



## Dynamics and Complexities of a Two Prey- One Predator System Involving Competition with Nonlinear Feed Back Controls

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**Abstract:** In this article, a three dimensional continuous time prey-predator system consisting of susceptible prey, infected prey and predator is proposed and analysed in the presence of intra-specific competition in predator and mortality of infected prey and predator. We assumed the predator preys on infected species only. We analysed the dynamics of the system mathematically such as, boundedness of the solutions, existence of nonnegative equilibria, local and global stability of the interior equilibrium point by constructing suitable Lyapunov function. Finally we introduced the nonlinear feedback control inputs in the system and stabilized the system. The system appears to exhibit a chaotic behaviour for a range of parametric values. The range of the system parameters for which the system converge the limit cycles is determined. Numerical simulations are carried out to support our results.

**Keywords:** Prey-predator, Stability, Local stability, Global stability, Lyapunov function.

### 1. Introduction

Mathematical models are used extensively in biology and ecology to examine population fluctuations, spread of disease in population, erosion and the spread of pollutants and in its control, to name just a few. Ecological systems may be extraordinarily complex-an inter-related system of plants and animals, prey, predators, insects, parasites etc. One important interaction, where some species use other species as their food supply is the prey-predator interaction. There has been growing interest in the study of three species prey-predator system with infection in prey. Everyone knows that the species does not exist alone in nature and any habitat contain thousands of species. There are possibilities of spreading of the disease in a community more rapidly because of interaction between the species. Hence, it is important to study the rate of transmission of the disease and its control in biology.

Anderson and May (1978) [1], remarked that the infectious diseases can regulate not only their host population but also the other species their host interacts. Anderson and May were the first who merged ecology and epidemiology and formulated a prey-predator model where the prey species were infected by some infectious diseases.

The subject of control of the dynamical system is growing rapidly in many different fields such as ecological models, biological systems, aerospace science, structural engineering and economics [2-8]. The nonlinear feedback controls, adaptive control, positive controls etc.. on prey-predator system has been studied by many authors [2-5]. Eco-epidemiological modelling provides challenges in both applied mathematics and theoretical ecology.

Further, in recent years eco-epidemiological system with disease in prey become most interesting part of research among all mathematical models. Such systems governed mainly by continuous time models and these studies investigate stability, boundedness and persistence. Krishnapada Das et.al and Prasenjet Das et al. [6-7] studied the prey predator system with disease in the predator population and discussed the chaos in this system. Many authors have explored the dynamics of prey- predator system with disease in prey see for example [9-15]. Pierre Auger et al. studied the prey- predator system with disease in predator [16]. The stabilization of prey-predator system with infection or disease in prey has become great interest [12-15]. Also the prey-predator model critically depends on the form of the functional response. In this paper, we apply the Holling type II functional response in the system. Prey-predator models with Holling type II functional response have also been studied extensively in several investigations [17-20].

However, not much work has been dealt with stability analysis of three species continuous time models involving intra-specific competition and mortality with nonlinear feedback controls. The interaction between organisms or species in ecology is called competition. Due to limited resources like food, water, space etc., competition between the species affect the community structure. Intraspecific competition is a particular form of competition in which the members of the same species competes for the same resources in an ecosystem [21-22].

Intraspecific predation, the process of both killing and eating an individual of the same species, is a significant and widespread process that until recently has not received the attention it merits. It is a major factor in the biology of many species and may influence population structure, life history, competition for mates and resources, and behaviour. It is commonly observed among many animals.

In this paper, we focus our attention to observe the effects of intraspecific competition and mortality in the prey-predator model with infection in prey of Vijaya Lakshmi et al [15]. Here, we consider the case where the predator eats the infected prey only because the infected individuals are less active and be caught more easily, for example, in the reference of Peterson and Page [23], they have indicated wolf attacks on moose are more often successful if the moose is heavily infected by "Echinococcus granulosus". We analysed the dynamics of the system mathematically such as, boundedness of the solutions, existence of nonnegative equilibria, local and global stability of the interior equilibrium point by constructing suitable Lyapunov function. Finally we introduced the nonlinear feedback control inputs in the system and stabilized the system. The system appears to exhibit a chaotic behaviour for a range of parametric values. The range of the system parameters for which the system converge the limit cycles is determined. Numerical simulations are carried out to support our results.

This paper is organized as follows: In section 2: we have given the basic model and modified it by introducing mortality rate to infected prey, predator and competition to predator only. In section 3: we prove for the boundedness of the non-dimensionalized model. In section 4: we find out the existence of the equilibrium points. In section 5: Local stability analysis for the trivial, axial and predator free equilibrium points are presented. In section 6: Local stability analysis for coexistent equilibrium point is presented. In section 7: Global stability analysis for the coexistent equilibrium point by constructing suitable Lyapunov function is presented. In section 8: The asymptotic stability of the total system (6) with the nonlinear feedback controls by using suitable Lyapunov function is presented. In section 9: Numerical simulations are carried out to support our analytical results. Finally, the last section 10, is devoted to the conclusion and remarks.

## 2. The Mathematical Model of the System

In this section, we study the dynamics of the continuous time three species prey-predator populations in which we will use the mathematical tools and biological assumptions for modelling the three species prey-predator system which consists of two preys and one predator.

## 2.1 The Basic Model and Assumptions

In this section, we consider the three species prey-predator system which consists of two preys, namely susceptible prey, infected prey (SI) and one predator in which prey species (SI) is infected. And also here we assume that the predator predate on only infected prey [9]. We assume that, the susceptible prey population grows according to a logistic law involving the only susceptible prey species. The transmission rate among the susceptible prey populations and infected prey populations follows the simple law of mass action. The disease is spread among the prey population only and that disease is genetically inherited. The infected prey populations do not recover or become immune. The predator population predate only the infected prey and the functional response is of Michaelis-Menten-Holling- type II. Such system can be described by the following set of nonlinear differential equations:

$$\begin{aligned}\dot{X}_1 &= rX_1 \left(1 - \frac{X_1}{K}\right) - PX_1X_2 \\ \dot{X}_2 &= PX_1X_2 - f(X_2, X_3)X_3 \\ \dot{X}_3 &= ef(X_2, X_3)X_3\end{aligned}\quad (1)$$

where

$X_1(t)$  : the number of the susceptible prey population at time  $t$ ,

$X_2(t)$  : the number of the infected prey population at time  $t$ ,

$X_3(t)$  : the number of the predator population at time  $t$ ,

$r$  : the growth rate of susceptible prey population,

$K$  : the environmental carrying capacity,

$P$  : the rate of transmission from susceptible to infected prey population,

$e$  : the conversion efficiency rate and

Now to formulate the modified mathematical model of a prey-predator system with disease in prey population involving intraspecific competition in predator, we make the following assumptions:

A1. In the absence of infection and predation the susceptible prey population grow logistically with intrinsic growth rate ( $r > 0$ ), carrying capacity ( $K > 0$ ) and then we have

$$\frac{dX_1}{dt} = rX_1 \left(1 - \frac{X_1}{K}\right) \quad (2)$$

A2. In the presence of infection, the prey population is divided into two groups namely susceptible prey denoted by  $X_1(t)$  and infected prey denoted by  $X_2(t)$  at all time  $t$ , the total population is  $N(t) = X_1(t) + X_2(t)$ .

A3. The disease is spread among the prey population only and the disease is not genetically inherited. The infected prey populations do not recover or become immune. We assume that the disease transmission follows the simple law of mass action  $PX_1(t)X_2(t)$  with  $P$  as the transmission rate.

A4. The infected prey  $X_2(t)$  is removed by the death rate  $d_1 (> 0)$  (by natural death of infected prey).

A5. We assume that the predator population consumes only on infected prey with Michaelis Menten-Holling functional response function:

$$f(X_2, X_3) = \frac{\gamma X_2}{X_3 + \gamma\beta X_2}, \quad (\gamma, \beta > 0) \tag{3}$$

That is,  $f$  is the Michaelis-Menten-Holling functional response for infected prey and it is a numerical response for predator. In this  $\gamma$  is the total attack rate for predator or predation coefficient and  $\beta$  is the handling time of predator to prey. The coefficient in conversion prey into predator is  $e$ , where  $0 < e < 1$ .

Also we assume for the predator population:

- (i)  $\xi$  ( $> 0$ ) be the removal rate due to intraspecific competition and
- (ii)  $d_2$  ( $> 0$ ) be the removal rate due to nature death or harvesting.

Therefore the modified of the model (1) becomes:

$$\begin{aligned} \frac{dX_1}{dt} &= rX_1 \left( 1 - \frac{X_1}{K} \right) - PX_1X_2 \\ \frac{dX_2}{dt} &= PX_1X_2 - d_1X_2 - \frac{\gamma X_2X_3}{X_3 + \gamma\beta X_2} \\ \frac{dX_3}{dt} &= \frac{e\gamma X_2X_3}{X_3 + \gamma\beta X_2} - \xi X_3^2 - d_2X_3 \end{aligned} \tag{4}$$

With initial data  $X_1(0) \geq 0, X_2(0) \geq 0, X_3(0) \geq 0$  and the coefficients  $r, K, P, \gamma, \beta, e, d_1, d_2$  and  $\xi$  in model (4) are all positive constants.

**2.2. Nondimensional model**

In the above model, we have specified nine parameters which makes the analysis difficult. Now to reduce the number of the system parameters we will transform the system (4) to the nondimensional form by

using the following transformation of the variables:  $x_1 = \frac{X_1}{K}, x_2 = \frac{X_2}{K}, x_3 = \frac{X_3}{\gamma\beta K}, t = r\tau$

$$\tag{5}$$

The modified Michaelis-Menten-Holling prey predator with vulnerable infected prey dynamics that is, using the transformation (5) the system (4) takes the nondimensional form:

$$\begin{aligned} \frac{dx_1}{dt} &= x_1(1 - x_1) - \kappa x_1x_2 \\ \frac{dx_2}{dt} &= \kappa x_1x_2 - \delta_1x_2 - b \frac{x_2x_3}{x_2 + x_3} \\ \frac{dx_3}{dt} &= c \frac{x_2x_3}{x_2 + x_3} - \xi_1x_3^2 - \delta_2x_3 \end{aligned} \tag{6}$$

where the relations between the nondimensional and dimensional parameters given by:

$$\kappa = \frac{PK}{r}, \quad b = \frac{\gamma}{r}, \quad \delta_1 = \frac{d_1}{r}, \quad \delta_2 = \frac{d_2}{r}, \quad c = \frac{e}{r\beta}, \quad \xi_1 = \frac{\gamma\beta\kappa\xi}{r} \tag{7}$$

The system (6) is more simplicity than (7) for the mathematical study, since the number of system parameters has been reduced from 9 to 6 only.

Now we will analyze the system (6) with the following initial conditions:

$$x_1(0) > 0, x_2(0) > 0, x_3(0) > 0 \tag{8}$$

The conditions (8) represent the conditions of positivity or biologically feasibility of the densities of susceptible prey, infected prey and predator populations respectively.

### 3. Analysis of the Model

#### 3.1 Existence and Dissipativeness

The model system (6) are continuous and have continuous partial derivatives on  $\mathbf{R}_+^3 = \{(x_1, x_2, x_3) \in \mathbf{R}^3 : x_1 \geq 0, x_2 \geq 0, x_3 \geq 0\}$  with interaction functions  $f_i (i=1,2,3)$ . Hence the solution of the system (6) with non-negative initial condition exists and is unique, as the solution of the model system (6) initiating in the non-negative octant is bounded. And also, the system is said to be dissipative that is, all population are uniformly limited in time by the environments, if all population initiating in  $\mathbf{R}_+^3$  are uniformly limited by their environment. The following theorem gives the dissipativeness of model system (6).

**Theorem 1:** All the non-negative solutions of the model system (6) that state in  $\mathbf{R}_+^3$  are uniformly bounded and dissipative.

**Proof:**

Let  $x_1(0) > 0, x_2(0) > 0, x_3(0) > 0$  be any solution of the system with positive initial conditions.

Now we define the function

$$\Omega(t) = x_1(t) + x_2(t) + x_3(t) \tag{9}$$

Therefore, time derivative gives we get

$$\frac{d\Omega}{dt} = \frac{dx_1}{dt} + \frac{dx_2}{dt} + \frac{dx_3}{dt} \tag{10}$$

$$= -\gamma x_1 - \gamma x_2 - \gamma x_3 + (1 + \gamma)x_1 - (x_1^2 + \xi_1 x_3^2) \tag{11}$$

where  $\gamma = \min(1, b, c, \gamma, \gamma_1, \gamma_2)$

Now, choose  $\xi_1 = -\frac{x_1^2}{x_3^2}$ , we get:

$$\frac{d\Omega}{dt} \leq (1 + \gamma) - \gamma\Omega \tag{12}$$

$$\frac{d\Omega}{dt} + \gamma\Omega \leq (1 + \gamma) = \varphi \text{ (say)} \tag{13}$$

$$\frac{d\Omega}{dt} + \gamma\Omega = \varphi \tag{14}$$

Now applying the theory of differential inequality (Birkoff and Rota, 1982), we obtain

$$0 \leq \Omega(x_1, x_2, x_3) \leq \frac{\varphi}{\gamma} + \Omega(x_{1_0}, x_{2_0}, x_{3_0}) e^{-\gamma t} \tag{15}$$

And for  $t \rightarrow \infty$ , we get

$$0 \leq \Omega(x_1, x_2, x_3) \leq \frac{\varphi}{\gamma} \tag{16}$$

Hence, all the solutions of the system (6) that initiate in  $\mathbf{R}_+^3$  are confined in the region B where,

$$B = \left\{ (x_1, x_2, x_3) \in \mathbf{R}_+^3 : 0 \leq \Omega \leq \frac{\varphi}{\gamma} + \varepsilon, \text{ for any } \varepsilon > 0 \right\} \tag{17}$$

Which implies all species are uniformly bounded for any initial value in  $\mathbf{R}_+^3$ . And also according to the above theorem we assume that there exists  $(\alpha_1, \alpha_2, \alpha_3) > 0$  such that  $\Omega(x_{1_0}, x_{2_0}, x_{3_0}) \subset \square_+^3 = \{(x_1, x_2, x_3) : 0 \leq x_1 \leq \alpha_1, 0 \leq x_2 \leq \alpha_2, 0 \leq x_3 \leq \alpha_3\}$ ,  $\forall (x_{1_0}, x_{2_0}, x_{3_0}) \geq 0$  where  $\Omega(x_{1_0}, x_{2_0}, x_{3_0}) \geq 0$  is the omega limit set of the orbit initiating at  $(x_{1_0}, x_{2_0}, x_{3_0})$ . Thus the model system (6) is uniformly limited in time by their environment. This completes the proof.

#### 4. Existence of Equilibria

The existence and stability condition for the system (6) as follows:

- (1) The trivial equilibrium point  $E_0(0,0,0)$  always exists.
- (2) The equilibrium point  $E_1(1,0,0)$  always exists as the prey population grows to the carrying capacity in the absence of predation.
- (3) In the absence of predator species the susceptible and infected prey species can survive.

Hence the equilibrium point  $E_2(\tilde{x}_1, \tilde{x}_2, 0)$  exists in the interior of positive quadrant of  $x_1x_2$  plane, where  $\tilde{x}_1$  and  $\tilde{x}_2$  are given as follows:

$$\tilde{x}_1 = \frac{\delta}{\kappa}, \quad \tilde{x}_2 = \frac{\kappa - \delta_1}{\kappa^2} \tag{18}$$

(4) Neither  $x_1$  nor  $x_3$  can survive in the absence of infected prey species  $x_2$ , hence there is no equilibrium point in  $x_1x_3$  plane. Due to the extinction scenario of susceptible prey  $x_1$ , there is no equilibrium point in  $x_2x_3$  plane.

(5) The positive equilibrium point  $E^*(x_1^*, x_2^*, x_3^*)$  exists in the interior of the first octant if and only if there is a positive solution to the following algebraic non-linear system:

$$\begin{aligned} 1 - x_1 - \kappa x_2 &= 0 \\ \kappa x_1 - \delta_1 - b \frac{x_3}{x_2 + x_3} &= 0 \\ c \frac{x_2}{x_2 + x_3} - \xi_1 x_3 - \delta_2 &= 0 \end{aligned} \tag{19}$$

**5. Stability Analysis of Boundary Equilibria**

In this section, we study the stability of the boundary equation points of the system (6).

The Variation matrix  $V(x_1, x_2, x_3)$  associated with model system (6) evaluated at  $(x_1, x_2, x_3)$  is given by

$$V_{(x_1, x_2, x_3)} = \begin{bmatrix} v_{11} & v_{12} & 0 \\ v_{21} & v_{22} & v_{23} \\ 0 & v_{32} & v_{33} \end{bmatrix} \tag{20}$$

$$v_{11} = 1 - 2x_1 - \kappa x_2; \quad v_{12} = -\kappa x_1$$

Where  $v_{21} = \kappa x_2; \quad v_{22} = \kappa x_1 - \delta_1 - b \frac{x_3^2}{(x_2 + x_3)^2}; \quad v_{23} = -b \frac{x_2^2}{(x_2 + x_3)^2};$

$$v_{32} = c \frac{x_3^2}{(x_2 + x_3)^2}; \quad v_{33} = c \frac{x_2^2}{(x_2 + x_3)^2} - 2\xi_1 x_3 - \delta_2$$

**Lemma 1**

The trivial equilibrium point  $E_0$  is locally asymptotically stable in the  $x_2x_3$  direction and is unstable otherwise.

**Proof:**

The system (6) cannot linearised at  $E_0$  and therefore here we discuss the local stability of  $E_0$  with intraspecific competition in predator.

The variation matrix  $V(E_0)$  at the equilibrium point  $E_0$  is

$$V(E_0) = \begin{bmatrix} 1 & 0 & 0 \\ 0 & -\delta_1 & 0 \\ 0 & 0 & -\delta_2 \end{bmatrix} \tag{21}$$

Since  $\lambda_2, \lambda_3$  are negative, hence  $E_0$  is asymptotically stable in the  $x_2x_3$  direction and since  $\lambda_1$  is positive  $E_0$  is unstable in  $x_1$  plane. Hence the Lemma.

**Lemma 2**

The boundary equilibrium point  $E_1$  is asymptotically stable in the  $x_1x_3$  plane and  $E_1$  is asymptotically stable in  $x_2$  direction if the transmission rate less than the ratio of the death rate of infected prey to the carrying capacity, otherwise unstable.

**Proof:**

The variation matrix  $V(E_1)$  at the equilibrium point  $E_1$  is

$$V(E_1) = \begin{bmatrix} -1 & -\kappa & 0 \\ 0 & \kappa - \delta_1 & 0 \\ 0 & 0 & -\delta_2 \end{bmatrix} \tag{22}$$

Here we easily say that  $E_1$  is locally asymptotically stable in the  $x_1x_3$  direction and  $E_1$  is asymptotically stable only if  $\delta_1 = \kappa$ , that is  $P < \frac{d_1}{\kappa}$  in  $x_2$  plane. Hence the Lemma.

**Lemma 3**

The predator free equilibrium point  $E_2(\tilde{x}_1, \tilde{x}_2, 0)$  exists if and only if  $\delta_2 > c$ , when this condition is satisfied  $\tilde{x}_1$  and  $\tilde{x}_2$  are given follows:

$$\tilde{x}_1 = \frac{\delta}{\kappa}, \quad \tilde{x}_2 = \frac{\kappa - \delta_1}{\kappa^2} \tag{23}$$

that is,  $E_2(\tilde{x}_1, \tilde{x}_2, 0)$  is asymptotically stable in  $x_3$  direction only if the ratio of the efficiency conversion of predator to the death rate of predator is less than the handling time, otherwise unstable.

**Proof:**

The variation matrix  $V(E_2)$  at the equilibrium point  $E_2$  is

$$V(E_2) = \begin{bmatrix} -\frac{\delta_1}{\kappa} & \delta_1 & 0 \\ 1 - \frac{\delta_1}{\kappa} & 0 & -b \\ 0 & 0 & c - \delta_2 \end{bmatrix} \tag{24}$$

The characteristic equation of  $V(E_2)$  is

$$(\lambda^2 + A_1\lambda + A_2)(\lambda - c + \delta_2) = 0 \tag{25}$$

Where  $A_1 = \frac{\delta_1}{\kappa} > 0$  and  $A_2 = \frac{\delta_1^2}{\kappa} - \delta_1 > 0$

Therefore, the eigen values are

$\lambda_{1,2} = \frac{-A_1 \pm \sqrt{A_1^2 - 4A_2}}{2}$  and  $\lambda_3 = c - \delta_2$ , since  $A_1 > 0$  and  $A_2 > 0$ , thus the signs of the real part of  $\lambda_1, \lambda_2$  are negative implies that  $E_2$  is locally asymptotically stable in the  $x_1x_2$  -plane.

Now  $E_2$  is locally asymptotically stable in the  $x_3$  direction only if  $\delta_2 > c$ , that is  $\frac{e}{d_2} < \beta$ . Hence the Lemma.



**6. The Interior Equilibrium Point: its Existence and Stability**

**6.1 The Necessary and sufficient conditions for the existence of  $E^*$**

Now let us we consider the existence and uniqueness of the interior equilibrium point  $E^*(x_1^*, x_2^*, x_3^*)$ .

**Lemma 4:** Assume that (C1), (C2) and (C3) hold. That is, the equilibrium point of  $E^*(x_1^*, x_2^*, x_3^*)$  of the system (6) exists if and only if, the following two conditions are satisfied.

(C1)  $x_2^* < \frac{1}{\kappa}$

(C2)  $\alpha_1, \alpha_2 < 0$

(C3)  $\alpha_3, \alpha_4 < 0$

Furthermore  $x_1^*, x_2^*, x_3^*$  are given below:

$x_1^* = 1 - \kappa x_2^*$ ;  $x_2^* = S_1$ ;  $x_3^* = S_2$ , in this

$$S_1 = \frac{-\alpha_1 \pm \sqrt{\alpha_1^2 - 4\alpha_2}}{2}; \quad S_2 = \frac{-\alpha_3 \pm \sqrt{\alpha_3^2 - 4\alpha_4}}{2},$$

$\alpha_1 = \left(\frac{\delta_1}{\kappa} - 1 + x_3^*\right)$ ;  $\alpha_2 = \left(\frac{b - \delta_1 - \kappa}{\kappa}\right)x_3^*$  and

$\alpha_3 = \left(\frac{\xi_1 S_1 + \delta_2}{\xi_1}\right)$ ;  $\alpha_4 = \left(\frac{\delta_2 - c}{\xi_1}\right)S_1$

In terms of original parameters of the system, the conditions (C1) and (C2) respectively becomes  $x_2^* < \frac{1}{\kappa}$ ,  $x_3^* < 1 - \frac{\delta_1}{\kappa}$ ;  $\kappa > b - \delta_1$ ; and  $\xi_1 S_1 + \delta_2 < 0$ ;  $\delta_2 < c$  respectively, which are the necessary and sufficient conditions for the co-existence of the susceptible prey, infected prey and predator.

**6.2 Local stability analysis of Interior Equilibrium Point  $E^*$**

The variation matrix of (3) at  $E^*$  is given below:

$$V(E^*) = \begin{bmatrix} v_{11}^* & v_{12}^* & 0 \\ v_{21}^* & v_{22}^* & v_{23}^* \\ 0 & v_{32}^* & v_{33}^* \end{bmatrix} \tag{26}$$

$v_{11}^* = 1 - 2x_1^* - \kappa x_2^*$ ;  $v_{12}^* = -\kappa x_1^*$

where  $v_{21}^* = \kappa x_2^*$ ;  $v_{22}^* = \kappa x_1^* - \delta_1 - b \frac{x_3^{*2}}{(x_2^* + x_3^*)^2}$ ;  $v_{23}^* = -b \frac{x_2^{*2}}{(x_2^* + x_3^*)^2}$ ;

$v_{32}^* = c \frac{x_3^{*2}}{(x_2^* + x_3^*)^2}$ ;  $v_{33}^* = c \frac{x_2^{*2}}{(x_2^* + x_3^*)^2} - 2\xi_1 x_3^* - \delta_2$

The characteristic equation is

$$\lambda^3 + Q_1\lambda^2 + Q_2\lambda + Q_3 = 0 \quad (27)$$

$$\text{where } Q_1 = -\text{tr}[V(E^*)] = -v_{11}^* - v_{22}^* - v_{33}^*$$

$$= -(D+L) ;$$

$$Q_2 = v_{11}^*v_{22}^* + v_{11}^*v_{33}^* + v_{22}^*v_{33}^* - v_{23}^*v_{32}^* - v_{12}^*v_{21}^*$$

$$= DL + (D+L)y_3 + \kappa^2 x_1^* x_2^* + y_1 y_2$$

$$Q_3 = -\det[V(E^*)] = v_{11}^*v_{23}^*v_{32}^* + v_{12}^*v_{21}^*v_{33}^* - v_{11}^*v_{22}^*v_{33}^*$$

$$= D - (\kappa^2 x_1^* x_2^* + Ly_3 + y_1 y_2 y_3)$$

in this,

$$y_1 = b \frac{x_3^{*2}}{(x_2^* + x_3^*)^2}; \quad y_2 = c \frac{x_2^{*2}}{(x_2^* + x_3^*)^2}; \quad y_3 = 1 - 2x_1^* - \kappa x_2^* ;$$

$$D = \kappa x_1^* - \delta_1 - y_1; \quad L = y_2 - 2\xi_1 x_3^* - \delta_2;$$

Now

$$\Delta = Q_1 Q_2 - Q_3 \quad (28)$$

$$\begin{aligned} &= -(v_{11}^*)^2 v_{22}^* - (v_{11}^*)^2 v_{33}^* + v_{11}^* v_{12}^* v_{21}^* - (v_{22}^*)^2 v_{33}^* - (v_{22}^*)^2 v_{11}^* \\ &\quad - 2v_{11}^* v_{22}^* v_{33}^* + v_{22}^* v_{12}^* v_{21}^* + v_{23}^* v_{32}^* v_{22}^* - (v_{33}^*)^2 v_{22}^* - (v_{33}^*)^2 v_{11}^* \\ &\quad + v_{23}^* v_{32}^* v_{33}^* \end{aligned} \quad (29)$$

$$= \kappa^2 x_1^* x_2^* (1 - D - L) + y_3 (y_1 y_2 - (D+L)^2 + L) - (D + (D+L)(DL + y_1 y_2)) \quad (30)$$

we discuss the local stability of their interior equation point  $E^*$ .

### Theorem 2:

The interior equilibrium point  $E^*$  is locally asymptotically stable if and only if

$$D+L < 0 \quad \text{and} \quad \Delta > 0 \quad (31)$$

### Proof:

We notice that

$$1. \quad D+L < 0 \Leftrightarrow Q_1 > 0$$

2.  $Q_3 > 0$ , for all values of the parameters, and
3.  $\Delta Q_1 Q_2 - Q_3 > 0$

According to Routh Hurwitz criterion, the theorem proved.

**7. Global Stability Analysis of Interior Equilibrium Point  $E^*$**

In this section, we shall study the global dynamics of the system (6) around the positive equilibrium point  $E^*(x_1^*, x_2^*, x_3^*)$ . We construct the suitable Lyapunov function to prove the global stability for the system (6).

**Theorem 3:**

Assume that the positive equilibrium point  $E^*(x_1^*, x_2^*, x_3^*)$  is locally asymptotically stable then, it is a globally asymptotically stable in the interior of positive octant assuming the below two conditions:

- (i) Choose carrying capacity and the strength of intraspecific competition rate as one.
- (ii) Choose  $b = (x_2 - x_2^*)^2 + c$

**Proof**

In order to prove the global stability, we define the following Lyapunov function

$$F(x_1^*, x_2^*, x_3^*) = F_1(x_1^*, x_2^*, x_3^*) + F_2(x_1^*, x_2^*, x_3^*) + F_3(x_1^*, x_2^*, x_3^*) \tag{32}$$

where  $F_1 = x_1 - x_1^* - x_1^* \ln\left(\frac{x_1}{x_1^*}\right)$

$$F_2 = x_2 - x_2^* - x_2^* \ln\left(\frac{x_2}{x_2^*}\right)$$

$$F_3 = x_3 - x_3^* - x_3^* \ln\left(\frac{x_3}{x_3^*}\right)$$

Now in order to investigate the global dynamics of the non-negative equilibrium point  $E^*(x_1^*, x_2^*, x_3^*)$  of the model system (6), the derivative of  $F$  with respect to time along the solution of the system (6) is computed as

$$\frac{dF}{dt} = \frac{dF_1}{dt} + \frac{dF_2}{dt} + \frac{dF_3}{dt} \tag{33}$$

The time derivative of the above function will be

$$\dot{F}(t) = z_1 \frac{\dot{x}_1}{x_1} + z_2 \frac{\dot{x}_2}{x_2} + z_3 \frac{\dot{x}_3}{x_3} \tag{34}$$

where  $z_1 = (x_1 - x_1^*)$ ,  $z_2 = (x_2 - x_2^*)$  and  $z_3 = (x_3 - x_3^*)$

Using the set of equations (6) and (34) we obtain

$$\dot{F}(x_1, x_2, x_3) = -x_1 z_1 - \kappa x_2 z_1 + \kappa x_1 z_2 - \frac{bx_3}{x_2 + x_3} z_2 + \frac{cx_2}{x_2 + x_3} z_3 - \xi_1 x_3 z_3$$

Now choosing the condition (i) , that is  $\kappa = \xi_1 = 1$  and (ii) which is given in the theorem statement, we get

$$\dot{F}(x_1, x_2, x_3) = -(x_1 - x_1^*)^2 - (x_2 - x_2^*)^2 - (x_3 - x_3^*)^2 \leq 0 \tag{35}$$

Therefore  $\dot{F}(x_1, x_2, x_3)$  is negative definite if the above conditions of the theorem are satisfied and consequently,  $F$  is a Lyapunov function with respect to all solutions in the interior of the positive octant, which proves the theorem.

**8. Non Linear Feedback Control**

**8.1 Problem Statement for Non Linear Feedback Control**

Consider the chaotic system described by the dynamics

$$\dot{x} = Ax + f(x) + u \tag{36}$$

where  $x \in R^n$  is the state of the system,  $A$  is the  $n \times n$  matrix of the system parameters, the matrix  $A$  have some unknown parameters.  $f : R^n \rightarrow R^n$  is the nonlinear part of the system.  $u \in R^n$  is the adaptive feedback controller.

The global control problem is essentially to find feedback controller  $u$  , so as to stabilize the dynamics (36) for all initial conditions  $x(0) \in R^n$  , i.e.  $\lim_{t \rightarrow \infty} \|x(t)\| = 0$  for all initial conditions  $x(0) \in R^n$  .

Lyapunov function methodology is used for establishing the feedback control of the system (36).

By the Lyapunov function methodology, a candidate Lyapunov function is taken as

$$V(x) = x^T Px \tag{37}$$

where  $P$  are  $n \times n$  positive definite matrix.

Note that  $V : R^n \rightarrow R^n$  is a positive definite function by construction. It is assumed that the parameters of the system (36) are measurable.

If a controller  $u$  found such that

$$\dot{V}(x) = -x^T Qx \tag{38}$$

where  $Q$  are positive definite matrix, then  $\dot{V}$  is a negative definite function.

Hence, by Lyapunov stability theory Hahn, (1967) [24], the dynamics (36) is globally exponentially stable and hence the condition  $\lim_{t \rightarrow \infty} \|x(t)\| = 0$  will be satisfied for all initial conditions  $x(0) \in R^n$  .

Then the states of the system (36) will be globally asymptotically stable.

**8.2 A Prey Predator Model With Nonlinear Feedback Controls**

**Theorem 4:** The dynamics of the modified Michaelis-Menten-Holling type II prey- predator system with predator competition involving infected prey is asymptotically stable with the following nonlinear controls:

$$u_1 = \kappa x_1 x_2 - 2x_1 + x_1^2 \tag{39}$$

$$u_2 = \frac{bx_2 x_3}{x_2 + x_3} - \kappa x_2 x_3 \tag{40}$$

$$u_3 = \xi_1 x_3^2 - \frac{cx_2 x_3}{x_2 + x_3} \tag{41}$$

**Proof:**

The modified prey predator model with competition in predator involving infected prey (6) with the nonlinear feedback controls is described by:

$$\begin{aligned} \frac{dx_1}{dt} &= x_1(1 - x_1) - \kappa x_1 x_2 + u_1 \\ \frac{dx_2}{dt} &= \kappa x_1 x_2 - \delta_1 x_2 - b \frac{x_2 x_3}{x_2 + x_3} + u_2 \\ \frac{dx_3}{dt} &= c \frac{x_2 x_3}{x_2 + x_3} - \xi_1 x_3^2 - \delta_2 x_3 + u_3 \end{aligned} \tag{42}$$

where  $x_1, x_2, x_3$  are the state variables and  $\kappa, b, \delta_1, \delta_2, c, \xi_1$  are positive parameters.

In this paper, we introduce the nonlinear feedback procedure to design the controllers  $u_1, u_2, u_3$ , where  $u_1, u_2, u_3$  are feedback controllers, which is the function of the state variables. As long as these feedbacks stabilize system (42) converge to zero as the time  $t$  goes to infinity. That means that, this gives the system (42)  $\lim_{t \rightarrow \infty} \|x(t)\| = 0$

The candidate Lyapunov function is taken as

$$G(x_1, x_2, x_3) = \frac{1}{2} x_1^2 + \frac{1}{2} x_2^2 + \frac{1}{2} x_3^2 \tag{43}$$

Differentiating (43) along the trajectories of the system (42), the simple calculation gives

$$\begin{aligned} \dot{G}(x_1, x_2, x_3) &= x_1(x_1(1 - x_1) - \kappa x_1 x_2 + u_1) \\ &+ x_2 \left( \kappa x_1 x_2 - \delta_1 x_2 - b \frac{x_2 x_3}{x_2 + x_3} + u_2 \right) \\ &+ x_3 \left( c \frac{x_2 x_3}{x_2 + x_3} - \xi_1 x_3^2 - \delta_2 x_3 + u_3 \right) \end{aligned} \tag{44}$$

Substituting equation (39),(40) and (41) into (44), then it implies that

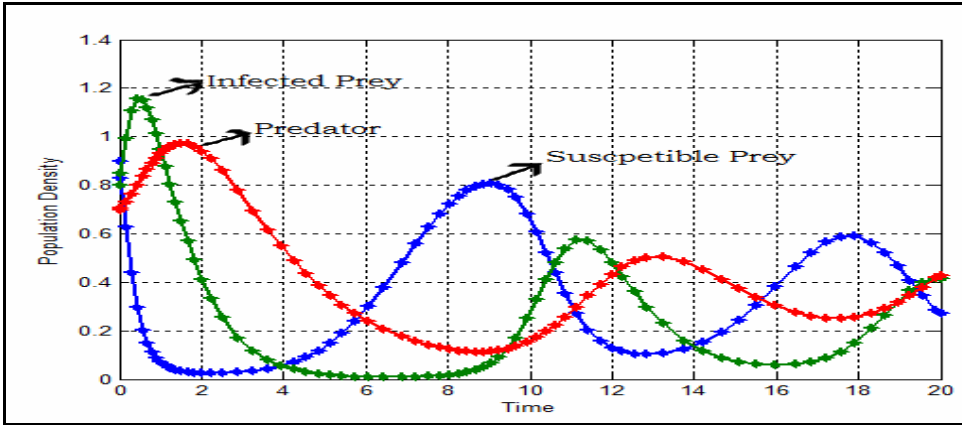
$$\dot{G}(x_1, x_2, x_3) = -x_1^2 - \delta_1 x_2^2 - \delta_2 x_3^2 \tag{45}$$

which is a negative definite function.

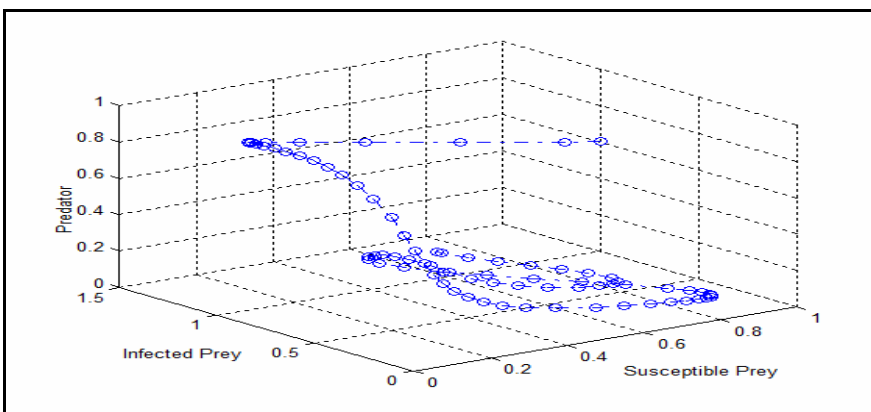
Thus by Lyapunov stability theory [33], the modified Michaelis-Menten-Holling prey predator with competition and mortality in predator involving infection and mortality in prey dynamics (6) is asymptotically stable.

**9. Numerical Simulation**

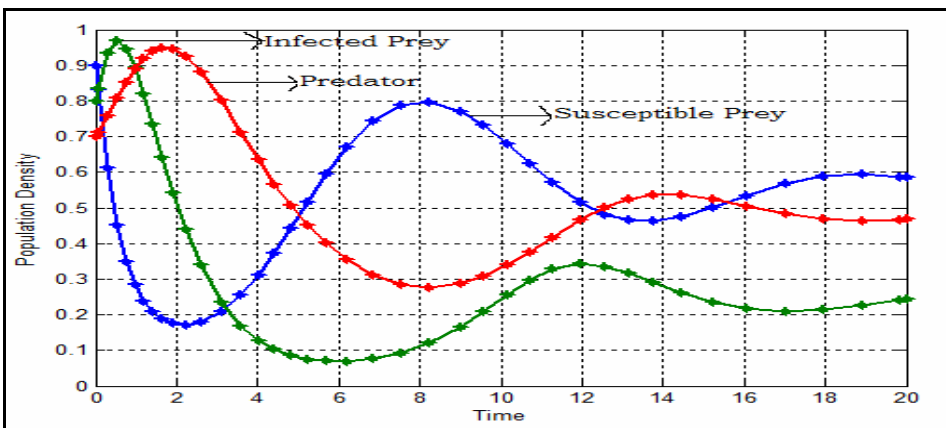
We perform the numerical simulations of systems (6) and (42) with the following set of parameters and explaining their complex dynamical nature. The phase portraits and the corresponding time series graph are obtained for the systems (6) and (42). We have fixed the parameter  $b = 0.9$ ;  $c = 1.5$ ;  $\delta_1 = 0.4$ ;  $\delta_2 = 0.4$ ;  $\xi_1 = 0.2$  and the initial densities  $x_1 = 0.9$ ;  $x_2 = 0.8$ ;  $x_3 = 0.7$ ;



**Figure 1 :** Time series of the trajectory of the system (6) approaches asymptotically to (0.2716, 0.4137, 0.4293) for  $\kappa = 3$



**Figure 2:** Phase portrait when  $\kappa = 3$  for the system (42) approaches asymptotically to the point (0.2716, 0.4137, 0.4293)



**Figure 3:** Time series of the trajectory of the system (6) approaches asymptotically to (0.5854, 0.2438, 0.4689) for  $\kappa = 1.8$

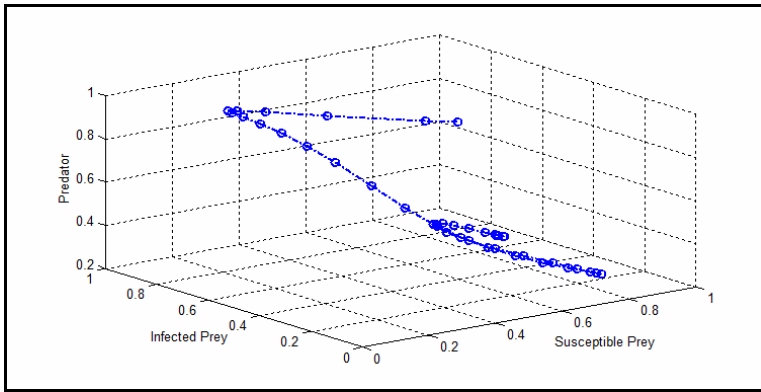


Figure 4 : Phase portrait when  $\kappa = 1.8$  for the system (42) approaches asymptotically to the point  $(0.5854, 0.2438, 0.4689)$

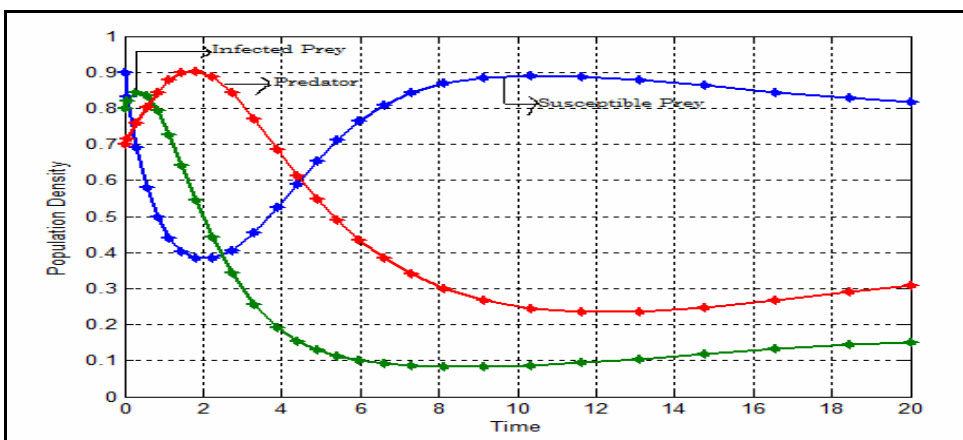


Figure 5 : Time series of the trajectory of the system (6) approaches asymptotically to  $(0.8174, 0.1515, 0.3081)$  for  $\kappa = 1.25$

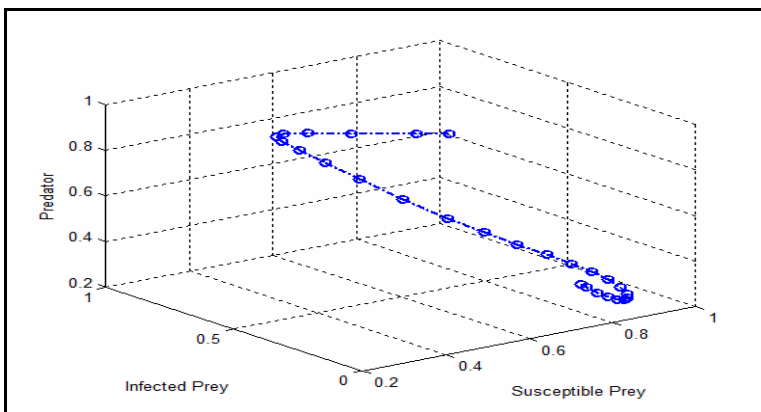


Figure 6 : Phase portrait when  $\kappa = 1.25$  for the system (42) approaches asymptotically to the point  $(0.8174, 0.1515, 0.3081)$

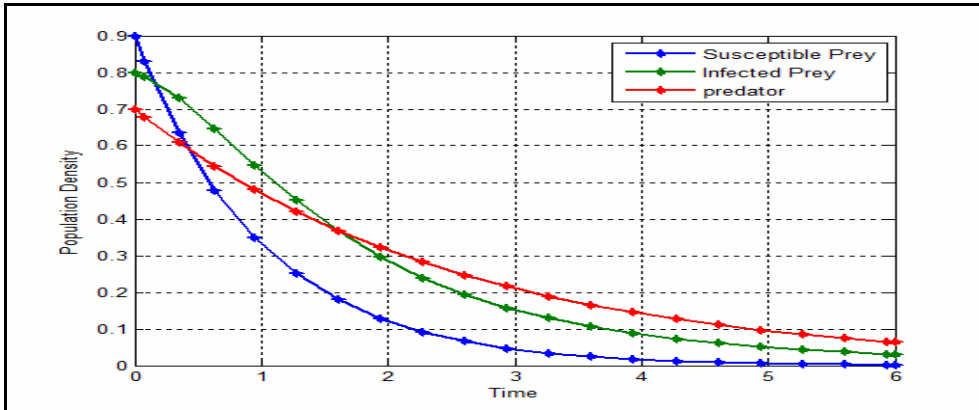


Figure 7 : Time series of the trajectory of the system (42) approaches asymptotically to (0.0022,0.0305,0.0635) for  $\kappa = 1.25$

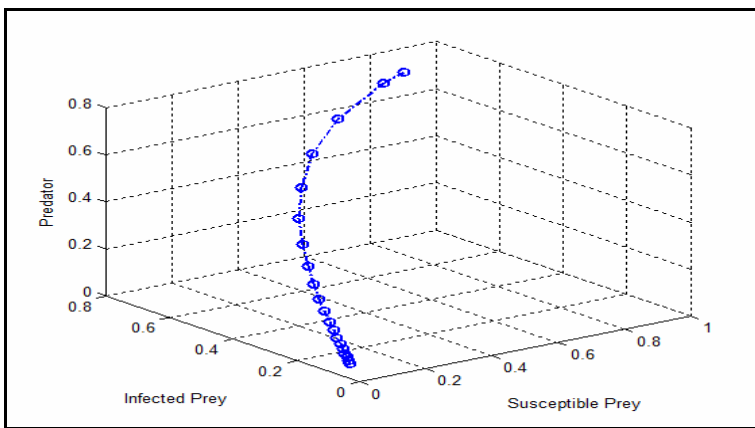


Figure 8 : Phase portrait when  $\kappa = 1.25$  for the system (42)

In the above numerical simulations, we keep the parameters  $b = 0.9$ ;  $c = 5$ ;  $\delta_1 = 0.4$ ;  $\delta_2 = 0.4$ ;  $\xi_1 = 0.2$  fixed with the initial densities  $x_1 = 0.9$ ;  $x_2 = 0.8$ ;  $x_3 = 0.7$ ; and just varying the disease transmission rate  $\kappa$ . The figure 1, shows the population variation when  $\kappa = 3$  approaches the stable point (0.2716, 0.4137, 0.4293) and figure 2 gives the corresponding phase plot. Now in figure3, we observe, when the disease transmission rate is decreased to  $\kappa = 1.8$ , the density of infected prey also decreased to 0.4137 to 0.2438 and the density of susceptible prey increased from 0.2716 to 0.5854. Figure 4 gives the corresponding phase plot.

When the disease transmission rate again decreased to  $\kappa = 1.25$ , in figure 5, we easily observe that the densities of infected prey and predator decreased and there is a increase in the density of susceptible prey. Figure 6 gives the corresponding phase plot.

In Figure 7, the population densities approaches the stable point (0.0022,0.0305,0.0635) quickly for the system (42) with nonlinear feedback controls for the same parameters which is mentioned above for any one of the disease transmission values  $\kappa = 3$   $\kappa = 1.8$   $\kappa = 1.25$ .

The above numerical simulations show the dynamic effect of the disease for the prey-predator system. The comparative analysis, that is, the figures between uncontrolled (6) and controlled system (42) proves our analytical results.

### 10. Conclusion

In this paper, we have considered the three species prey-predator system consists of susceptible, infected prey and predator involving mortality in both species. In this we have introduced intraspecific



competition for the predator at the rate of  $\xi_1$  and also the mortality rates  $d_1$  and  $d_2$  for the infected prey and predator respectively. Incorporating the mortality in infected prey and predator, intraspecific competition in predator in the system (6) provides a more realistic model. An intraspecific competition and mortality can be important for the biological control of prey-predator population, however, increasing the amount of competition of predators can increase prey densities and lead to population outbreaks. The non-dimensionalised system (6) is uniformly bounded, which implies that the system is biologically well behaved is shown in theorem 1. Although, most of the prey-predator models with prey infection we observe in nature correspond to stable equilibria of the models, we have presented the trivial, boundary, predator free and coaxial equilibrium points. The stable criteria given in Lemma 1, 2 and 3 are the conditions for the asymptotic stability of the equilibrium points  $E_0$ ,  $E_1$  and  $E_2$ . Now we observed that the predator free equilibrium  $E_2$  exists if and only if  $\frac{e}{d} < \beta$ , which implies that if the ratio of the efficiency conversion of predator to the death rate of predator is less than the handling time, then the predator becomes extinct and conversely.

The stable criteria given in Lemma 4 and Theorem 2 are the conditions for the asymptotic stable coexistence of the susceptible prey, infected prey and predator population  $E^*$ . Now we observe that the interior equilibrium point  $E^*$  exists if the conditions  $\kappa < \frac{1}{x_2^*}$ ;  $\kappa < \frac{\delta_1 + b}{1 - x_2^*}$  and  $\delta_2 + \xi_1 S_2 < 0$ ;  $\delta_2 < c$  are satisfied and also we have show  $E^*$  is asymptotically stable under Routh Hurwitz criterion. The global stability analysis of the coaxial equilibrium point has been presented in the Theorem 3 by constructing the necessary Lyapunov function.

Next the problem of nonlinear feedback control of three species consisting of susceptible prey, infected prey and predator is studied. The asymptotic stability of the controlled system is proved by using suitable Lyapunov function. The necessary control inputs for stabilization is obtained as nonlinear feedback. Finally, extensive numerical examples and simulations are presented by using MATLAB software which supports our results. The numerical simulation were designed to observe the effect of the disease on the three species and it was found that if the contact rate between susceptible prey and infected prey decreased the density of susceptible prey increased and at the same time density of predator decreased, which proves our result.

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