA_iA_i^3K \beta r + A_i^2A_iA_{mr} \right), \quad N_2 = \left( A_i^3r + K \lambda A_iA_i - KrA_iA_i + A_iA_iA_iA_i + A_iA_iA_iA_iK - \right) \]

\[
N_3 = \left( \beta rA_iA_i - K \lambda A_iA_i + rKrA_iA_i - A_i^2A_iA_i - KrA_iA_i - A_iA_iA_iK - K \lambda A_iA_i + rKrA_iA_i - A_iA_iA_iK - \right), \quad N_4 = \left( -A_iA_iA_i + \lambda A_iA_i \right),
\]

\[
N_5 = K \alpha, \quad N_6 = KA_iA_iT.
\]

Thus the endemic equilibrium point for \( T_1, T_2, T_3 \) becomes

\[
\begin{bmatrix}
S^* \\
I^* \\
Y^* \\
T^*
\end{bmatrix} = \begin{bmatrix}
A_i - A_iT_2 \\
A_iT_2 \\
\frac{N_1T_3 - N_2T_2^2 + N_3T_1 + N_4}{N_5 + N_6T_3} \\
\frac{N_5 + N_6T_3}{T_3}
\end{bmatrix}
\]

(32)

### 3.4 Stability analysis at trivial equilibrium point \( E_0(0,0,0,0) \)

The local stability of the equilibrium points is established by using variational matrix

\[
\mathbf{V} = \begin{bmatrix}
\frac{\partial f}{\partial S} & \frac{\partial f}{\partial I} & \frac{\partial f}{\partial Y} & \frac{\partial f}{\partial T} \\
\frac{\partial g}{\partial S} & \frac{\partial g}{\partial I} & \frac{\partial g}{\partial Y} & \frac{\partial g}{\partial T} \\
\frac{\partial h}{\partial S} & \frac{\partial h}{\partial I} & \frac{\partial h}{\partial Y} & \frac{\partial h}{\partial T} \\
\frac{\partial Q}{\partial S} & \frac{\partial Q}{\partial I} & \frac{\partial Q}{\partial Y} & \frac{\partial Q}{\partial T}
\end{bmatrix}.
\]
The variational matrix at $E_0(0,0,0,0)$ becomes

$$
V_0 = \begin{bmatrix}
    r & 0 & 0 & \rho \\
    0 & -(\mu + a) & 0 & 0 \\
    0 & 0 & -\mu_2 & 0 \\
    0 & a & 0 & -(\mu_3 + \rho)
\end{bmatrix}
$$

(33)

The eigenvalues of the variational matrix is obtained as

Thus the eigenvalues obtained in matrix form are

$$
\lambda_1 = r, \quad \lambda_2 = -(a + \mu_1), \quad \lambda_3 = -\mu_2, \quad \lambda_4 = -(\rho + \mu_3)
$$

(34)

The trivial equilibrium points are stable when eigenvalues are negative otherwise unstable.

$$
\lambda_1 = r > 0, \quad \lambda_2 = -(a + \mu_1) < 0, \quad \lambda_3 = -\mu_2 < 0, \quad \lambda_4 = -(\rho + \mu_3) < 0.
$$

Therefore $E_0(0,0,0,0)$ is a saddle point which is locally stable manifold in $I,Y,T$ and locally unstable manifold in the $S$ direction.

### 3.5.3 Stability analysis at axial equilibrium point

The variational matrix at axial equilibrium point $E_i(K,0,0,0)$ is

$$
V_i = \begin{bmatrix}
    -r & -K\lambda & -\frac{K\alpha_i}{K+\beta} & \rho \\
    0 & -a + K\lambda - \mu_i & 0 & 0 \\
    0 & 0 & -\mu_2 & 0 \\
    0 & a & 0 & -(\rho + \mu_i)
\end{bmatrix}
$$

(35)

Thus the eigenvalues at the axial equilibrium point are

$$
\lambda_1 = -r, \quad \lambda_2 = -a + K\lambda - \mu_i, \quad \lambda_3 = \frac{K\alpha_i - K\mu_2 - \beta\mu_5}{K+\beta}, \quad \lambda_4 = -(\rho + \mu_i)
$$

(36)

The axial equilibrium points are stable when eigenvalues are negative otherwise unstable.

$$
\lambda_1 = -r < 0, \quad \lambda_2 = -a + K\lambda - \mu_i, \quad \lambda_3 = \frac{K\alpha_i - K\mu_2 - \beta\mu_5}{K+\beta} \quad \text{and} \quad \lambda_4 = -(\rho + \mu_i) < 0.
$$
In order for the axial equilibrium point to be asymptotically stable the following conditions should be satisfied:

\[-a + \lambda K - \mu_i < 0, \quad \frac{K\theta_i - K\mu_i - \beta \mu_i}{K + \beta} < 0,\]

Otherwise an axial equilibrium point becomes unstable.

### 3.4.4 Stability analysis at disease free equilibrium

The variational matrix at disease free equilibrium is given as

\[
V_2 = \begin{bmatrix}
D_1 - D_2 + D_3 + D_4 & D_5 & D_6 & \rho \\
0 & D_7 & 0 & 0 \\
D_8 & D_9 & 0 & 0 \\
0 & a & 0 & D_{10}
\end{bmatrix},
\]

where

\[
D_1 = \frac{\beta \theta^2 K r (K + 2 \beta) (\mu_z - \theta)}{K^2 \beta \theta^2 (\mu_z - \theta)} , \quad D_2 = \frac{\theta^2 K r \beta (\mu_z - \theta)}{K^2 \beta \theta^2 (\mu_z - \theta)},
\]

\[
D_3 = \frac{(K + \beta) r \beta \mu_z}{K^2 \beta \theta^2 (\mu_z - \theta)} (\mu_z - \theta) , \quad D_4 = 2 \frac{K r \beta^2 \theta^3}{K^2 \beta \theta^2 (\mu_z - \theta)},
\]

\[
D_5 = \frac{\mu_z (-K^2 \beta^2 \lambda \theta^2 + m \beta \theta \lambda)(K \theta_i - (K + \beta) \mu_z)}{K^2 \beta \theta^2 (\theta_i - \mu_z)} , \quad D_6 = -\frac{\alpha_i \mu_i}{\theta_i},
\]

\[
D_7 = \frac{-K \theta_i r \beta \theta_i \alpha_z + (K + \beta) r \beta \theta_i \alpha_z \mu_z + K^2 \beta \alpha \theta_i \mu_z (\theta_i (a + \mu_i) + (a + \beta \lambda + \mu_i) \mu_z)}{K^2 \beta \alpha \theta_i (\theta_i - \mu_z)} ,
\]

\[
D_8 = \frac{-K \theta_i r \beta \theta_i \alpha_z + (K + \beta) r \beta \theta_i \alpha_z \mu_z + K^2 \beta \alpha \theta_i \mu_z (\theta_i (a + \mu_i) + (a + \beta \lambda + \mu_i) \mu_z)}{K^2 \beta \alpha \theta_i \mu_z (\theta_i - \mu_z)},
\]

\[
D_9 = \frac{r \beta \theta_i (K \theta_i - (K + \beta) \mu_z)}{K^2 \beta \alpha \theta_i (\theta_i - \mu_z)}, \quad D_{10} = \frac{r \beta \theta_i (\theta_i - \theta_2 + m \mu_z)(K \theta_i + (K + \beta) \mu_z)}{K^2 \beta \alpha \theta_i (\theta_i - \mu_z)}.
\]

The Eigenvalue of the variational matrix are

\[
\begin{bmatrix}
\lambda_1 & H_1 + H_2 + H_3 + H_4 \\
\lambda_2 & G_1 - G_3 \\
\lambda_3 & G_1 + G_2 \\
\lambda_4 & -\rho - \mu_i
\end{bmatrix},
\]

where
The disease free equilibrium points are stable when eigenvalues are negative otherwise unstable.

\[ \lambda_1 = H_1 + H_2 + H_3 + H_4, \quad \lambda_2 = \frac{G_i - G_3}{G_3}, \quad \lambda_3 = \frac{G_i + G_2}{G_3}, \quad \lambda_4 = -\rho - \mu_i < 0. \]

Therefore disease free equilibrium points is locally stable manifold in \( r \) direction and with locally unstable manifold in \( s, i, y \) unless the following condition hold.

\[ \frac{G_i - G_3}{G_3} < 0 \rightarrow G_4 < G_3, \quad H_1 + H_2 + H_3 + H_4 < 0 \quad \text{and} \quad \frac{G_i + G_2}{G_3} < 0. \]

### 3.4.5 Stability analysis at predator free equilibrium points

The variational matrix at predator free equilibrium becomes

\[
V_3 = \begin{bmatrix}
c_{11} & c_{12} & c_{13} & c_{14} \\
c_{21} & c_{22} & c_{23} & c_{24} \\
c_{31} & c_{32} & c_{33} & c_{34} \\
c_{41} & c_{42} & c_{43} & c_{44}
\end{bmatrix}
\]

where

\[
c_{11} = r + \lambda \left( -1 + \frac{1}{1 + A T} \right), \quad c_{13} = -\frac{(A_i + A_T) \alpha_i}{\beta + m \alpha T + A_1 + A_T},
\]

\[
c_{23} = \frac{\lambda A T}{1 + A T}, \quad c_{44} = \rho, \quad c_{24} = -\frac{\lambda (A_i + A_T)}{(1 + A T)^2}, \quad c_{12} = -\frac{\lambda (A_i + A_T)}{(1 + A T)},
\]

\[
c_{31} = c_{32} = c_{34} = 0, \quad c_{24} = 0, \quad c_{23} = -\frac{A_i \alpha_T}{\beta + m \alpha T + A_1 + A_T},
\]

\[
c_{22} = \frac{a - \lambda A_i + \mu_i + T \left( -\lambda A_i + A_1 (2 + A_T) (a + \mu_i) \right)}{(1 + A_T)^2},
\]

\[
c_{33} = \frac{(A_i + A_T) \theta_i + A_i \theta_T}{\beta + m \alpha T + A_1 + A_T} - \mu_2, \quad c_{41} = c_{43} = 0, \quad c_{42} = a \quad \text{and} \quad c_{44} = -\rho - \mu_i.
\]

Then the eigenvalues are
\[ \lambda^4 + M_1 \lambda^3 + M_2 \lambda^2 + M_3 \lambda + M_4 = 0 \]  

(40)

where

\[ M_1 = \frac{8}{16} (c_1 + c_2 + c_3 + c_4) \quad , \quad M_2 = \frac{4}{16} \left( c_{22}c_{33} - c_{12}c_{21} + c_{22}c_{44} + c_{33}c_{44} \right) + c_1c_4 + c_1c_{22} + c_1c_{33} \],

\[ M_3 = \frac{2}{16} \left( -a\rho c_{21} + c_1c_2c_3c_3 - c_1c_2c_3c_3 + c_2c_2c_4c_4 - c_1c_2c_4c_4 \right) \],

\[ M_4 = \frac{1}{16} (c_1c_2c_3c_4 + a\rho c_{21} - c_1c_2c_3c_4) \cdot \]

Due to complexity in determining the stability predator free equilibrium, Routh Hurwitz criterion predator equilibrium is employed.

By the Routh Hurwitz criterion, predator free equilibrium point is stable if

\[ M_1, M_3, M_4 > 0, \]

\[ M_1M_3M_4 > M_1^2 + M_3^2M_4. \]

3.4.6. Stability analysis for endemic equilibrium points \( E \left( S, I, Y, T \right) \)

The variational matrix becomes

\[ V_4 = v_4 = \begin{pmatrix} v_{11} & v_{12} & v_{13} & \rho \\ v_{21} & v_{22} & v_{23} & 0 \\ 0 & 0 & v_{33} & 0 \\ 0 & a & 0 & v_{44} \end{pmatrix} \]

(41)

where

\[ v_{11} = \left( -1 + \frac{1}{1 + A_T} \right) - \left( 2r(A_i - A_T) \right) \frac{\left( \beta + mA_T \right) \left( N_1 - N_2 + N_1 + N_4 \right) \alpha_1}{\left( \beta + A_i + mA_T - A_T \right) \left( N_5 + N_6T \right)}, \]

\[ v_{12} = (A_i - A_T) \left[ -\frac{\lambda}{(1 + A_T)^2} \frac{m \left( T \left( N_1 - N_2 + N_1 + N_4 \right) \alpha_1 \right)}{\left( \beta + A_i + mA_T - A_T \right) \left( N_5 + N_6T \right)} \right], \]

\[ v_{13} = -\frac{(A_i + A_T)\alpha_1}{\beta + A_i + mA_T - A_T}, \quad v_{14} = \rho \]

\[ v_{21} = A_T \left( \frac{\lambda}{1 + A_T} + \frac{T \left( N_1 - N_2 + N_1 + N_4 \right) \alpha_2}{\left( \beta + A_i + mA_T - A_T \right) \left( N_5 + N_6T \right)} \right), \]

\[ v_{22} = -a + \frac{\lambda A_i}{(1 + A_T)^2} - \frac{\lambda A_T}{(1 + A_T)^2} \left( \frac{\beta + A_i - A_T \left( T \left( N_1 - N_2 + N_1 + N_4 \right) \alpha_2 \right)}{\left( \beta + A_i + mA_T - A_T \right)^2 \left( N_5 + N_6T \right)} \right), \]

\[ v_{23} = -\frac{A_i\alpha_T}{\beta + A_i + mA_T - A_T}, \quad v_{31} = \frac{T \left( N_1 - N_2 + N_1 + N_4 \right) \left( \beta + mA_T \right) \theta - A_i \theta_T \alpha_2}{\left( \beta + A_i + mA_T - A_T \right)^2 \left( N_5 + N_6T \right)}, \quad v_{24} = 0 \]
\[ v_{32} = \frac{T(N_1 - N_2 + N_3) + N_4}{\beta + A + m\lambda T - A T} \left( N_3 + N_6 T \right), \]
\[ v_{44} = -\rho - \mu_i, \]
\[ v_{33} = \frac{(A - A T)\theta + A T\theta}{\beta + A + m\lambda T - A T} - \mu_2, \] \[ v_{44} = 0, \quad v_{41} = 0, \quad v_{42} = a \quad \text{and} \quad v_{41} = 0. \]
\[ \forall T \in T_1, T_2, T_3. \]

The eigenvalues corresponding to the equilibrium point are
\[ \lambda^4 + W_1\lambda^3 + W_2\lambda^2 + W_3\lambda + W_4 = 0, \] \[ (42) \]
where
\[ W_i = -\frac{1}{2} (2v_{11} + v_{22} + v_{33} + v_{44}) \quad , \quad W_2 = \frac{1}{4} \left( v_{22}v_{33} + 4v_{11}v_{44} + 2v_{33}v_{44} - 2v_{12}v_{21} \right), \]
\[ W_3 = \frac{1}{4} \left( v_{12}v_{21}v_{33} - v_{11}v_{22}v_{33} + 2v_{12}v_{21}v_{44} - 2v_{11}v_{22}v_{44} - \lambda v_{11}v_{33}v_{44} - \lambda v_{11}v_{33}v_{44} \right) \quad \text{and} \]
\[ W_4 = \frac{1}{4} \left( v_{12}v_{21}v_{33} - v_{11}v_{22}v_{33} + 2v_{12}v_{21}v_{44} - 2v_{11}v_{22}v_{44} - \lambda v_{11}v_{33}v_{44} \right). \]

By the Routh Hurwitz criterion the endemic equilibrium point is locally asymptotically stable if
\[ W_1, W_2, W_3 > 0, \]
\[ W_4 > W_1^2 + W_1^2. \]

**IV. NUMERICAL SIMULATIONS**

In order to illustrate the analytical results of the study, numerical simulations of the normalised model system (1) are carried out using the set of parameter values below:

<table>
<thead>
<tr>
<th>Symbols/Parameter</th>
<th>Value</th>
<th>Reference/Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$r$</td>
<td>0.4</td>
<td>estimated</td>
</tr>
<tr>
<td>$k$</td>
<td>500</td>
<td>[3]</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>0.1</td>
<td>estimated</td>
</tr>
<tr>
<td>$\alpha_1$</td>
<td>0.1</td>
<td>estimated</td>
</tr>
<tr>
<td>$\alpha_2$</td>
<td>0.2</td>
<td>estimated</td>
</tr>
<tr>
<td>$\beta$</td>
<td>50</td>
<td>[3]</td>
</tr>
<tr>
<td>$m$</td>
<td>0.75</td>
<td>[3]</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>0.75</td>
<td>[3]</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>0.5</td>
<td>[3]</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>0.1</td>
<td>[3]</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>0.3</td>
<td>estimated</td>
</tr>
<tr>
<td>$a$</td>
<td>0.2</td>
<td>estimated</td>
</tr>
<tr>
<td>$\mu_3$</td>
<td>0.1</td>
<td>estimated</td>
</tr>
</tbody>
</table>
Figure (4.1) shows the distribution of population with time in all classes of susceptible prey, infected prey, predator and treated population with the predation rate $\alpha_1$ and $\alpha_2$.

![Graph showing proportion of susceptible prey with time for different treatment rates.](image1)

**Figure 4.1:** Proportion of susceptible prey with time for different treatment rate

It is initially observed that when rate of treatment of infective prey is high ($a = 0.9$) then proportion of prey population increases rapidly reaching its equilibrium point. Whereas, proportion of predator population increases slightly to attain its equilibrium point. Infected prey population seems to gradually decrease due to high treatment rate.

Figure (4.1) clearly shows that as the treatment rate say ($a = 0, 0.3, 0.6, 0.9$) increases with time then the susceptible prey increases and vice versa. This is due to the fact that the infective prey population will be posed to treatment and become susceptible prey reaching the equilibrium position.

Figure (4.2) shows the variation of the infective prey with time for different values of treatment rate.

![Graph showing variation of infective prey with time for different values of treatment rate.](image2)

**Figure 4.2:** Variation of the infective prey with time for different values of treatment rate.
In figure (4.2) it is observed that as the treatment rate \((a = 0, 0.3, 0.6, 0.9)\) increases then the proportion of infective prey class decreases with time reaching to its equilibrium position.

Figure (4.3) shows the variation of treated population versus time for different values of treatment rate.

![Figure 4.3: Variation of treated population versus time for different values of treatment rate.](image)

Figure (4.3) clearly shows that increasing treatment rate results to increase in treated population reaching to its equilibrium position.

Figure (4.4) shows the variation of susceptible prey population versus time for different values of recovery rate.

![Figure 4.4: Variation of susceptible prey population versus time for different values of recovery rate.](image)
In figure (4.4) it is observed that increasing the value of recovery rate of the infected prey population results to increase in susceptible prey population with time and vice versa. Eventually it levels off exponentially as a result of increasing the predator population attaining its equilibrium position.

Figure 4.5 shows the variation of infective prey versus time for different values of recovery rates.

![Variation of infective prey versus time for different values of recovery rates.](image)

It is observed that increasing the values of recovery rate results to the decrease in infected prey population which leads to increase in predator population attaining its equilibrium position.

Figure 4.6 shows the variation of susceptible prey population versus time for different values infection rates.

![Variation of susceptible prey population versus time for different values infection rate.](image)
It is observed that increasing the values of infection rates results to the decrease in susceptible prey population as the contact rate is high, and eventually goes to extinction attaining its equilibrium point.

Figure 4.7 shows the variation of susceptible prey population versus time for different values intrinsic growth rate.

![Graph showing variation of susceptible prey population versus time for different intrinsic growth rates](image)

Figure 4.7: Variation of susceptible prey population versus time for different values intrinsic growth rate.

The figure clearly shows that increasing the intrinsic growth rate results to the increase in susceptible prey population and vice versa.

Figure 4.8 shows the variation of susceptible prey population versus time for different values carrying capacity of the environment.

![Graph showing variation of susceptible prey population versus time for different carrying capacities](image)

Figure 4.8: Variation of susceptible prey population versus time for different values carrying capacity of the environment.
It is observed that increasing the values of carrying capacity results to the increase in susceptible prey population.

Figure 4.9 shows the variation of susceptible prey population versus time for different values death rate of predator

![Graph showing variation of susceptible prey population versus time for different death rates of predator](image)

Figure 4.9: Variation of susceptible prey population versus time for different values death rate of predator

It is observed that when the death of predator is high then the susceptible prey increases. This is because the prey will not be consumed and increases as the death of predator increases reaching to its equilibrium position.

V. CONCLUSIONS

In this paper, the dynamical behaviour of an eco-epidemiological model was proposed and analysed for nonlinear incidence and infective prey treatment. The model was well posed. Boundedness and positivity of the system hold, showing that the system is well behaved. The qualitative analysis shows that the model have five equilibrium points which are trivial equilibrium $E_0$, axial equilibrium $E_1$, disease free equilibrium $E_2$, predator equilibrium $E_3$ and positive equilibrium $E_4$. Also the effective reproduction number computed. The equilibrium points of the system were found to be stable if and only if the eigenvalues had negative real parts. Numerical simulations were performed and it was observed that the increase in treatment rate has potential influence of increasing both susceptible prey and predator population.

Using phase portraits of the model with different initial values revealed that increase in susceptible prey leads to increase in infected prey, predator population and treated population.

Model analysis showed that treatment of infective prey populations has the effect of increasing both predator population and susceptible prey population which save the population from extinction.

REFERENCES


