# Dynamical behavior of Susceptible prey – Infected prey – **Predator Populations**

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Abstract: Parasitism and predation are two ecological interactions that can occur simultaneously in any system of species. Specially, predation becomes particularly interesting in host/prey – parasite systems because predation can significantly modify the abundance of parasites and their host populations. The combined effect of parasites and predator on host/prey population leads to a larger effect on the dynamics of the population sizes. In this paper a prev – predator system is considered. The host species or prev population is categorized into susceptible and infected due to the presence of parasites. Predators are assumed to consume both the susceptible and infected hosts/prey with some partial preference given to susceptible ones. Thus, a mathematical model is developed to describing the population dynamics of susceptible prev – Infected prev – Predator system. Positivity and boundedness of the model are verified. Disease free equilibrium is found and shown that it is locally and asymptotically stable. Interior equilibrium is also identified and shown that it is locally, asymptotically and globally stable. Simulation study is conducted so as to verify the results of mathematical analysis. Different simulation scenarios are presented by assigning varying values to the parameters of the system using mathematical soft ware. Lastly, conclusions of the results are forwarded

*Keywords*: eco-epidemiological model; Stability; Predator; numerical simulations. \_\_\_\_\_

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# I. Introduction

In real systems parasites affect interactions among the species. Parasites influence predatory interactions in different ways depending on whether they infect the prey or the predator, or the both. Parasites infecting prey and predator are considered as two separate cases and examined. It is assumed that a microparasite harms or injures an established predator-prey interaction depending on the population densities and their immunity [1-3].

On one hand, the predators may hold the prey population below the threshold density so that the parasites cannot spread. At the other extreme, the parasite may regulate the host at the level required to sustain the predator and in this case the predator is excluded from direct competition with the parasite. Within these two limits, the parasite, prey and predator populations coexist with oscillatory dynamics that would not have occurred in absence of the parasite. At the other extreme, if attacks on prey by both parasite and predator were introduced then parasite would weaken the prey so that predator would catch more prey and as a result predator's population size would increase [4].

It is shown that two enemies can have a density dependent differential impact on prey resulting in a complex dynamics of the populations. A parasite infecting the predator rather than the prey is also investigated. In this case, either the parasite cannot be maintained if the predator, in the absence of the parasite, is at a density lower than the threshold or all three species coexist [5-11].

Historically, emphasis has been placed on the role of parasites in behavioral manipulation of prey, intermediate hosts and thereby facilitating parasite transmission to the host [1,4]. These systems are examples where the parasite infects either one of or both predator and prey.

Hence, parasitic manipulation may affect Predator-Prey relationships beyond those involved in the lifecycle of the parasite. Indeed, most theoretical treatments of this problem examine systems in which the predators are not part of the parasite lifecycle.

It is argued that parasites tend to destabilize Predator-Prey dynamics if parasites are introduced in to a Lotka–Volterra Predator–Prey model [12–15, 18].

In light of these studies and results, the effect of disease in ecological system is an important issue from a mathematical as well as an ecological point of view.

Most models for the transmission of infectious diseases originated from the classic work [12]. In the last few decades, mathematical models have become extremely important tools in understanding and analyzing the spread and control of infectious diseases.

To the best of our knowledge, the influence of predation on epidemics has not yet been studied considerably except few works [16–20]. Most of these works have dealt with predator-prey models with disease in the prey [21-24].

Recently the effects of a disease affecting a predator on the dynamics of a predator-prey system have been studied. In that study two possible asymptotic behaviors are observed:

(i) Either the predator population dies out and the prey tends to its carrying capacity, or (ii) the predator and prey coexist. In the latter case, the predator population tends either to a disease-free or to a disease-endemic state. In this issue mathematical epidemiology almost remains silent [2, 7].

In ecology the population dynamics with the inclusion of various features like mutualism, commensalism, and predation are considered. In epidemiology host – parasite systems are considered. In the present study both ecological and epidemiological features are combined and a hybrid system containing susceptible prey – infected prey – predator populations is developed.

The paper is organized as follows: In Section 2, model assumptions are listed out and based on those a mathematical model is developed. In Section 3, equilibrium points of the system are found and their stability analysis is carried out. In Section 4, simulation study is conducted for some reasonable numerical examples and results are presented. The paper ends with concluding remarks in Section 5.

# II. Basic assumptions and Model formulation

The classical form of a prey – predator or PP model is represented as dH/dt = H g(H) - P v(H)

(1) (2)

dP/dt = m P v(H) - d P (2) Here in (1), *H* and *P* are respectively the densities of prey and predator populations; g(H) is the per capita growth rate of prey in absence of predation; *d* is predator mortality rate; v(H) is the functional response and mv(H) is the numerical response of the predator; and m is the conversion efficiency.

Similarly, combining the assumptions of susceptible and infected outbreaks with a predator - prey model, a typical Susceptible – Infective or SI epidemic model with variable population sizes can be formulated as

$$dx/dt = x g(x) - h(x, y)$$
(3)  
$$dy/dt = h(x, y) - d y$$
(4)

Here in (3) and (4), x and y are the densities of susceptible and infected population respectively; g(x) is the intrinsic growth rate of the susceptible population, h(x, y) is the rate at which infections occur; and d is the sum of the death rates due to disease and the natural reasons.

Now the basic eco-epidemiological model is formulated by combining the predator-prey ecological model (1) - (2) and the *SI* epidemic model (3) - (4). In formulation of eco-epidemiological model the basic assumptions are made in the following.

# 2.1. Assumptions and formulation of the existing model

- (a) Variations in prey population densities are modeled as the influence of parasites.
- (b) Predation is incorporated into this epidemiological structure yielding three differential equations representing the densities of the susceptible prey x, the infected prey y and the predator z.
- (c) The model consists of three spices: susceptible prey, infected prey, and predator.
- (d) Susceptible Infected epidemic scheme is imposed on prey in prey predator model that track the population dynamics of susceptible prey x, infected prey y and the predator z.
- (e) In absence of infected prey and predator populations, the susceptible prey population grows following logistic function.
- (f) The susceptible prey population size decreases due to infected prey according to linear functional response.
- (g) The infected prey population size increases due to susceptible prey according to linear functional response.
- (h) The infected prey population size decreases due to predation according to linear functional response and also due to natural deaths.
- (i) The predator population grows according to a linear functional response due to infected prey; but reduces due to natural deaths.
- (j) On consuming infected prey, the predator is assumed not to suffer from the infection.

Basing of the foregoing assumptions an existing model is formulated as

$$\frac{dx}{dt} = rx\left(1 - \frac{x+y}{k}\right) - axy \tag{5}$$
$$\frac{dy}{dt} = axy - myz - dy \tag{6}$$

$$\frac{dz}{dt} = mayz - ez \tag{7}$$

# 2.2. Assumptions and formulation of the modified model

The existing model is modified with alteration of some existing assumptions and with the inclusion of some reasonable assumptions. The altered and newly included assumptions are listed as follows:

Let  $m_1$  be the decay rate of the infected prey due to predation and  $m_2$  be the growth rate of predator (a) due to infected prey. In the existing model these parameter are considered to be equal i.e.,  $m_1 = m_2 = m$ . But, it is reasonable to assume that these parameters need not be equal and thus in the modified model it is considered that  $m_1 \neq m_2$ .

(b) The predator is assumed to consume both the susceptible and the infected prey with different attack rates.

(c) Predator has different functional responses on susceptible and infected prevs.

Thus, the existing model is modified to formulate a modified model as

$$\frac{dx}{dt} = rx\left(1 - \frac{x+y}{k}\right) - \alpha xy - \frac{axz}{1+ahx}$$
(8)
$$\frac{dy}{dt} = \alpha xy - m_1 yz - \mu y$$
(9)

$$\frac{dz}{dt} = m_2 yz - \delta z + \frac{\gamma a x z}{1 + a h x}$$
(10)

The summarized notations and descriptions of the model parameters are given in table1 and 2. Model conceptual and schematic flow diagram is presented in Figure 1.



Figure 1. Schematic diagram of population dynamics of the modified model

Here in Figure 1, g(x) is the per capita growth rate of prey in the absence of predation; f(y) is linear response; and G(x) is the Monod functional response of predator.

The variables and parameters appearing in the system of model equations (1) are described and interpreted in Table 1 and Table 2 respectively.

Table 1: Notations and descriptions of the variables used in the model		
Variable	Description	
x(t)	Density of susceptible prey population at a time t	
y(t)	Density of infectious prey population at a time t	
z(t)	Density of predator population at a time t	

Table 1:	Notations and	descriptions	of the variables	used in the model

Table 2: N	Notations and	descriptions	of the para	meters used	in the model
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parameter	Description
r	Per capita birth rate of prey population
k	Carrying capacity of the environment for prey population
а	Predation on susceptible prey
α	The rate of infection infecting susceptible prey
γ	Growth rate of predator due to susceptible prey
δ	Natural mortality rate of predator
μ	Total death rate due to disease and the natural reasons
h	Predator's handling time of susceptible prey
$m_1$	Predation rate of infected prey
$m_2$	Growth rate of predator due to infected prey

# III. Dynamical behavior of the system

In this section analysis of some mathematical aspects of the model are performed. It is shown that the model variables are both positive and bounded. Equilibrium points are identified and stability analysis is conducted.

# **3.1** Positivity and boundedness of the model variables

Now it is to be shown that the system is biologically feasible i.e., the model variables are both positive and bounded.

**Proposition 1** All solutions of model equations (4) are positive.

**Positivity of** x(t): Consider the model equation for the susceptible prey which is  $dx/dt = rx\{1 - [(x + y)/k]\} - \alpha xy - [axz/(1 + ahx)]$ . On integrating the solution is obtained as  $x(t) = x_0 \exp \int \{r[1 - [(x + y)/k]] - \alpha y - [az/(1 + ahx)]\} dt$ . It is well known that for any exponential value the exponential function is always non – negative. Also, here the initial susceptible prey population is assumed to be a positive quantity, i.e.,  $x_0 > 0$ . Therefore, it is straight forward to conclude that x(t) > 0 for all  $t \ge 0$ .

**Positivity of** y(t): Consider the model equation for the infected prey which is  $dy/dt = \alpha xy - m_1yz - \mu y$ . The application of variables separable method reduces it to  $dy/y = (\alpha x - m_1 z - \mu)dt$ . On integrating the solution is obtained as  $y(t) = y_0 exp \int (\alpha x - m_1 z - \mu)dt$ . It is well known that for any exponential value the exponential function is always non – negative. Also, here the initial infected prey population is assumed to be a positive quantity, i.e.,  $y_0 > 0$ . Therefore, it is straight forward to conclude that y(t) > 0 for all  $t \ge 0$ .

**Positivity of** z(t): Consider the model equation for the predator which is  $dz/dt = m_2yz - \delta z + [\gamma axz/(1 + ahx)]$ . The application of variables separable method and integration leads to the solution as  $z(t) = z_0 \exp \int \{m_2y + [\gamma ax/(1 + ahx)] - \delta\} dt$ . As already it is mentioned that for any exponential value the exponential function is always non -negative. Also, here the initial predator population is assumed to be a positive quantity, i.e.,  $z_0 > 0$ . Therefore, it is straight forward to conclude that z(t) > 0 for all  $t \ge 0$ .

Hence, all solutions of the system variables are positive quantities i.e., x(t) > 0, y(t) > 0, z(t) > 0for all  $t \ge 0$  with the stated initial conditions.

**Proposition 2** All solutions of model equations (4) are bounded in the region  $R^3$ .

**Boundedness of** x(t): Consider the model equation for the susceptible prey which is  $dx/dt = rx\{1 - [(x + y)/k]\} - \alpha xy - [axz/(1 + ahx)]$ . It can be observed that the expression  $-\alpha xy - [axz/(1 + ahx)]$  is a negative quantity since all the components and members are positives. Thus, without loss of generality the equation can be expressed as an inequality as  $dx/dt \le rx[1 - (x/k)]$ . On applying the variables separable method and on integrating the solution is obtained as  $x(t) \le [Ck/(C - e^{-rt})]$ , where C is an arbitrary integral constant. The limit as  $t \to \infty$  leads to the result that  $x(t) \le k$ . Recall that here k is the environmental carrying capacity of the population x and by assumption it is a positive quantity. Thus, it can be concluded that x(t) is a bounded variable.

**Boundedness of** M(t) = x(t) + y(t) + z(t): The differentiation of M with respect to t and on using the model system of equations (4) simplifies to  $dM/dt = (dx/dt) + (dy/dt) + (dz/dt) = rx[1 - x+yk+axz(y-1)1+ahx+(m2-m1)yz-(\mu y+\delta z)$ . Since the parameters are assumed to satisfy the conditions  $m_2 \le m_1$ ,  $\gamma \le 1$  without loss of generality the equation can be expressed as an inequality as  $dM/dt \le rx[1 - (x/k)] - (\mu y + \delta z)$  or equivalently  $dM/dt \le 2rx - (rx + \mu y + \delta z)$ . Now introduce a parameter u satisfying  $u = min\{r, \mu, \delta\}$  and thus uM = ux + uy + uz. Thus the foregoing inequality of dM/dt can be expressed in terms of u as  $dM/dt + uM \le 2rx$ . But  $x(t) \le k$  and thus obtained  $(dM/dt) + uM \le 2rk$ . Integrating both sides and some algebraic simplifications lead to the final result as  $M(t) \le (2rk/u)$ . This implies that the size of total population in the model M(t) is bounded for all  $t \ge 0$ . Thus, each population is also bounded. Thus, it can be concluded that the model variables are bounded above.

#### 3.2 Existence of Steady states

To study the dynamical behavior of the system getting the equilibrium points is the first step. Now the fixed points are obtained as the solutions of (dx/dt) = (dy/dt) = (dz/dt) = 0. Using this and the model equations (4) it is found that there exist six steady states that are listed as follows:

(i) 
$$E_0(0, 0, 0)$$
  
(ii)  $E_1(k, 0, 0)$   
(iii)  $E_2(0, \frac{\delta}{m_2}, 0)$   
(iv)  $E_3(\frac{\mu}{\alpha}, (\frac{r}{\alpha})(\frac{\alpha k - \mu}{r + k\alpha})$   
(v)  $E_4(p^*, 0, q^*p^*)$ 

(iv)  $\begin{array}{l} E_3\left(\frac{\mu}{\alpha}, \left(\frac{r}{\alpha}\right)\left(\frac{ak-\mu}{r+k\alpha}\right), 0\right) \\ (v) & E_4\left(p^*, 0, q^*p^*[r(k-1)]\right) \text{ where } p^* = \delta/\alpha(\gamma-\delta h), q^* = \gamma/ak(\gamma-\delta h) \\ (vi) & E_5\left(\frac{p\pm R}{2}, \frac{\delta}{m_2} - \frac{\gamma ax^*}{m_2(1+ahx^*)}, \frac{ax^*-\mu}{m_1}\right) \end{array}$ 

Note that in E<sub>5</sub> the notations *p*, *q* and *R* are used to represent the expressions as follows:  $p = \frac{m_1}{m_2} \left[ \left( m_2 k + \gamma h + \alpha k \gamma h - \delta + \delta \alpha r + m 2 h \alpha + \alpha k m 2 h r \right) \right]$ 

$$\frac{q}{R} = \frac{[km_2(rm_1 + a\mu) - \delta m_1(r + k\alpha)]}{(rahm_2)}$$
$$R = \sqrt{p^2 + 4q}$$

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### 3.3 Community matrix of the model system

Let the model equations (4) be expressed as dx/dt = f(x, y, z), dy/dt = g(x, y, z) and dz/dt = h(x, y, z) then the variation matrix is defined as

$$M = \begin{pmatrix} f_x & f_y & f_z \\ g_x & g_y & g_z \\ h_x & h_y & h_z \end{pmatrix}$$

Here  $f_x$  denotes the partial derivative of the function f with respect to x i.e.  $f_x = \partial f / \partial x$  and similarly the same is true for the remaining elements of the matrix M.

In view of the system (4) the elements of the variation matrix M can be computed as;  $f_x = r(1 - (2x + y)/k) - (\alpha y + az/(1 + ahx)^2)$ ,  $f_y = -x(k\alpha + r)/k$ ,

 $f_z = -[ax/(1 + ahx)]$ ,  $g_x = \alpha y$ ,  $g_y = -(\alpha x + m_1 z + \mu)$ ,  $g_z = -m_1 y$ ;

 $h_x = \gamma a z / (1 + a h x)^2$ ,  $h_y = m_2 z$ ,  $h_z = m_2 y - \delta + \gamma a x / 1 + a h x$ .

#### **3.4** Stability analysis of the system

#### **3.4.1** Local stability of the system

In this section, the local stability analyses of the system (4) around each of the equilibrium points are discussed. **Theorem 1** The trivial steady state  $E_0$  exists and unstable.

**Proof:** Let the variation matrix M takes the form as  $M_0$  at the equilibrium point  $E_0$ . Then the characteristic equation of the matrix  $M_0$  can be expressed as  $\det(M_0 - \lambda I) = 0$  and its evaluation reduces to  $(r - \lambda)(-\mu - \lambda - \delta - \lambda = 0$  giving the three eigenvalues as  $\lambda 1 = r$ ,  $\lambda 2 = -\mu, \lambda 3 = -\delta$ . It is straight forward to observe that  $\lambda 1$  is positive while  $\lambda_2$ ,  $\lambda_3$  are negative eigenvalues. That is,  $\lambda_1 > 0$ ,  $\lambda_2 < 0$ ,  $\lambda_3 < 0$ . Thus, the trivial equilibrium  $E_0$  is unstable.

**Theorem 2** The steady state  $E_1$  is locally asymptotically stable if the model parameters satisfy the conditions  $a < (\mu/k)$  and  $\gamma < \delta$  [(1 + *ahk*)/*ak*].

**Proof:** Let the variation matrix M takes the form as  $M_1$  at the equilibrium point  $E_1$ . Then the characteristic equation of the matrix  $M_1$  can be expressed as  $det(M_1 - \lambda I) = 0$  and its evaluation gives the eigenvalues as  $\lambda_1 = -r$ ,  $\lambda_2 = ak - \mu$ ,  $\lambda_3 = -\delta + [\gamma ak/(1 + ahk)]$ . Here it can be observed that (i)  $\lambda_1$  is unconditionally negative since r is a positive quantity, i.e.,  $\lambda_1 < 0$  (ii) the Eigenvalue  $\lambda_2$  is negative if the negative condition  $a < (\mu/k)$ is satisfied and (iii) the eigenvalue  $\lambda_3$  is if the condition  $\gamma < \delta [(1 + ahk)/ak]$  is satisfied. Hence, the statement is proved.

**Theorem 3** The steady state  $E_2$  is locally asymptotically stable if the model parameters satisfy the conditions  $m_2 < m_1 < \delta(1 + ak)$ .

**Proof** Note that at the equilibrium  $E_2$  the susceptible prey and the predator population's extinct but only infected prey population exists. That is, at  $E_2$  infected prey population alone exists. At the equilibrium point  $E_2$  the variation matrix M takes the form as

$$M_2 = \begin{pmatrix} u_1 & 0 & 0 \\ u_4 & u_2 & u_5 \\ 0 & 0 & u_3 \end{pmatrix}$$

Here in  $M_2$  some notations are used to represent the expressions as  $u_1 = r[1 - (\delta/km_1)] - (\alpha\delta/m_1)$ ;  $u_2 = -\mu$ ;  $u_3 = \delta[(m_2/m_1) - 1]$ ;  $u_4 = (\alpha\delta/m_1)$  and  $u_5 = -\delta$ . The characteristic equation  $\det(M_2 - \lambda I) = 0$ can be expressed in a factorization form as  $(u_1 - \lambda)(u_2 - \lambda)(u_3 - \lambda) = 0$  giving the three eigenvalues as  $\lambda_1 = u_1$ ,  $\lambda_2 = u_2$ ,  $\lambda_3 = u_3$ . Now, it is easy to observe that (i)  $\lambda_1$  is negative if the condition  $m_1 < \delta(1 + ak)$  is satisfied (ii)  $\lambda_2$  is unconditionally negative and (iii)  $\lambda_3$  is negative if the condition  $m_2 < m_1$  is satisfied. Combining all the above it can be concluded that the steady state  $E_2$  is locally asymptotically stable if the model parameters satisfy the conditions  $m_2 < m_1 < \delta(1 + ak)$ .

**Theorem 4** The steady state  $E_3$  is locally asymptotically stable if the model parameters satisfy the condition  $\{m_2[(\alpha rk - r\mu)/(\alpha (r + k\alpha))] - \delta + [\gamma a\mu/(\alpha + ah\mu)]\} < 0.$ 

**Proof:** Note that at the equilibrium  $E_3$  both the susceptible and infected prey populations exist but, the predator population extinct. At the equilibrium point  $E_3$  the variation matrix M takes the form as

$$M_3 = \begin{pmatrix} c_{11} & c_{12} & c_{13} \\ c_{21} & c_{22} & c_{23} \\ 0 & 0 & c_{33} \end{pmatrix}$$

Here in  $M_3$  some notations are used to represent the expressions as  $c_{11} = -r(\mu/\alpha k)$ ;  $c_{12} = -[\mu + r\alpha k; c_{13}] = -a\mu\alpha + ah\mu$ ;

$$c_{21} = [(\alpha rk - r\mu)/(r + k\alpha)]; c_{22} = -2\mu; c_{23} = -m_1[(\alpha rk - r\mu)/(\alpha(r + \alpha k))]$$
 and

 $c_{33} = \left\{ m_2 \left[ (\alpha r k - r \mu) / (\alpha (r + k \alpha)) \right] - \delta + \left[ \gamma a \mu / (\alpha + a h \mu) \right] \right\}.$ 

Solving the characteristic equation  $det(M_3 - \lambda I) = 0$  gives the three eigenvalues as  $\lambda_1 = (1/2) [m + 1/2] [m +$ m2-4D,  $\lambda 2=12 m-m2-4D$ ,  $\lambda 3=c33$ . Here the notations represent the expressions as m=c11+c22 and  $D = c_{11}c_{22} - c_{12}c_{21}$ . Observe that the eigenvalues  $\lambda_1$  as well as  $\lambda_2$  are unconditionally negative since m < 0, D > 0 and also  $m^2 - 4D > 0$ . However, the eigenvalues  $\lambda_3$  is negative if the condition  $\{m_2[(\alpha rk - r\mu)/(\alpha (r + k\alpha))] - \delta + [\gamma \alpha \mu/(\alpha + \alpha h\mu)]\} < 0$  is satisfied. Hence, the theorem is proved.

**Theorem 5** The steady state  $E_4$  is locally asymptotically stable if the model parameters satisfy (i)  $[m_2 r(\alpha k - \mu)/\alpha (r + \alpha k)] + \{[a\gamma \mu - \delta (\alpha + ah\mu)]/(\alpha + ah\mu)\} < 0 \text{ and (ii) } ak < \mu.$ 

**Proof** Note that at the equilibrium point  $E_4$  susceptible prey and predator populations present but the infected prey population is absent. Hence,  $E_4$  may be termed as disease free equilibrium point. At the equilibrium point  $E_4$  the variation matrix M takes the form as

$$M_4 = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & 0 & a_{23} \\ 0 & 0 & a_{33} \end{pmatrix}$$

Here in  $M_4$ , some notations are used and they are  $a_{11} = [r\mu(r-1)/(r+k\alpha)];$ 

 $a_{12} = -\mu [(r/\alpha k) + 1]; a_{13} = -[a\mu/(\alpha + ah\mu)]; a_{21} = [(\alpha rk - r\mu)/(r + k\alpha)];$ 

$$a_{23} = -m_1 [(\alpha r k - r\mu/(\alpha (r + k\alpha)))];$$

 $a_{33} = [m_2 r(\alpha k - \mu)/\alpha (r + \alpha k)] + \{[\alpha \gamma \mu - \delta (\alpha + ah\mu)]/(\alpha + ah\mu)\}.$ 

Now, the characteristics equation  $det(M_4 - \lambda I) = 0$  reduces to the factorized form as  $(a_{21} - \lambda)(a_{12} - \lambda$  $\lambda a 33 - \lambda = 0$  giving the three eigenvalues as  $\lambda 1 = a 21$ ,  $\lambda 2 = a 12$ ,  $\lambda 3 = a 33$ . It is straight forward to observe that (i)  $\lambda_1$  is negative if the condition  $ak < \mu$  is satisfied (ii)  $\lambda_2$  is negative unconditionally and (iii) (i)  $\lambda_3$  is negative if the condition  $[m_2 r(\alpha k - \mu)/\alpha (r + \alpha k)] + \{[a\gamma \mu - \delta (\alpha + ah\mu)]/(\alpha + ah\mu)\} < 0$  is satisfied. Since all the three eigenvalues are negative values under the stated conditions, the statement of the theorem is proved.

**Theorem 6** The interior equilibrium point  $E_5$  is locally asymptotically stable if the model parameters satisfy the condition  $a > [m_1 \delta(\alpha + r)/\gamma]$ .

**Proof**: Note that at the interior equilibrium point  $E_5$  all the three populations of the model viz., susceptible prey, infected prey and predator are present.

At the equilibrium point  $E_5$  the variation matrix M takes the form as

$$M_5 = \begin{pmatrix} A & B & C \\ G & H & J \\ D & E & F \end{pmatrix}$$

Here in  $M_5$ , some notations are used and they are represented as:

$$\begin{split} A &= r(1 - (2x^* + y^*)/k) - (\alpha y^* + az^*/(1 + ahx^*)^2), \ B &= -[(\alpha + r)x^*/k], \\ G &= \alpha y^*, C \ = -[ax^*/1 + ahx^*], \ D &= \gamma az^*/(1 + ahx^*)^2; \ E &= m_2 z^* \ , \end{split}$$

 $F = m_2 y^* - \delta + \gamma a x^* / 1 + a h x^*$ ;  $H = -(\alpha x + m_1 z^* + \mu)$ ;  $J = -m_1 y^*$ .

The characteristic equation in this case  $det(M_5 - \lambda I) = 0$  reduces to the form as:

 $\lambda^3 + w\lambda^2 + v\lambda + u = 0$ . Here w = -(A + H + F); v = (AH + AF + HF - CD - EJ - BJ)and u = (AEJ + BGF + CDH - AHF - BDJ - CGF). Here it can be observed here that (i) w is a positive quantity if the condition  $a > [m_1 \delta(\alpha + r)/\gamma]$  is satisfied and (ii) v and u are unconditionally positive quantities. Thus following Routh-Hurwitz criterion it can be concluded that all the three eigenvalues are negative quantities if the condition  $a > [m_1 \delta(\alpha + r)/\gamma]$  is satisfied. Thus, the interior equilibrium point is asymptotically stable, for the assumed condition. Hence, the theorem is proved

#### 3.3.1 Global stability of the system.

Here, the stability analysis of the model (8)-(10) is conducted using suitable Lyapunov functions and the results are presented in the form of theorems followed by their proofs.

**Theorem 7:** The equilibrium point  $E_1$  is globally asymptotically stable.

**Proof:** Consider the Lyapunov function  $v(x, y, z) = [x - x^* - x^* ln(x/x^*)]$ . On differentiating v with respect to t, substituting expression for dx/dt and after some simplification it is obtained as  $\left(\frac{x-x^*}{x}\right) \left(\frac{dx}{dt}\right) = \left(\frac{x-x^*}{x}\right) \left[rx\left(1-\frac{x+y}{k}\right) - \alpha xy - \frac{axz}{1+ahx}\right] = (x-x^*) \left[-(r/k)(x-x^*)\right] = -(r/k) (x-x^*)^2 < 0.$ Thus,  $\frac{dv}{dt} < 0$  i.e., v is positive definite and also  $v(x^*, y^*, z^*) = 0.$ 

Therefore  $E_1$  is globally asymptotically stable.

**Theorem 8**: The steady state  $E_3$  is globally asymptotically stable if the condition  $m \le 1$  is satisfied.

**Proof:** Consider the Lyapunov functions as  $v(x, y, z) = [x - x^* - x^* ln(x/x^*)] + m[y - y^* - x^* ln(x/x^*)]$ y\*lnyy\*. Now, the differential of  $\nu$  with respect to t and after some algebraic manipulations reduces to the following form:

$$\dot{v} = [(x - x^*)/x](dx/dt) + m[(y - y^*)/y](dy/dt) = l [(x - x^*)/x] \left\{ rx \left[ 1 - \frac{x + y}{k} \right] - \alpha xy - \frac{axz}{1 + ahx} \right\} + m [(y - y^*)/y] [\alpha xy - m_1 yz - \mu y)] = -(r/k)(x - x^*)^2 - \alpha (y - y^*)(x - x^*) + m\alpha (y - y^*) (x - x^*) = -(r/k)(x - x^*)^2 + [\alpha (y - y^*)(x - x^*)(m - 1)]$$

Observe that in the expression for  $\dot{v}$ , the term  $-(r/k)(x-x^*)^2$  is negative and the expression  $\alpha(y-x)^2$ y\*x-x\* is positive. Thus, it is straight forward to conclude that dvdt < 0 if the condition  $0 \le m \le 1$  holds true.

Therefore,  $E_3$  is globally asymptotically stable.

**Theorem 9:** The interior steady state  $E_5$  is globally asymptotically stable if the model parameters satisfy the condition  $\gamma a m_1 < \alpha m_2$ .

**Proof**: Consider the Lyapunov function as  $v(x, y, z) = [x - x^* - x^* ln(x/x^*)] + l[y - y^* - y^* ln(y/y^*)] + l[y - y^* - y^* ln(y/y^*)]$  $n[z - z^* - z^* ln(z/z^*)].$ 

On differentiating v with respect to t gives the result as

 $\dot{v} = \left(\frac{x-x^*}{x}\right)\left(\frac{dx}{dt}\right) + l\left(\frac{y-y^*}{y}\right)\left(\frac{dy}{dt}\right) + n\left(\frac{z-z^*}{z}\right)\left(\frac{dz}{dt}\right).$ Now substituting the expressions for dx/dt, dy/dt, dz/dt and after some algebraic manipulations, it reduces to

$$\dot{v} = [(x - x^*)/x] \{ rx \{ 1 - [(x + y)/k] \} - \alpha xy - \{ axz/[1 + ahx] \} \} \\ + m[(y - y^*)/y] [\alpha xy - m_1yz - \mu y] + n [(z - z^*)/z] [m_2yz - \delta z + [\gamma axz/(1 + ahx)] ]$$

$$= -\{(x - x^*)^2[(r/k) + d_1]\} + (y - y^*)(x - x^*)(l\alpha - \alpha) + (z - z^*)(y - y^*)(nm_2 - lm_1) + (z - z^*)(x - x^*)[(nv\alpha - \alpha)/(1 + ahx)]$$

Here in  $\dot{v}$ , the notations used are  $n = m_1/m_2$  and  $d_1 = [\alpha z^*/(1 + ahx)(1 + ahx^*)]$ .

Now fixing the parameter at l = 1, the expression for  $\dot{v}$  reduces to

 $\dot{v} = -\{(x - x^*)^2 \left[ (r/k) + d_1 \right] \} + (z - z^*) \left( x - x_1^* \right) \left[ (n\gamma a - \alpha)/(1 + ahx) \right] \,.$ 

Since in  $\dot{v}$ , the term  $-\{(x - x^*)^2 [(r/k) + d_1]\}$  is negative and the expression  $(z-z^*)(x-x^*)/(1+ahx)$  is positive it can be concluded that dv/dt < 0 if the condition on parameters  $\gamma a m_1 < \alpha m_2$  holds true.

Therefore, by LaSalle invariant principle the theorem holds.

#### **IV.** Numerical simulations

In this section, numerically simulation study is conducted to understand the dynamics of the deterministic model. For that purposed, certain values are assigned to the parameters taken from their reasonable ranges.







In, Figures 1 and 2, Numerical examples of the model around  $E_1$  are presented. In both the figures other variables are set as r = 0.72, k = 1.44,  $\alpha = 1.71$ ,  $m_1 = 0.06$ , h = 0.435,  $\mu = 0.74$ , a = 3.08. It can be observed that the Predator population initially grows because sufficient prey is available and later it decreases and finally all the populations will die out.



Figure 3 Population dynamics around  $E_3$  with r = 5.25, k = 2.525,  $\alpha = 1.6923$ ,  $\delta = 1.1760$ ,  $\gamma = 0.1450$ ,  $\mu = 0.891$ 



0.2060,  $\mu = 0.7200$ 

In Figures 3 and 4, Numerical simulations of the model around the equilibrium point  $E_3$  are presented. The population sizes of both infected and uninfected preys experience some fluctuations initially and then they would stabilize over a period of time. However, the predator population decreases continuously till it reaches its equilibrium value. The values of the fixed parameters used in both the simulations are  $m_1 = 0.2744$ , a = 0.3600, h = 0.0954,  $m_2 = 1.8800$ .



Figure 6 Population dynamics around  $E_4$  with  $m_2 = 1.137$ ,  $\mu = 3.345$ , h = 0.351

In figures 5 and 6, Numerical simulations of the model around  $E_4$  are presented. The initial population sizes of both susceptible and infected preys are chosen to be higher than that of the predator. However, over a period of time all the populations will reach their equilibrium values.

It is observed that the equilibrium values of susceptible prey and predator are positive quantities and are very close to each other. However, the equilibrium value of the infected prey is zero. The values of the fixed parameters used in both the simulations are r = 8.26, k = 2.16,  $\alpha = 1.098$ ,  $m_1 = 4.016$ ,  $\delta = 2.96$ ,  $\gamma = 0.784$ , a = 3.08.





In Figures 7 and 8, numerical examples of the model around the interior point  $E_5$  are considered. The populations are observed regulating one other. In both the figures, the susceptible prey population lies higher than both the infected prey and the predator. However, in Figure 7 the infected prey population dominates the predator population but the reverse occurs in Figure 8. The values of the fixed parameters used in both the simulations are r = 2.5600, k = 1.4650,  $\alpha = 2.7180$ ,  $m_1 = 0.0763$ ,  $\delta = 0.6560$ ,  $\gamma = 0.5140$ , a = 0.2990, h = 0.2745,  $\mu = 0.1000$ .

# V. Conclusion

In this study a mathematical model is developed to describe the population dynamics of susceptible prey – Infected prey – Predator system taking into account that the functional response and interaction coefficients of predator population are different.

It is verified that the model variables are both positive and bounded and thus the model is biologically meaningful. Further, disease free equilibrium is shown to be locally and asymptotically stable. Also, the Interior equilibrium is locally, asymptotically and globally stable. Moreover, the dynamical behavior of the system has been investigated locally as well as globally by considering certain conditions.

To understand the effects of varying parameters on the dynamics of the system, simulation study is conducted in support of the mathematical analysis and which the following conclusions are drawn:

(i) If the predator prefers to consume infected prey then the susceptible prey population size will increase.

(ii) If the predator prefers to consume susceptible prey and since the infected prey decrease due to disease then the combined prey population size will decrease.

(iii) If the predator has no preference to consume either susceptible or the infected prey i.e., the predator prefers susceptible and infected prey equally, the joint population size of the prey remains unaltered.

(iv) From figure 6 it is observed that if the predator holds the prey population below the threshold the transmission of disease can't spread. Moreover, when the infected prey is regulated, the predator is excluded from the interaction figure (3) and (4), so that inclusion of predator in either linear response or type II functional response both in infected and susceptible prey interactions respectively would be considered as indicated in figure (7) and (8) to stabilize the whole population dynamics.

(v) Moreover, based on some parametric values the functional response of the predator interaction is differ for both free living and infected prey as per the assumption.

#### References

- [1]. Anderson R. and May R. Infectious Diseases of Humans, Dynamics and Control. Oxford University Press, Oxford (1991).
- [2]. D. Adak and N. Bairagi. Dynamic behavior of a predator-prey-parasite model with nonlinear incidence rate, J. Biol. Syst. 1 (1) (2014).
- [3]. Shashi K., Vivek K. Dynamics of a prey-predator system with Infection in prey. Electronic Journal of Differential Equations, 209 (2017), 1-27.
- Sen P., Das K. Simultaneous Effects of Prey Defense and Predator Infection on a Predator Prey System. Ann. Bio. Sci., 2017, 5 (1):37-46
- [5]. B. Mukhopadhyay R. Bhattacharyya. Role of predator switching in an eco-epidemiological model with disease in the prey, Ecol. Model. 220 (2009) 931.
- [6]. X. Wei. Global stability and Hopf bifurcation of a host-parasite system, Math. Biosci. 10(4) (2017).17
- [7]. Jana S. and Kar T. K. Modeling and Analysis of a prey-predator system with disease in the prey. Chaos, Solutions and Fractals 47 (2013).
- [8]. Mukhopadhyay B. Bhattacharyya R. Dynamics of a delay-diffusion prey-predator Model with disease in the prey. J. Appl. Math and Computing 17 (2005): 361.

- [9]. R. Bhattacharyya, B. Mukhopadhyay. On an eco-epidemiological model with prey harvesting and predator switching: local and global perspectives, Nonlinear Anal.: RWA 11 (2010) 3824.
- [10]. U. Ufuktepe and S. Kapcak. Stability analysis of a host parasite model, Adv. Diff. Equations 79 (2013) 1–7.
- [11]. D. Greenhalgh and M. Haque, "A predator-prey model with disease in the prey species only," *Mathematical Methods in the Applied Sciences*, vol. 30, no. 8, pp. 911–929, 2007.
- [12]. M. L. Rosenzweig, R. H. MacArthur. Graphical representation and stability conditions of predator-prey interactions, Am. Nat. 97 (1963) 209
- [13]. M. Haque and E. Venturino. "Increase of the prey may decrease the healthy predator population in presence of a disease in the predator," *Hermis*, Vol. 7, Pp. 38–59, 2006.
- [14]. Y. Xiao and F. Van Den Bosch. "The dynamics of an eco-epidemic model with biological control," *Ecological Modeling*, Vol. 168, No. 1-2, Pp. 203–214, 2003.
- [15]. N. Bairagi, S. Chaudhury and J. Chattopadhyay. Harvesting as a disease control measure in an eco-epidemiological system a theoretical study, Math. Biosci. 217, 134-144, 2009.
- [16]. Diekmann O. and Heesterbeek J. A. P. Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. Wiley Series in Mathematical and Computational Biology (2000).
- [17]. Naji R. K. Mustafa A. N. The Dynamics of an Eco-Epidemiological Model with Nonlinear Incidence Rate. Journal of Applied Mathematics http://dx.doi.org/10.1155/2012/852631, 2012.
- [18]. E. Venturino. "The influence of disease on Lotka-Volterra systems," Rocky Mountain Journal of Mathematics, Vol. 24, Pp. 381– 402, 1994.
- [19]. B. Mukhopadhyay, R. Bhattacharyya. Effects of deterministic and random refuge in a prey-predator model with parasite infection, J. Math. Biol. 239 (2012) 124-130.
- [20]. J. Chattopadhyay, O. Arino. A predator-prey model with disease in the prey, Nonlinear Anal. 36 (1999) 747.
- [21]. S. R. Hall, M. A. Duffy, C. E. Caceres. Selective predation and productivity jointly drive complex behavior in host-parasite systems, Am. Nat. 165 (1) (2005) 70.
- [22]. B. Mukhopadhyay and R. Bhattacharyya. Effects of deterministic and random refuge in a prey-predator model with parasite infection. Mathematical Biosciences 239 (2012): 124–130.
- [23] L. Han, Z. Ma and H. W. Hethcote. "Four predator prey models with infectious diseases," *Mathematical and Computer Modeling*, Vol. 34, No. 7-8, Pp. 849–858, 2001.
- [24]. M. Haque and E. Venturino. "An eco-epidemiological model with disease in predator: the ratio-dependent case," *Mathematical Methods in the Applied Sciences*, Vol. 30, No. 14, Pp. 1791–1809, 2007.

Geremew Kenassa Edessa." Dynamical behavior of Susceptible prey – Infected prey – Predator Populations. IOSR Journal of Mathematics (IOSR-JM) 14.4 (2018) PP: 31-41.