DISEASE AND ALTERNATIVE FOOD IN THE INTERMEDIATE PREDATOR STABILIZE CHAOTIC DYNAMICS-CONCLUSION DRAWN FROM A TRI-TROPHIC FOOD CHAIN MODEL

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ABSTRACT

Parasite are an integral part of virtually all food webs and species communities. Here we have considered a tri-trophic food chain model with disease circulating in the intermediate predator and there is an alternative food source of the intermediate predator. We have analyzed the local stability of the model system around the biologically feasible equilibria. We also introduce the ecological as well as the disease basic reproduction numbers and analyzed the community structure of the model systems by the help of these numbers. We have worked out theconditions of persistence of the model system. Our numerical results reveals that in low infection level the system shows chaotic dynamics but in high infection level chaotic dynamics becomes stable focus. So our finding contradicts predictions from previous models suggesting a destabilizing effect of parasites. We have also observed the role of alternative food on chaotic dynamics. When the alternative food increases the chaotic dynamics remain same but when the alternative food decreases chaos disappears and the infected intermediate predator is washed out from the system.

KEYWORDS

Disease in intermediate predator, chaos, period-doubling, limit cycle, stable focus and alternative food.

1.INTRODUCTION

Both the theoretical ecology and the theoretical epidemiology are developed research fields and are treated separately. However, there are some common features between these two systems and merging the two areas may show interesting dynamics. Eco-epidemiology is a branch in mathematical biology which considers both the ecological and epidemiological issues simultaneously. Anderson and May [1] were the first who merged the above two fields and formulated a predator-prey model where prey species were infected by some disease. In the subsequent time, many researchers have proposed and studied different predator-prey models in

the presence of disease. The literature in the field of eco-epidemiology has grown enormously in the last two decades and we are mentioning few of them [7, 10, 13, 18, 37, 40].

Most of the earlier studies [11, 19, 43] pay attention to the disease transmission in prey population only. To the best our knowledge, the influence of predation on epidemics has not yet been studied considerably, except the works of Anderson and May[2], Hadeler and Freedman[18], Hochberg[34],Venturino [41, 42], Chattopadhyay and Arino [7], Han et al.[16], Xiao and Chen[43], Hethcote et al.[19] Greenhalgh and Haque[37], Haque and Venturino[36]. Most of these works have dealt with predator prey models with disease in the prey (except Venturino[40], Haque and Venturino[36]). But the study of a predator prey system with an infected predator has a great importance so long as the question of the predator control is concerned. To the best of our knowledge, mathematical epidemiology almost remained silent in this issue. But as far as our knowledge goes a little effort has been made to understand such dynamics in tri-trophic food web with disease in the intermediate-predator. Here we consider a tri-trophic food chain model with an infectious disease circulating in the intermediate-predator population and we also consider the alternative food of the intermediate predator.

In the present study we pay attention to the chaotic dynamics for variation of the force of infection. Most of the earlier works are based on finding the stability and persistence of a system [38] and are analyzed by considering linear approximation to the non-linear equations that ecologists conventionally assume to the more complex situations[17]. But now the scenario is changing. The terms chaos, strange attractor, fractal are becoming familiar to many if not all, ecologists[35]. Although chaos is commonly predicted by mathematical models, evidence for its existece in the natural world is scarce and inconclusive[8]. Chaotic dynamics is common in tri-trophic food chain and is of common interest to both modelers and experimental ecologists. Hastings and Powell (HP) [17] produced a chaotic population system in a simple tri-trophic food chain model with type-II functional response. McCann and Yodzis [4] reformulated Hastings and Powell's model[17] and showed that the system produce chaotic dynamics for plausible combination of parameter values. Not only in ecological systems, but chaos can also occur in epidemiological systems. Extensive analysis have suggested that measles is a very good candidate for chaos, though there is as yet no unequivocal evidence [33, 39, 31]. Grenfell et al. [32] investigated the effects of locally chaotic dynamics on global persistence in the standard epidemiological model[3]. Though the study of chaos in an eco-epidemiological system is new but the literature of chaos in ecological system is very rich. Recently Chatterjee et al. [6] observed that the rate of infection and the predation rate are two prime factors that govern the chaotic dynamic in eco-epidemiological system. We investigate the chaotic dynamics in our model for variation of the force of infection.

In this study another important factor is alternative food of the intermediate predator. In nature, when prey population falls below a certain level, the predator would migrate to another region and the ecological system would collapse. It is argued that alternative food may play an important role in promoting the persistence of prey - predator system. Again alternative food is very important for survival of consumer species in an ecosystem. In scientific literature, these foods fundamentally shape the life histories of many predator species. Alternative food source for the intermediate predator is another important factor in the ecological species interaction and inclusion of this factor in eco-epidemiological studies might give some interesting results. Predators do not generally feed on a single prey species, but will switch to the alternative food sources when the density of the preferred prey is low [18]. It is also well-known fact that foraging

theory that when prey density drops below a threshold value due to infection in the preferred prey population or any other cause, optimally foraging predators will switch to alternative food sources, either by including the alternative food source in their diet (in a fine-grained environment) or by moving to the alternative food source (in a coarse-grained environment). Charnov[5] published a well-known model of optimal diets that consists of a stepwise switch from a diet of profitable prey only to mixed diet including alternative food. Fryxell and Lundberg[14] have demonstrated, using numerical simulation studies of one-predator/two-prey models, that the predator will switch to low-quality prey only when they have reduced the more profitable prey to low levels. They also observed that when prey density is low, such a switch will diminish predation pressure on the profitable prey. The presence of alternative food for a predator leads to reduction in equilibrium prey densities, and is termed as apparent competition[23].

The present investigation is an attempt to observe chaotic dynamics in the system and finally we observe the role of alternative food on chaotic dynamics. Another investigation is community structure of our model system. For this we introduce the concept of the ecological as well as the disease basic reproduction numbers. The reproduction number can be defined as the expected number of offspring a typical individual produces in its life or in epizootiology as the expected number of secondary infections produced by a single infective individual in a completely susceptible population during its entire infectious period. While the concept of the reproduction numbers was initially developed in demography already in the early 20th century [25], they became a standard tool in epidemiology since the work of Anderson and May [3] and Diekmann et al.[9]. We will use reproduction numbers as helpful tools in determining the persistence or extinction of a species. This will allow us to categorize the community composition of prey, predators and disease. The threshold concept inherent in reproduction numbers has been used in previous studies of eco-epidemiological models[18, 16, 43, 19].

The paper is organized as follows. In the section(2), we outline the mathematical model with some basic assumption. We study the stability of the equilibrium points and Hopf bifurcation in section(3) and the permanence and impermanence of the system in section(4). We perform an extensive numerical simulation in section (5). The article ends with a discussion

2 MODEL FORMULATION

The HP model [17] with pairwise interactions between three species, namely, X, Y, Z, which incorporates a Holling type II functional response in both consumers species, namely Y and Z is as follows:

$$\begin{aligned} \frac{dX}{dT} &= R_0X\left(1 - \frac{X}{K_0}\right) - \frac{A_1XY}{B_1 + X} \\ \frac{dY}{dT} &= \frac{e_1A_1XY}{B_1 + X} - \frac{A_2YZ}{B_2 + Y} - D_1Y \\ \frac{dZ}{dT} &= \frac{e_2A_2YZ}{B_2 + Y} - D_2Z \end{aligned} \tag{1}$$

Here X is the numbers of the species at the lower level of the food chain, Y the numbers of the species that preys upon X, and Z the numbers of the species that preys upon Y. Here T is time. The constant R_0 is the intrinsic growth rate and the constant K_0 is the carrying capacity of the species X. The constants e_1 and e_2 are food conversion efficiency for the species Y and Z

respectively; D_1 and D_2 are constant death rates for species Y and Z respectively. The constants A_i and B_i for i = 1, 2 are maximal predation rate and half saturation constants for Y and Z respectively. Hastings and Powell[17] demonstrated that the dynamic interaction between prey and predators in simple three-species food chain is chaotic in a certain region of parametric space.

The parasite is assumed to be horizontally transmitted. We further assume that the parasite attacks the predator population only. Disease is transmitted in predator population at the rate λ following the mass action law.

By above assumptions the system(1) can be written as the following set of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dX}{dT} &= R_0X\left(1 - \frac{X}{K_0}\right) - \frac{A_1X(Y_1 + Y_2)}{B_1 + X} \\
\frac{dY_1}{dT} &= R_1Y_1\left(1 - \frac{Y_1}{K_1}\right) + \frac{e_1A_1X(Y_1 + Y_2)}{B_1 + X} - \frac{A_2Y_1Z}{B_2 + Y_1} - \lambda Y_1Y_2 - D_1Y_1 \qquad (2) \\
\frac{dY_2}{dT} &= \lambda Y_1Y_2 - \frac{A_2Y_2Z}{B_2 + Y_2} - D_2Y_2 \\
\frac{dZ}{dT} &= \frac{Ze_2A_2Y_1}{B_2 + Y_1} + \frac{Ze_3A_2Y_2}{B_2 + Y_2} - D_3Z
\end{aligned}$$

To reduce the number of parameters and to determine which combinations of parameters control the behavior of the system, we dimensionalize the system with the following scalling:

$$N = \frac{X}{K_0},\ S = \frac{Y_1}{K_0},\ I = \frac{Y_2}{K_0},\ P = \frac{Z}{K_0} \text{ and } t = R_0T.$$

Then the system (2) takes the form (after some simplification)

$$\begin{aligned}
\frac{dN}{dt} &= N(1 - N) - \frac{a_1N(S + I)}{1 + b_1N} \\
\frac{dS}{dt} &= S\left(r - d_1 - \frac{S}{k}\right) + \frac{e_1a_1N(S + I)}{1 + b_1N} - \frac{a_2SP}{1 + b_2S} - \beta SI \qquad (3) \\
\frac{dI}{dt} &= \beta SI - \frac{a_2IP}{1 + b_2I} - d_2I \\
\frac{dP}{dt} &= \frac{e_2a_2SP}{1 + b_2S} + \frac{e_3a_2IP}{1 + b_2I} - d_3P,
\end{aligned}$$

Where

$$a_1 = \frac{A_1K_0}{R_0B_1},\ b_1 = \frac{K_0}{B_1},\ a_2 = \frac{A_2K_0}{R_0B_2},\ b_2 = \frac{K_0}{B_2},\ r = \frac{R_1}{R_0},\ k = \frac{K_1}{K_0},\ d_1 = \frac{D_1}{R_0},\ d_2 = \frac{D_2}{R_0},\ \beta = \frac{K_0\lambda}{R_0},\ d_3 = \frac{D_3}{R_0}$$

System (3) has to be analyzed with the following initial conditions:

$N(0) > 0$, $S(0) > 0$, $I(0) > 0$ and $P(0) > 0$.

3.MODEL ANALYSIS

3.1 LOCAL STABILITY OF EQUILIBRIUM POINTS

The system has seven equilibrium points. The trivial equilibrium point $E_0(0, 0, 0, 0)$ and the axial equilibrium point $E_1(1, 0, 0, 0)$ exist for all parametric values. The axial equilibrium point is $E_2(0, \theta, 0, 0)$, where $\theta = k(r - d_1) > 0$. Predator free equilibrium point is $E_3(N_3, S_3, 0, 0)$ where N_3 is the positive root of the equation: $b_1^2N_3^3 + (2b_1 - b_1^2)N_3^2 + (e_1a_1^2k + 1 - 2b_1 + (r - d_1)a_1b_1k)N_3 + a_1k(r - d_1) - 1 = 0$ (4).

and $S_3 = \frac{(1 + b_1N_3)(1- N_3)}{a_1}$.

The disease-free equilibrium point is $E_4(N_4, S_4, 0, P_4)$ where N_4 is the positive root of the equation $b_1(e_2a_2 - b_2d_3)N_4^2 + (e_2a_2 - b_2d_3)(1 – b_1)N_4 + a_1d_3 - (e_2a_2 - b_2d_3) = 0$. $S_4 = \frac{d_3}{e_2a_2 - b_2d_3}$ and $P_4 = \frac{(1 + b_2S_4)}{a_2} [(r - d_1 - \frac{S_4}{k}) + \frac{e_1a_1N_4}{1 + b_1N_4}]$.

The endemic equilibrium point is $E_5(N_5, S_5, I_5, 0)$, where N_5 is the positive root of the equation $S_5[k(r - d_1) - S_5](1 + b_1N_5) + e_1a_1kN_5(S_5 + I_5) - \beta kS_5I_5(1 + b_1N_5) = 0$, $S_5 = \frac{d_2}{\beta}$ and $I_5 = \frac{\beta(1 - N_5)(1 + b_1N_5) - a_1d_2}{a_1\beta}$.

The interior equilibrium point is given by $E_6(N_6, S_6, I_6, P_6)$ where N_6, S_6, I_6 and P_6 satisfy the following equations:

$$(1 - N_6) - \frac{a_1(S_6 + I_6)}{1 + b_1N_6} = 0$$

$$S_6(r - d_1 - \frac{S_6}{k}) + \frac{e_1a_1N_6(S_6 + I_6)}{1 + b_1N_6} - \frac{a_2S_6P_6}{1 + b_2S_6} - \beta S_6I_6 = 0$$

$$\beta S_6 - \frac{a_2P_6}{1 + b_2I_6} - d_2 = 0$$

$$\frac{e_2a_2S_6}{1 + b_2S_6} + \frac{e_3a_2I_6}{1 + b_2I_6} - d_3 = 0$$

The Jacobian matrix J of the system(3) at any arbitrary point(N, S, I, P) is given by $(J_{ij})_{4\times4}$ where, $J_{11} = 1 - 2N - \frac{a_1(S + I)}{(1 + b_1N)^2}$, $J_{12} = J_{13} = \frac{-a_1N}{1 + b_1N}$, $J_{14} = 0$, $J_{21} = \frac{e_1a_1(S + I)}{(1 + b_1N)^2}$, $J_{22} = r - d_1 - \frac{2S}{k} + \frac{e_1a_1N}{1 + b_1N} - \frac{a_2P}{(1 + b_2S)^2} - \beta I$, $J_{23} = \frac{e_1a_1N}{1 + b_1N} - \beta S$, $J_{24} = \frac{- a_2S}{1 + b_2S}$, $J_{31} = 0$, $J_{32} = \beta I$, $J_{33} = \beta S - \frac{a_2P}{(1 + b_2I)^2} - d_2$,

$J_{34} = \frac{- a_2I}{1 + b_2I}$, $J_{41} = 0$, $J_{42} = \frac{e_2a_2P}{(1 + b_2S)^2}$, $J_{43} = \frac{e_3a_2P}{(1 + b_2I)^2}$, $J_{44} = \frac{e_2a_2S}{1 + b_2S} + \frac{e_3a_2I}{1 + b_2I} - d_3$.

Theorem 1: The trivial equilibrium point E_0 is always unstable. The axial equilibrium point E_1 is locally stable if $R_1 < 1$ where $R_1 = (\frac{1}{d_1 - r})(\frac{e_1a_1}{1 + b_1})$. The predator free equilibrium point E_2 is locally asymptotically stable if $R_{20} < 1$, $R_{21} < 1$ and $R_{22} < 1$. The disease free equilibrium point E_3 is locally asymptotically stable if $(1 + b_1N_3)^2 > a_1b_1S_3$ and $R_{30} < 1$, $R_{31} < 1$. The meaning of R_{20}, R_{21}, R_{22}, R_{30} and R_{31} are given in the proof section.

Proof: Since one of the eigen value associated with the Jacobian matrix computed around E_0 is $1 > 0$, so the equilibrium point E_0 is always unstable.

The characteristic roots of the Jacobian matrix at E_1 are -1, $(r - d_1) + \frac{e_1a_1}{1 + b_1}$, $-d_2$ and $-d_3$. Hence E_1 is stable if $(r - d_1) + \frac{e_1a_1}{1 + b_1} < 0$ which implies $R_1 < 1$ where $R_1 = (\frac{1}{d_1 - r})(\frac{e_1a_1}{1 + b_1})$.

The Jacobian matrix J_2 at predator free equilibrium point E_2 is given by $J_2 = (C_{ij})_{4\times4}$ where

$C_{11} = 1 - a_1\theta$, $C_{12} = C_{13} = C_{14} = 0$, $C_{21} = e_1a_1\theta$, $C_{22} = -\frac{\theta}{k}$, $C_{23} = -\beta\theta$, $C_{24} = \frac{-a_2\theta}{1 + b_2\theta}$, $C_{31} = C_{32} = 0$,

$C_{33} = \beta\theta - d_2$, $C_{34} = C_{41} = C_{42} = C_{43} = 0$, $C_{44} = \frac{e_2a_2\theta}{1 + b_2\theta} - d_3$.

The characteristic roots of the Jacobian matrix J_2 are $1 - a_1\theta$, $-\frac{\theta}{k}$, $\beta\theta - d_2$ and $\frac{e_2a_2\theta}{1 + b_2\theta} - d_3$ where $\theta = k(r - d_1)$. Hence it is clear that E_2 is stable if $1 - a_1\theta < 1$, $\beta\theta - d_2 < 1$ and $\frac{e_2a_2\theta}{1 + b_2\theta} - d_3 < 1$ which implies that if $R_{20} < 1$, $R_{21} < 1$ and $R_{22} < 1$ where $R_{20} = \frac{1}{a_1\theta}$, $R_{21} = \frac{\beta\theta}{d_2}$ and $R_{22} = \frac{e_2a_2\theta}{(1 + b_2\theta)d_3}$.

The Jacobian matrix J_3 at disease free equilibrium point E_3 is given by $J_3 = (D_{ij})_{4\times4}$ where

$D_{11} = -N_3 + \frac{a_1b_1N_3S_3}{(1 + b_1N_3)^2}$, $D_{12} = D_{13} = -\frac{a_1N_3}{1+b_1N_3}$, $D_{14} = 0$, $D_{21} = \frac{e_1a_1S_3}{(1 + b_1N_3)^2}$, $D_{22} = -\frac{S_3}{k}$,

$D_{23} = \frac{e_1a_1N_3}{1+b_1N_3} - \beta S_3$, $D_{24} = -\frac{a_2S_3}{1+b_2S_3}$, $D_{31} = D_{32} = 0$, $D_{33} = \beta S_3 - d_2$, $D_{34} = D_{41} = D_{42} = D_{43} = 0$,

$D_{44} = \frac{e_2a_2S_3}{1+b_2S_3} - d_3$.

The chatacteristic roots of the Jacobian matrix J_3 are $\beta S_3 - d_2$, $\frac{e_2a_2S_3}{1+b_2S_3} - d_3$ and the roots of the equation-

$\lambda^2 + (N_3 - \frac{a_1b_1N_3S_3}{(1+b_1N_3)^2} + \frac{S_3}{k})\lambda + (N_3 - \frac{a_1b_1N_3S_3}{(1+b_1N_3)^2})\frac{S_3}{k} + (\frac{a_1N_3}{1+b_1N_3})(\frac{e_1a_1S_3}{(1+b_1S_3)^2}) = 0.$

Now it is clear that E_3 is stable if, $N_3 - \frac{a_1b_1N_3S_3}{(1 + b_1N_3)^2} > 0$,

i.e. $N_3 > \frac{a_1b_1N_3S_3}{(1 + b_1N_3)^2}$, i.e. $(1+b_1N_3)^2 > a_1b_1S_3$,

and $\beta S_3 - d_2 < 0$, $\frac{e_2a_2S_3}{1+b_2S_3} - d_3 < 0$, i.e. $R_{30} < 1$, $R_{31} < 1$ where $R_{30} = \frac{\beta S_3}{d_2}$ and $R_{31} = \frac{e_2a_2S_3}{d_3(1+b_2S_3)}$.

Theorem 2. The disease free equilibrium point $E_4(N_4, S_4, 0, P_4)$ is asymptotically stable if $\beta S_4 - a_2P_4 - d_2 < 0$ with $H_{11} < 0$, $H_{22} < 0$ and $H_{44} < 0$. The endemic equilibrium point is $E_5(N_5, S_5, I_5, 0)$ is asymptotically stable if $\frac{e_2a_2S_5}{1+b_2S_5} + \frac{e_3a_2I_5}{1+b_2I_5} - d_3 < 0$ with $M_{11} < 0$, $M_{22} < 0$, $M_{33} < 0$ and $M_{23} = 0$.

Proof: The Jacobian matrix J_4 at disease free equilibrium point E_4 is given by $J_4 = (H_{ij})_{4\times4}$ where,

$H_{11} = 1 - 2N_4 - \frac{a_1S_4}{(1 + b_1N_4)^2}$, $H_{12} = H_{13} = -\frac{a_1N_4}{1+b_1N_4}$, $H_{14} = 0$, $H_{21} = \frac{e_1a_1S_4}{(1 + b_1N_4)^2}$,

$H_{22} = r - d_1 - \frac{2S_4}{k} + \frac{e_1a_1N_4}{1+b_1N_4} - \frac{a_2P_4}{(1 + b_2S_4)^2}$, $H_{23} = \frac{e_1a_1N_4}{1+b_1N_4} - \beta S_4$, $H_{24} = -\frac{a_2S_4}{1+b_2S_4}$, $H_{31} = H_{32} = 0$,

$H_{33} = \beta S_4 - a_2P_4 - d_2$, $H_{34} = H_{41} = 0$, $H_{42} = \frac{e_2a_2P_4}{(1 + b_2S_4)^2}$, $H_{43} = e_2a_2P_4$, $H_{44} = \frac{e_2a_2S_4}{1+b_2S_4} - d_3$.

The characteristic roots of the Jacobian matrix J_4 are $\beta S_4 - a_2P_4 - d_2$ and the roots of the equation: $\lambda^3 + \Phi_1\lambda^2 + \Phi_2\lambda + \Phi_3 = 0$, where $\Phi_1 = -(H_{11} + H_{22} + H_{44})$, $\Phi_2 = H_{11}H_{22} + H_{11}H_{44} + H_{22}H_{44} - H_{24}H_{42} - H_{12}H_{21}$ and $\Phi_3 = -(H_{11}H_{22}H_{44} - H_{11}H_{24}H_{42} - H_{12}H_{21}H_{44})$.

Thus, if the conditions stated in the theorem(i.e. if $\beta S_4 - a_2P_4 - d_2 < 0$ with $H_{11} < 0$, $H_{22} < 0$ and $H_{44} < 0$) holds, then all the Routh-Hurwitz criteria (i) all Φ_1, Φ_2 , $\Phi_3 > 0$ and (ii) $\Phi_1\Phi_2 - \Phi_3 > 0$ are satisfied, and the disease free equilibrium point $E_4(N_4, S_4, 0, P_4)$ is asymptotically stable.

Again, the Jacobian matrix J_5 at endemic equilibrium point $E_5(N_5, S_5, I_5, 0)$ is given by $J_5 = (M_{ij})_{4\times4}$ where,

$M_{11} = 1 - 2N_5 - \frac{a_1(S_5 + I_5)}{(1 + b_1N_5)^2}$, $M_{12} = M_{13} = -\frac{a_1N_5}{1+b_1N_5}$, $M_{14} = 0$, $M_{21} = \frac{e_1a_1(S_5 + I_5)}{(1 + b_1N_5)^2}$,

$M_{22} = r - d_1 - \frac{2S_5}{k} + \frac{e_1a_1N_5}{1+b_1N_5} - \beta I_5$, $M_{23} = \frac{e_1a_1N_5}{1+b_1N_5} - \beta S_5$, $M_{24} = -\frac{a_2S_5}{1+b_2S_5}$, $M_{31} = 0$, $M_{32} = \beta I_5$,

$M_{33} = \beta S_5 - d_2$, $M_{34} = -\frac{a_2I_5}{1+b_2I_5}$, $M_{41} = M_{42} = M_{43} = 0$, $M_{44} = \frac{e_2a_2S_5}{1+b_2S_5} + \frac{e_3a_2I_5}{1+b_2I_5} - d_3$.

The characteristic roots of the Jacobian matrix J_5 are $\frac{e_2a_2S_5}{1+b_2S_5} + \frac{e_3a_2I_5}{1+b_2I_5} - d_3$ and the roots of the equation:

$\lambda^3 + \Psi_1\lambda^2 + \Psi_2\lambda + \Psi_3 = 0$, where $\Psi_1 = -(M_{11} + M_{22} + M_{33})$, $\Psi_2 = M_{11}M_{22} + M_{11}M_{33} + M_{22}M_{33} - M_{12}M_{21} - M_{23}M_{32}$ and $\Psi_3 = -(M_{11}M_{22}M_{33} + M_{13}M_{21}M_{32} - M_{11}M_{23}M_{32} - M_{12}M_{21}M_{33})$.

Now, all the Routh-Hurwitz criteria (i) all Ψ_1, Ψ_2, $\Psi_3 > 0$ and (ii) $\Psi_1\Psi_2 - \Psi_3 > 0$ will be satisfied if the conditions stated in the theorem(i.e. $\frac{e_2a_2S_5}{1+b_2S_5} + \frac{e_3a_2I_5}{1+b_2I_5} - d_3 < 0$ with $M_{11} < 0$, $M_{22} < 0$, $M_{33} < 0$ and $M_{23} = 0$) holds. Then the endemic equilibrium point is $E_5(N_5, S_5, I_5, 0)$ is asymptotically stable.

3.2 Stability of the interior equilibrium point $E_6(N_6, S_6, I_6, P_6)$:

The Jacobian matrix at the interior equilibrium point E_6 is given by $J_6 = (V_{ij})_{4\times4}$ where,

$V_{11} = \frac{b_1a_1N_6(S_6 + I_6)}{(1 + b_1N_6)^2} - N_6$, $V_{12} = V_{13} = -\frac{a_1N_6}{1+b_1N_6}$, $V_{14} = 0$, $V_{21} = \frac{e_1a_1(S_6 + I_6)}{(1 + b_1N_6)^2}$,

$V_{22} = -\frac{S_6}{k} + \frac{e_1a_1N_6I_6}{S_6(1+b_1N_6)} + \frac{a_2b_2P_6S_6}{(1 + b_2S_6)^2}$, $V_{23} = \frac{e_1a_1N_6}{1+b_1N_6} - \beta S_6$, $V_{24} = -\frac{a_2S_6}{1+b_2S_6}$, $V_{31} = 0$, $V_{32} = \beta I_6$

$V_{33} = \frac{a_2b_2P_6I_6}{(1 + b_2I_6)^2}$, $V_{34} = -\frac{a_2I_6}{1+b_2I_6}$, $V_{41} = 0$, $V_{42} = \frac{e_2a_2P_6}{(1 + b_2S_6)^2}$, $V_{43} = \frac{e_3a_2P_6}{(1 + b_2I_6)^2}$, $V_{44} = 0$.

Now the characteristic equation of the matrix $J_6 = (V_{ij})_{4\times4}$ is given by

$\lambda + \sigma_1\lambda^3 + \sigma_2\lambda^2 + \sigma_3\lambda + \sigma_4 = 0$, where
$\sigma_1 = -(V_{11} + V_{22} + V_{33})$,
$\sigma_2 = V_{11}V_{22} + V_{11}V_{33} + V_{22}V_{33} - V_{12}V_{21} - V_{23}V_{32} - V_{24}V_{42} - V_{34}V_{43}$,
$\sigma_3 = V_{11}V_{34}V_{43} + V_{11}V_{23}V_{32} + V_{11}V_{24}V_{42} + V_{22}V_{34}V_{43} + V_{33}V_{24}V_{42} + V_{33}V_{12}V_{21} - V_{11}V_{22}V_{33} - V_{23}V_{34}V_{42} - V_{24}V_{32}V_{43} - V_{13}V_{21}V_{32}$,
$\sigma_4 = V_{11}V_{23}V_{34}V_{42} + V_{11}V_{24}V_{32}V_{43} + V_{12}V_{21}V_{34}V_{43} - V_{11}V_{22}V_{34}V_{43} - V_{11}V_{33}V_{24}V_{42} - V_{13}V_{21}V_{34}V_{42}$,

Therefore, the interior equilibrium point E_6 will be asymptotically stable if σ_1 , σ_2 , σ_3 , σ_4 satisfy all the Routh-Hurwitz conditions (i) all σ_1 , σ_2 , σ_3 , $\sigma_4 > 0$, (ii) $\sigma_1\sigma_2 > \sigma_3$ and (iii) $\sigma_1\sigma_2\sigma_3 > \sigma_3^2 + \sigma_1^2\sigma_4$.

Now we shall find out the conditions for which the interior equilibrium point E_6 enters into Hopf bifurcation as β varies over R. Routh-Hurewitz Criterion and Hopf bifurcation: Let Ψ: $(0, \infty) \rightarrow R$ be the following continuously differentiable function of β:

$$\Psi(\beta) := \sigma_1(\beta)\sigma_2(\beta)\sigma_3(\beta) - \sigma_3^2(\beta) - \sigma_1^2(\beta)\sigma_4(\beta)$$

The assumptions for Hopf bifurcation to occur are the usual ones and these require that the spectrum

$\sigma(\beta) = \{\lambda : D(\lambda) = 0\}$ of the characteristic equation such is that
(a)There exists $\beta^* \in (0, \infty)$, at which a pair of complex eigen values $\lambda(\beta^*)$, $\bar{\lambda}(\beta^*) \in \sigma(\beta)$ are such that

$$Re\ \lambda(\beta^*) = 0,\ Im\ \lambda(\beta^*) = \omega_0 > 0,$$

and the transversality condition

$$\left[\frac{dRe\lambda(\beta)}{d\beta}\right]_{at\ (\beta^*)} \neq 0\ ;$$

(b) all other elements of $\sigma(\beta)$ have negatives real parts.

Now we present a theorem for Hopf bifurcation.

Theorem 3. The Hopf bifurcation of the interior equilibrium point E_6 occurs at $\beta = \beta^* \in (0, \infty)$ if and only if

$$\Psi(\beta^*) = 0,\ \left[\frac{dRe\lambda(\beta)}{d\beta}\right]_{at\ (\beta^*)} \neq 0$$

and all other eigen values are of negative real parts, where $\lambda(\beta)$ is purely imaginary at $\beta = \beta^*$.

Proof: By the condition $\Psi(\beta^*) = 0$, the characteristic equation can be written as

$$(\lambda^2 + \frac{\sigma_3}{\sigma_1})(\lambda^2 + \sigma_1\lambda + \frac{\sigma_1\sigma_4}{\sigma_3}) = 0$$

If it has four roots, say $\lambda_i (i = 1, 2, 3, 4)$ with the pair of purely imaginary roots at $\beta = \beta^*$ as $\lambda_1 = \bar{\lambda}_2$, then we have

$$\lambda_3 + \lambda_4 = -\sigma_1$$
$$\omega_0^2 + \lambda_3\lambda_4 = \sigma_2$$
$$\omega_0^2(\lambda_3 + \lambda_4) = -\sigma_3$$
$$\omega_0^2\lambda_3\lambda_4 = \sigma_4$$

where $\omega_0 = Im\ \lambda_1(\beta^*)$. By above $\omega_0 = \frac{\sigma_3}{\sigma_1}$. Now, if λ_3 and λ_4 are complex conjugate, then from(), it follows that $2Re\lambda_3 = -\sigma_1$; if they are real roots, then by () and () $\lambda_3 < 0$ and $\lambda_4 < 0$. To complete the discussion, it remains to verify the transversality condition.

As $\Psi(\beta^*)$ is a continuous function of all its roots, so there exists an open interval $\beta \in (\beta^* - \varepsilon, \beta^* + \varepsilon)$ where λ_1 and λ_2 are complex conjugate for β. Suppose, their general forms in this neibourhood are

$$\lambda_1(\beta) = \mu(\beta) + i\upsilon(\beta),$$
$$\lambda_2(\beta) = \mu(\beta) - i\upsilon(\beta),$$

Now, we shall verify the transversality condition

$$[\frac{dRe(\lambda_j(\beta))}{d\beta}]_{at\ (\beta = \beta^*)} \neq 0\ ,\ j = 1, 2.$$

Substituting $\lambda_j(\beta) = \mu(\beta) \pm i\upsilon(\beta)$, into () and calculating the derivatives, we have

$$K(\beta)\mu'(\beta) - L(\beta)\upsilon'(\beta) + M(\beta) = 0$$
$$K(\beta)\mu'(\beta) + L(\beta)\upsilon'(\beta) + N(\beta) = 0$$

where

$$K(\beta) = 4\mu^3 - 12\mu\upsilon^2 + 3\sigma_1(\mu^2 - \upsilon^2) + 2\sigma_2\mu + \sigma_3$$
$$L(\beta) = 12\mu^2\upsilon + 6\sigma_1\mu\upsilon - 4\mu^3 + 2\sigma_2\mu$$
$$M(\beta) = \sigma_1\mu^3 - 3\sigma_1'\mu\upsilon^2 + \sigma_2'(\mu^2 - \upsilon^2) + \sigma_3'\mu$$
$$N(\beta) = 3\sigma_1'\mu^2\upsilon - \sigma_1'\upsilon + 2\sigma_2'\mu\upsilon + \sigma_3'\mu$$

Solving for $\mu'(\beta^*)$ we have

$$[\frac{dRe(\lambda_j(\beta))}{d\beta}]_{at\ (\beta = \beta^*)} = \mu'(\beta)_{\beta = \beta^*} = -\frac{L(\beta^*)\,N(\beta^*) + K(\beta^*)\,M(\beta^*)}{K^2(\beta^*) + L^2(\beta^*)} \neq 0$$

since $L(\beta^*)\,N(\beta^*) + K(\beta^*)\,M(\beta^*) \neq 0$. Thus the transversality conditions hold and hence Hopf bifurcation occurs at $\beta = \beta^*$.

Hence the theorem.

4. NUMERICAL RESULTS AND DISCUSSION

In this study we will perform extensive numerical experiments to observe the global dynamical behavior of the model system. In this study the infection rate β in the intermediate predator population is new modification of most of the earlier studies. We have taken a set of hypothetical

parameter values $a_1 = 4.9$, $a_2 = 0.1$, $b_1 = 2.9$, $b_2 = 2.0$, $d_1 = 0.4$, $d_2 = 0.41$, $d_3 = 0.01$, $e_1 = 0.98$, $e_2 = 0.95$, $e_3 = 0.6$, $r = 0.01$, $k = 0.5$ and $\beta = 8.2$. Throughout this numerical experiment we have fixed the above set of hypothetical parameter values and most of the parameter values are taken from Hastings and Powells[17] . We first observe the dynamics of our proposed system for variation of the force of infection in the intermediate predator. Finally we observe the role of alternative food.

The force infection in the intermediate predator of a tri-trophic food chain model is vital issue. We observe chaotic dynamics in our proposed system for the set of parameter values (Figure (1)). We draw a phase portrait of chaotic dynamics and we observe that it will take a shape of tea-cup attractor (Figure (2)). Now we will analyze the trajectory of the tea-cup attractor. The dynamics within the attractor are given roughly as follows. Starting in the handle of the teacup, the system moves to the wide part of the teacup and then spirals along the teacup to the narrow end, entering the handle again. In term of species behavior, the top-predator Z crashes, allowing wide swings in the population levels of X and $Y_1 + Y_2$. As Z increases in numbers, the swings in X and $Y_1 + Y_2$ become damped until Z causes the levels of $Y_1 + Y_2$ to crash. This leads to a crash in Z and outbreak in X, starting the process again. The sequence of events, in terms of species numbers, always follows the same general pattern. What is unpredictable is the timing. One way to express this is that the time between crashes of species Z varies in an erratic fashion. Also, the number of peaks in species Y between major crashes varies and the population size at the peaks varies. The sensitive dependence of future dynamics on the current state, the signature of chaos, is apparent

from the fact that all the trajectories in the handle of the teacup are very close together.Now we shall try to find whether the strange attractor shown in the Figure(1) is chaotic or not. For this, we computed Lyapunov exponents, which are sophisticated mathematical tools that help to analyze the dynamical behavior of autonomous systems in order to conclude the actual nature of it. Computing all Lyapunov exponents (Figure(10)) corresponding to the strange attractor depicted in Figure(1), we observe that the largest Lyapunov exponent is positive(0.1635) and other three Lyapunov exponents are, one is zero and other two is negative(-0.46075 and -1.4933). Thus, we may conclude that the strange attractor is chaotic attractor.

If we increases the values of β from 8.2 to 9.6 we observe quasi-periodic dynamics (Figure(3)). If we further increase the force of infection β we observe that quasi-periodic dynamics reduces to limit cycle oscillations (Figure(4)). We finally observe that system enters into stable focus from limit cycle oscillations (Figure(5)). To get clear dynamical behavior of our system we draw a bifurcation diagram (Figure(6)) and from this diagram we observe that system enters into stable focus from chaotic dynamics for increasing the force of infection. So, it is clear that the system enters into stable focus from chaos for increasing the force of infection β. We observe that the system enters into quasi-periodic from chaos; limit cycle oscillations from quasi-periodic dynamics and finally stable focus from limit cycle oscillations for increasing the force of infection. So it is clear that when the force of infection is low level the system shows chaotic dynamics and when the force of infection is higher level chaos reduces to stable focus. Existing mathematical models suggest that disease introduction into the predator population tends to destabilize established predator-prey communities. This has been observed for microparasites with both direct[2, 29, 10] and indirect life cycles [11, 12]. Macro-parasite models generally have a tendency to unstable dynamics, because they consider the parasite burden in the host in an additional equation [1, 15, 11]. Here we show that chaotic dynamics reduces to stable focus for increasing the force of infection.

The effect of disease introduction in the intermediate predator of a tri-trophic food chain model can be quite the opposite, namely to stabilize chaotic predator-prey dynamics. We demonstrate this in a tri-trophic food chain model with disease in the intermediate predator. Our model thus brings together the two fields of ecology and epidemiology, as it extends classical epidemiological approaches by demography and ecological interactions. From above discussion we observe that when disease is low level, the system shows chaotic dynamics. Now we want to observe the role of alternative food on the chaotic dynamics. For this we will vary the carrying capacity k of the intermediate predator. From Figure(7) it is observe that the system shows chaotic dynamics for k = 0.5. Now if we decrease the value of k from 0.5 to 0.2 system enters into quasi-periodic position (Figure(4)). If we further decrease the value of k we observe that quasi-period is disappeared and the infected intermediate predator goes to extinction (Figure(8)). We also draw a bifurcation diagram (Figure(9)) to observe clear dynamical behavior for variation of k. From this it clear that when the alternative food decreases chaos disappear and the infected predator wash out from the system but when alternative food increases the chaos remain same in our system. So the alternative food can be used as the controlling agent in our system.

Now we describe what is the new insight of this present study. We observe that in low infection level the system shows chaotic dynamics and in high infection level chaos becomes stable focus. We also observe several nonlinear phenomena for increasing the force of infection of the intermediate predator such as chaos to quasi-period; quasi-period to limit cycle; limit cycle to stable focus. We also observe the role of alternative food on chaotic dynamics. It is observed that when the alternative food is in low level chaos disappears and the infected intermediate predator has been washed out from the system but in higher level of alternative food chaos remain same in the system. We have shown, to our knowledge that the chaotic dynamic observed in system can be stabilized by an infectious disease spreading within the intermediate predator population. This challenges the current view of destabilizing disease impacts[15, 2, 11, 18, 29, 30, 12, 10]. Our findings are also of relevance for biological control, as infectious diseases can be used as control agents of undesirable species such as biological invaders. This study interestingly suggests that parasites can have regulating effects on more than one trophic level and be utilized for management purposes in multi-species systems. The introduction of disease can not only control or eradicate the predator, but also allow the prey species to recover. For example, pathogens could potentially be used to control mammal pest species such as feral domestic cats (predators) on oceanic islands that have devastating impacts on native prey species(e.g.seabirds)[24, 27, 28]. Alternative food is also a controlling agent in our model system. This study provides insightful ecological and epidemiological reproduction numbers for understanding how parasites structure the community composition. We also present a comparative study with most of the earlier studies. Hadeler and Freedman[18] considered a predator-prey model where both species are subjected to parasitism, is developed and analyzed. They also assume that the predators could get infection by eating prey and the prey could obtained the disease from parasites spread into the environment by the predators, they obtained a threshold condition above which an endemic equilibrium or an endemic periodic solution could arise in the case where there is coexistence of the predator with the uninfected prey. Furthermore, they also showed that in the case where the predator can not survive only on the prey in a disease-free environment, the parasitization could lead to persistence of the predator since the predator could only survive on the predator if some of the prey were more easily captured due to being diseased, provided a certain threshold for disease transmission is surpassed. In the work of Hsieh and Hsiao[20], the predator can be infected upon contact with or being in the those vicinity of an infected prey during the process of predation, but the predator cannot infect each other. They observed that the infected predator plays a minor and indirect role

in the spread of disease mainly due to the assumption that the infected predators are unable to infect other members of the population. Recently Hilker and Schmitz [21](2008) considered the invasion of a resident predator-prey system by an infectious disease with frequency dependent transmission spreading within the predator population. They had derived biologically plausible and insightful quantities (demographic and epizootiological reproduction numbers) that allow them to completely determine community composition. Their findings contradict predictions from previous models suggesting a destabilizing effect of parasites and they show that predator infection counteracts the paradox of enrichment. In the present study consider a tri-trophic food chain model with disease in the intermediate predator only and there is an alternative food source for the intermediate predator. We observe that chaotic dynamics becomes stable focus for increasing the force of infection. We also observe the role of alternative food on chaotic dynamics. It is observed that in low level of the alternative food chaos disappears and the infected intermediate predator is washed out from the system but in higher value of the alternative food chaos remains same in our system. Recently Mandal et al.[26](2009) consider a predator-prey system with disease in the prey population. They observe the dynamics of such a system under the influence of severe as well as unnoticeable parasite attack and also alternative food sources for predator population. They assume the predator population will prefer only infected prey for their diet as those are more vulnerable. Their results indicate that in the case of severe parasitic attack the predator population will prefer the alternative food source and not the infected one. But the strategy is reverse in the case of unnoticeable parasite attack. In the present study we consider a tri-trophic food chain model with disease circulating in intermediate predator population and there is a alternative food source of intermediate predator population. We observe that when infection is low level system shows chaotic dynamics and in higher infection level chaos becomes stable focus. We also observe the role of alternative food on chaotic dynamics. When alternative food increases the chaos remain same in the system but when alternative food decreases chaos disappears and infected intermediate predator is washed out from the system.

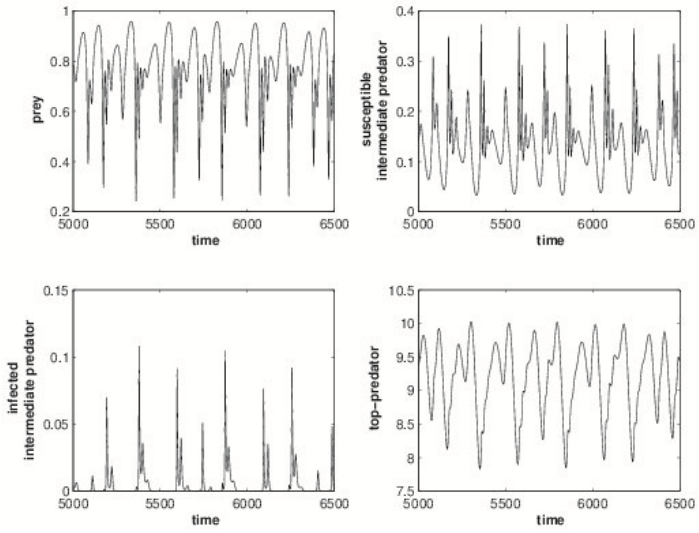

Figure-1: The time series solution of the model system (3) for $a_1 = 4.9$, $a_2 = 0.1$, $b_2 = 2.0$, $d_1 = 0.4$, $d_2 = 0.41$, $d_3 = 0.01$, $e_1 = 0.98$, $e_2 = 0.95$, $e_3 = 0.6$, $b_1 = 2.9$, $r = 0.01$, $k = 0.5$ and $\beta = 8.2$.

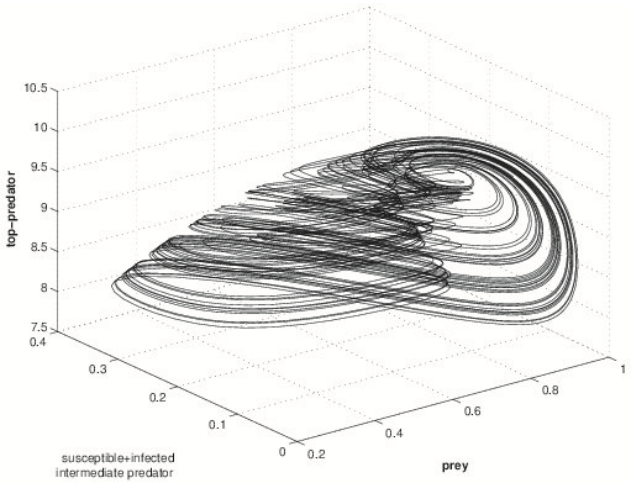

Figure-2: The phase plane of the model system (3) for $a_1 = 4.9$, $a_2 = 0.1$, $b_2 = 2.0$, $d_1 = 0.4$, $d_2 = 0.41$, $d_3 = 0.01$, $e_1 = 0.98$, $e_2 = 0.95$, $e_3 = 0.6$, $b_1 = 2.9$, $r = 0.01$, $k = 0.5$ and $\beta = 8.2$.

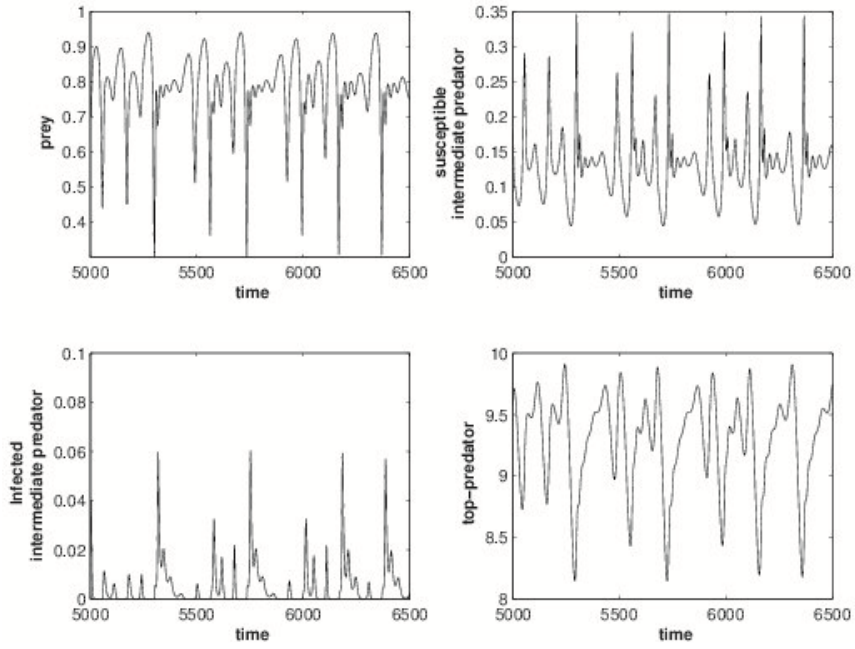

Figure-3: The time series quasi-periodic dynamics of the system (3) for $\beta = 9.6$ and other parameter values given in the Figure-1.

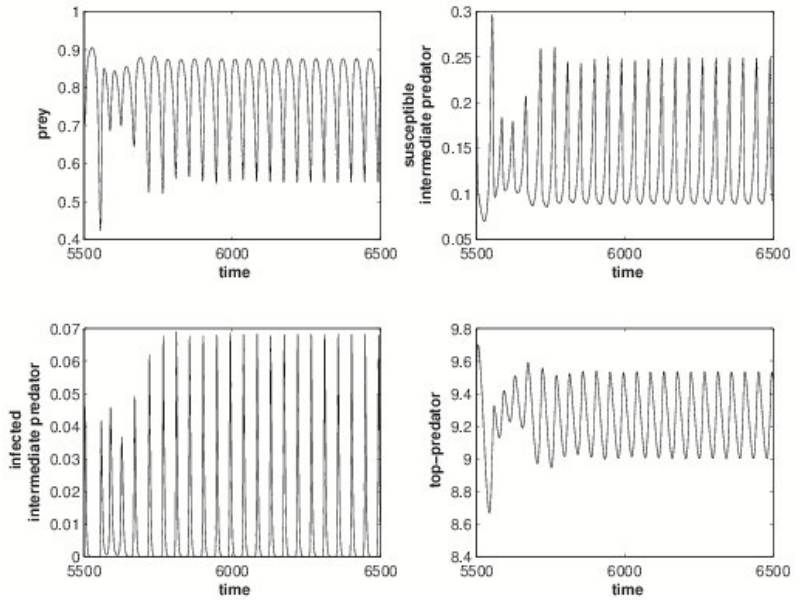

Figure-4: The time series limit cycle oscillation of the system (3) for β = 10.2 and other parameter values given in the Figure-1.

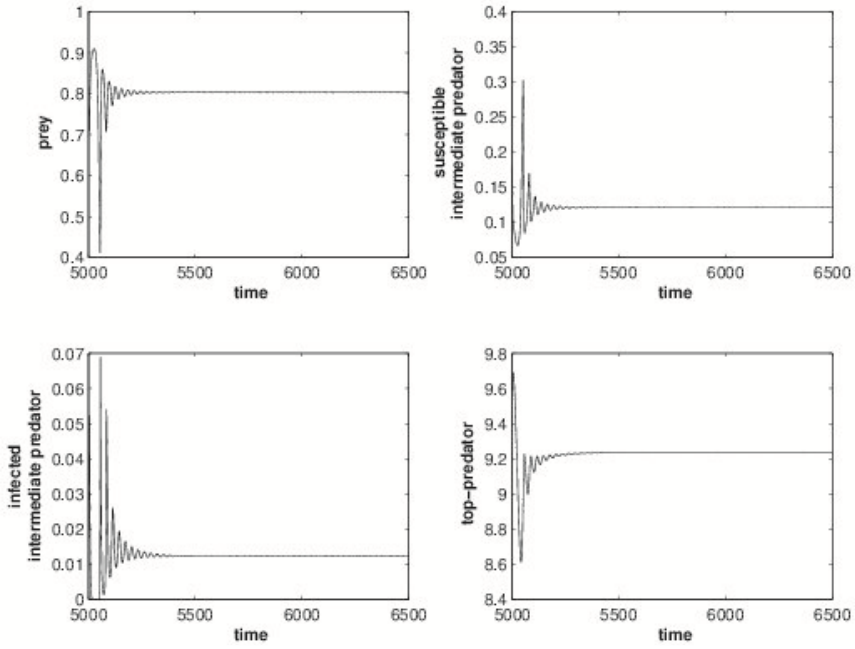

Figure-5: The time series stable solution of the system (3) for β = 10.5 and other parameter values given in the Figure-1

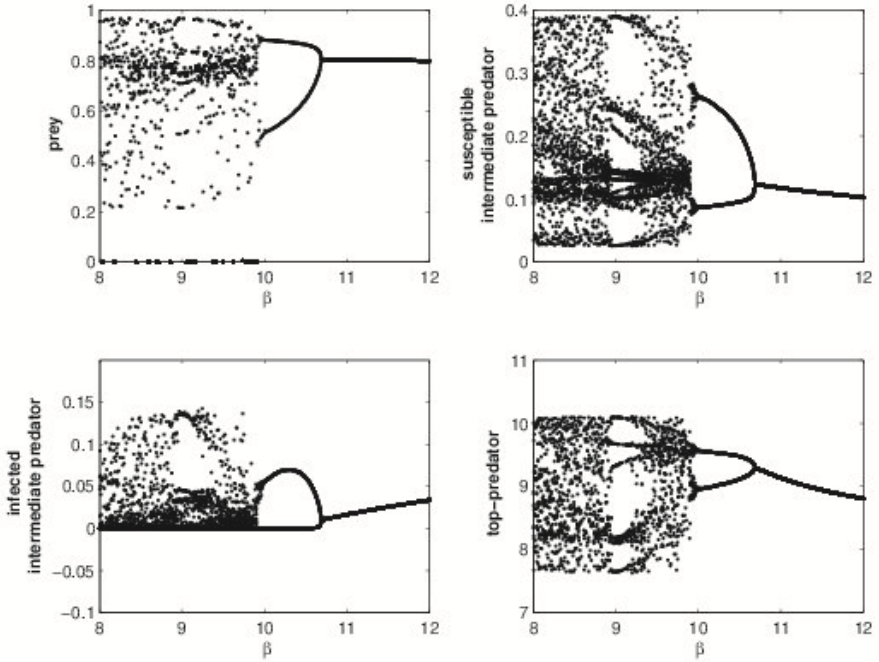

Figure-6: Figure shows the bifurcation diagram for β ϵ [8.0, 11.0] and other parameter values given in Figure-1.

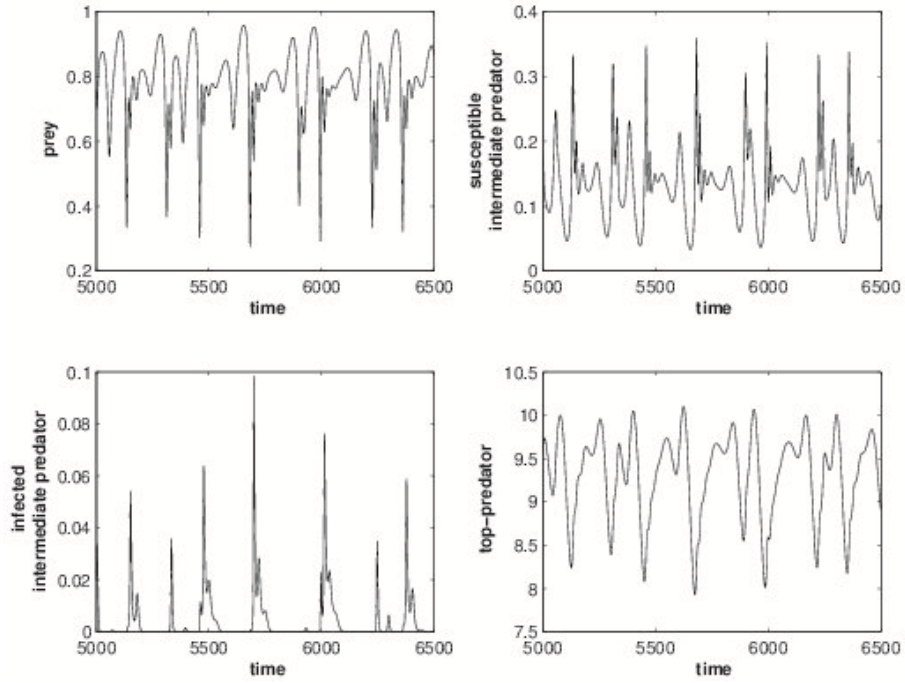

Figure-7: The time series chaotic dynamics of the system (3) for k = 0.2, r = 0.01, β = 9.0 and other parameter values given in the Figure-1.

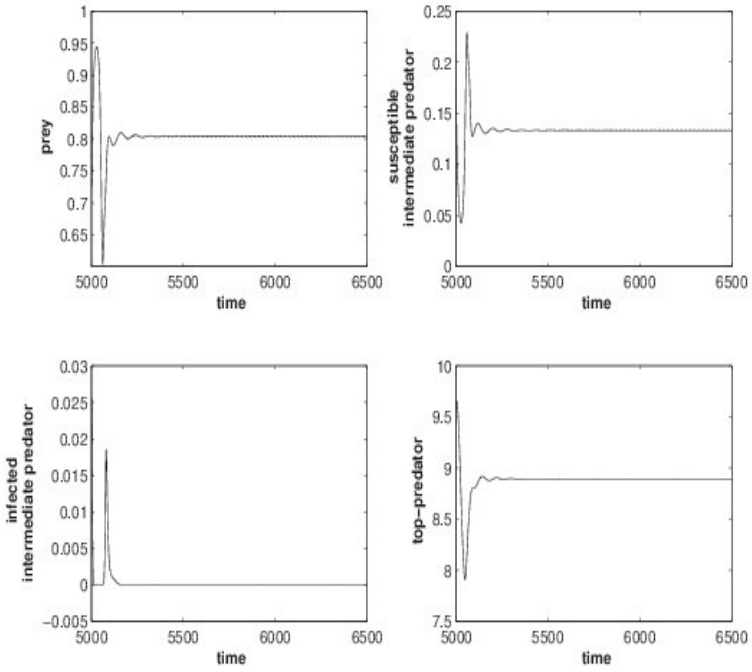

Figure-8: The time series chaotic dynamics of the system (3) for k = 0.02, r = 0.01, β = 9.0 and other parameter values given in the Figure-4.

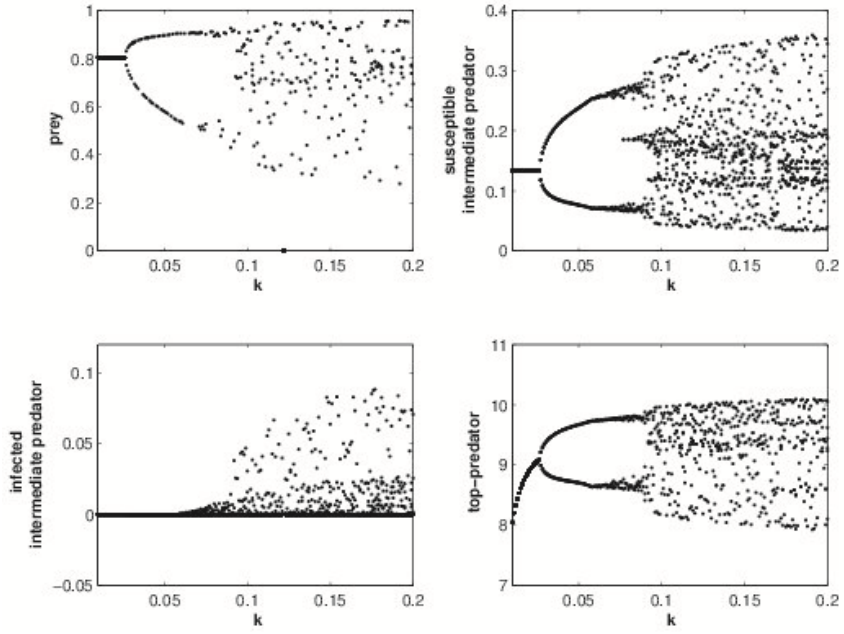

Figure-9: The bifurcation diagram of the system (3) for k ϵ [0.02, 0.2], r = 0.01 and other parameter values given in Figure-1.

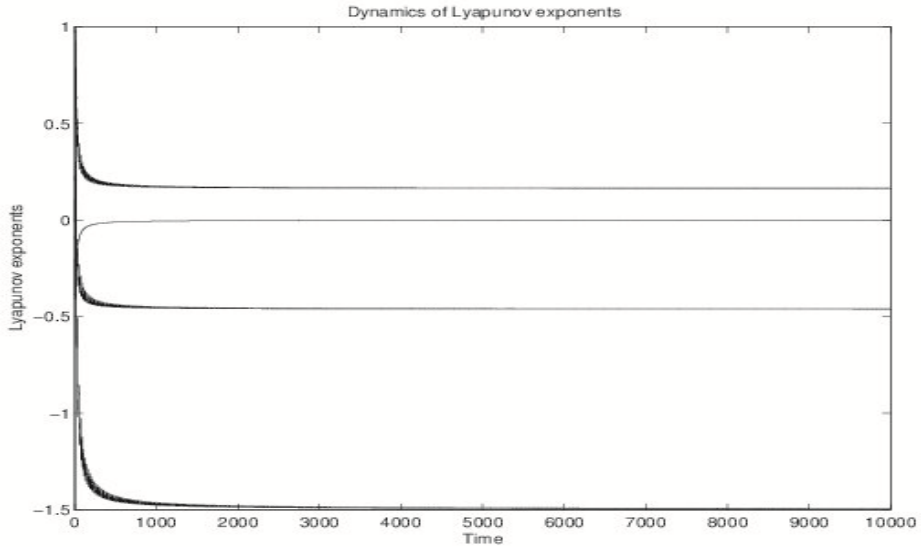

Figure-10: The spectrum of Lyapunov exponent for the system around the strange attractor shown in Figure-1.

CONCLUSION

Infectious diseases can have regulating effects not only on their host population, but also on other species their host interacts with [2] (Anderson and May, 1986). In this study we consider a tri-trophic food chain model with disease circulating in the intermediate predator and there is an alternative food source for the intermediate predator. We have observed the local stability of our model systems around the biological feasible equilibria. We have introduced the ecological and the disease basic reproduction numbers and analyzed the community structure of our model system. To observe the global behavior of our model system we perform extensive numerical simulations. We have observed the chaotic dynamics when the infection in the intermediate predator is low and also observed period-doubling and limit cycle when the force of infection increases. Finally we have observed that chaos becomes stable focus when the infection is high. Nonlinear interactions between the predators and the prey are well known to generate endogenous oscillations. We have shown to our knowledge that the chaotic fluctuations can be stabilized by an infectious disease spreading in the intermediate predator of a tri-trophic food chain model. This is challenges the current view of destabilizing disease impacts[22, 2] (Dobson and Keymer, 1985; Anderson and May, 1986). We have also observed the role of alternative food on the chaotic dynamics and it is observed that when the alternative food increases, the chaos remains same in system but when the alternative food decreases chaos becomes stable focus and the infected intermediate predator is washed out from the system.

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