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Original Research Article

The study of chaotic dynamics of an eco-epidemiological predator-prey model with alternative food

Prodip Roy[1], Abhishek Sarkar[2], Kulbhushan Agnihotri[2], Krishna Pada Das[1,*]

[1] *Department of Mathematics, Mahadevananda Mahavidyalaya, Monirampore, Barrackpore, West Bengal 700120, India*

[2] *Department of Applied Science and Humanities, Shaheed Bhagat Singh State University, Ferozepur, Punjab 152001, India*

*** Corresponding author:** Krishna Pada Das, krishnaisi@yahoo.co.in

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ABSTRACT: Parasites can alter the quality and quantity of participants. On the other hand, the spread of diseases between individuals or species is an important research topic. Here we consider a tritrophic food model in which bacteria spread among the animal's environment where other nutrients are present. We analyze the local stability of the model around the efficiency of the equation. We also report significant numbers of offspring in terms of ecology and disease and use these numbers to analyze community structure of the sample. We started from this situation as a model. Our numerical results show that at low infection level the body causes conflict, but at high infection level conflict prioritizes safety. Our findings therefore challenge previous models' predictions that the parasite has a negative impact. We also looked at the impact of other foods on chaotic dynamics. When other nutrients increase, stress does not change, but when other nutrients decrease, chaos disappears and the disease among animals is eliminated from the body.

KEYWORDS: disease in intermediate predator; chaos; period-doubling; limit cycle; stable focuses; alternative food

1. Introduction

The interaction between animals and animals is one of the most important interactions in ecology and epidemiology due to its importance in our real life. Ecological epidemiology is a branch of mathematical biology that considers both ecological and epidemiological problems. Anderson and May[1] were the first to combine the above two fields and create a model of animal diseases in which animals are infected. In the following period, many scientists prepared and examined different animal-prey models in the presence of disease. Knowledge in the field of ecological epidemiology has increased tremendously in the last two years, of which we have only mentioned a few[2–7]. Early studies[8–10] focused solely on infection in victims.

To our knowledge, Anderson and May[11], Hadeler and Freedman[5], Hochberg[12], Venturino[13] and[14], Chattopadhyay and Arino[2], Han et al.[15], Xiao WS, Chen WS, Hethcote et al.[9] Greenhalgh and Haqu[6] and Haque and Venturino[16]. Most of these studies involve animal models in which the animal is infected with the disease (except for Venturino[7], Haque and Venturino[16]). However, when there is a problem in animal control, it is very important to investigate animal diseases when killing animals. As we know, infectious diseases remain almost silent on this issue. However, to our knowledge, there have been few efforts to understand the dynamics of tritrophic food webs and

intermediate animal diseases. Here we consider a three-trophic trophic pattern endemic to the medium-sized animal population and also consider other food sources for medium-sized animals.

In this study, we focus on the power transfer of chaotic dynamics. Much of the early work was based on the search for stability and physical risk[17] and analyzed from the linear equation to nonlinear equation involving complex situations often needed by ecologists[18]. But now things are changing. The terms chaos, strange attractor, and fractal are familiar to most, if not all, ecologists[19]. Although stress is often predicted by mathematical models, the evidence for its existence in the natural world is mixed and uncertain[20]. Chaotic dynamics exist in tritrophic food webs and are of interest to model and experimental ecologists. Hastings and Powell (HP)[18] constructed a population crisis with a type II response function in a simple threetrophic food model. McCann and Yodzis[21] modified Hastings and Powell's model[18] and found that the system produces pressure for a suitable connection between critical parameters. Impacts can occur not only in ecosystems but also in infectious diseases. General observations suggest that measles is a good candidate for infection, but conclusive evidence is still lacking[22–24]. Grenfell et al.[25] investigated the impact of local chaotic dynamics on global persistence in the epidemiological model. Although the study of chaos in ecoepidemiological systems is new, the literature on chaos in ecosystems is quite rich. Recently, Chatterjee et al.[26] found that disease transmission and predation rates are two main factors controlling the chaotic dynamics of ecoepidemiological systems. In our model, we examine the conflict between changes in the epidemic.

Another important factor in this study is the alternative food for mesopredators. Another food source for mesopredators is an important factor in the interaction between species, and it may be useful to include this factor in eco-epidemiological studies. Predators generally do not feed on one type of prey, but turn to other food sources when the density of their preferred prey is low[5]. It is well known that foraging theory states that when the number of animals falls below the threshold due to disease of the preferred animals or other reasons, the top animals will switch to other food sources or include other foods in their diet. (in a fine-grained medium) or by switching to another food source (in a coarse-grained medium). Charnov[27] published a well-known nutritional formula that involves replacing taken-for-granted food with a diet mixed only with other foods. Fryxell and Lundberg[28] demonstrated from a numerical simulation study of one predator/two predators that predators will only switch to lower predators when they reduce prey availability, providing greater benefits to livestock. They also showed that this change would also reduce the stress on animal earnings when the number of animals is smaller. The availability of other foods for livestock can lead to a decrease in livestock balance, referred to as apparent competition[29].

This study tries to show the chaos in the body and finally we look at the role of other foods and the impact of foods on chaotic dynamics. Another research method is the community structure of our sample. To this end, we introduce the ecological concept and the concept of disease numbers of reproduction. Parity can be defined as the number of offspring produced by an individual during its lifetime, or the number of offspring expected from the second disease that an infected individual produces in the affected population during the period of transmission. Although the concept of reproduction in population was first developed at the beginning of the 20th century[30], it has become a standard tool in epidemiology since the work of Anderson and May[31] and Diekmann et al.[32]. We will use the number published as an important tool for determining the existence or demise of a species. This will allow us to separate the community composition of animals, predators, and diseases. The concept of threshold in number of births has been used in previous eco-epidemiological studies[5,9,10,15].

The paper is organized as follows. In section 2, we outline the mathematical model with some basic assumptions. 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[18] with pairwise interactions between three species, namely, X, Y, and Z, which incorporates a Holling type II functional response in both consumers species, namely Y and Z are as follows:

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

where X is the number of animals that are low on food, Y is the number of animals exposed to Here T is time. The constant R0 is the growth rate of the medium and the constant K0 is the carrying capacity of species X. The constants e1 and e2 are the transfer rate of the animal to type Y and Z, respectively; D1 and D2 are Y and Z type constants respectively. For i = 1 and 2, the constants Ai and Bi are the maximum predation rate and halfsaturation constant for Y and Z, respectively. Hastings and Powell[18] showed that the interaction between predators and prey on three simple food types is chaotic in one region of the parameter space.

This disease is assumed to spread horizontally. We also assume that parasites only kill predator populations. The disease is spread by predators at a rate λ according to the law of mass order.
According to the above assumptions, Equation (1) can be written as the following set of non-differentiable differential equations:

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

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 scaling:

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

Then Equation (2) takes the form (after some simplification),

$$\begin{aligned} \frac{dN}{dt} &= N(1-N)-\frac{a_1N(S+I)}{1+b_1N} \\ \frac{dS}{dt} &= S\left(r-d_1-\frac{S}{k}\right)+\frac{e_1a_1N(S+I)}{1+b_1N}-\frac{a_2SP}{1+b_2S}-\beta SI \end{aligned} \tag{3}$$

$$\frac{dI}{dt} = \beta SI - \frac{a_2IP}{1 + b_2I} - d_2I$$

$$\frac{dP}{dt} = \frac{e_2a_2SP}{1 + b_2S} + \frac{e_3a_2IP}{1 + b_2I} - d_3P$$

where

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

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

$$N(0) > 0,\ S(0) > 0,\ I(0) > 0 \text{ and } P(0) > 0.$$

3. Model analysis

3.1. Positivity and boundedness of model system

The positivity and boundedness of the system in theoretical ecology indicate the system's biologically well-behaved character. The provided results in this part will guarantee the positivity and boundedness of the suggested model system's solutions.

Theorem 1. *All the solutions of the Equation (3) are positive.*

Proof. From the first equation of Equation (3) we can write,

$$\frac{dN}{N} = [(1 - N) - \frac{a1(S + I)}{1 + b1N}]\, dt,$$

which implies $\frac{dN}{N} = \alpha_1(N, S)\, dt$, where $\alpha_1(N, S) = [(1 - N) - \frac{a1(S + I)}{1 + b1N}]$.

Now integrating above differential equation in the region [0, t] we have,

$$N(t) = N(0)e^{\int_0^t \alpha_1(N,S)dt} > 0,\ \forall t.$$

from the second equation of Equation (3) we can write,

$$\frac{dS}{S} = [(r - d_1 - \frac{S}{k}) + \frac{e1a1N(S + I)}{S(1 + b1N)} - \frac{a2P}{1 + b2S} - \beta I]\, dt$$

which implies $\frac{dS}{S} = \alpha_2(N, S, I)\, dt$, where $\alpha_2(N, S, I) = [(r - d_1 - \frac{S}{k}) + \frac{e1a1N(S + I)}{S(1 + b1N)} - \frac{a2P}{1 + b2S} - \beta I]$.

Now integrating above differential equation in the region [0, t] we have,

$$S(t) = S(0)e^{\int_0^t \alpha_2(N,S,I)dt} > 0,\ \forall t.$$

From the third equation of Equation (3) we can write,

$$\frac{dI}{I} = [\beta S - \frac{a2P}{1 + b2I} - d_2]\, dt$$

which implies $\frac{dI}{I} = \alpha_3(S, I)\, dt$, where $\alpha_3(S, I) = [\beta S - \frac{a2P}{1 + b2I} - d_2]$.

Now integrating above differential equation in the region [0, t] we have,

$$I(t) = I(0)e^{\int_0^t \alpha_{3(S,I)}dt} > 0,\ \forall t.$$

From the last equation of Equation (3) we can write,

$$\frac{dP}{P} = [\frac{e2a2S}{1 + b2S} + \frac{e3a2I}{1 + b2I} - d3]\, dt$$

which implies $\frac{dP}{dt} = \alpha_4(S, I)\, dt$, where $\alpha_4(S, I) = [\frac{e2a2S}{1 + b2S} + \frac{e3a2I}{1 + b2I} - d_3]$.

Now integrating above differential equation in the region [0, t] we have,

$$N(t) = N(0)e^{\int_0^t \alpha_{4(S,I)} dt} > 0, \forall t.$$

Hence, we conclude that all the solutions of Equation (4) are always positive. □

Theorem 2. *All the solutions of the Equation (4) which start in R_+^3 are uniformly bounded.*

Proof. Let N(t), S(t), I(t), P(t) be any solution of the system with positive initial conditions. Since from the first equation of Equation (3).

$$\frac{dN}{dt} \leq N(1 - N),$$

by standard comparison theorem, we have,

$$\lim_{t\to\infty} \sup N(t) \leq 1.$$

We define a function,

$$W = N + S + I + P.$$

Its time derivative along the solutions of Equation (3) is,

$$\frac{dW}{dt} = \frac{dN}{dt} + \frac{dS}{dt} + \frac{dI}{dt} + \frac{dP}{dt},$$

$$\frac{dW}{dt} = N(1 - N) - \beta SI - d_2 I - d_3 P,$$

$$\frac{dW}{dt} = N - N^2 - 1 - N + 1 + N - \beta SI - d_2 I - d_3 P,$$

$$\frac{dW}{dt} = [-(N^2 - 2N + 1) + 1 - N - \beta SI - d_2 I - d_3 P].$$

The following inequality holds:

$$\frac{dW}{dt} + LW \leq -(N - 1)^2 + 1,$$

where, $L = \min(1, \beta, d_2, d_3)$.

Hence,

$$\frac{dW}{dt} + LW \leq 1.$$

Applying a theory of differential equation we obtain,

$$0 < W(N, S, I, P) < \frac{1 - \exp(-Lt)}{L} + W(N(0), S(0), I(0), P(0))\exp(-Lt).$$

For $t \to \infty$, we have $0 < W < \frac{1}{L}$. All the solutions of the Equation (3) which start in R_+^3 are uniformly bounded.

From the above theorem we observed that all the solutions of the model Equation (3) initiating in R_+^3 eventually lie in the region B defined

$$B = \{(N, S, I, P): 0 \leq W \leq \frac{1}{L} + \varepsilon, \text{ for any } \varepsilon > 0\},$$

that means all trajectories of the model Equation (3) initiating from any point in R_+^3 ultimately lie in fixed bounded region defined by B. Hence, the dynamical system associated with model Equation (3) is dissipative. □

3.2. 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:

$$b_1^2N_3^3 + (2b_1 - b_1^2)N_3^2 + (e_1a_1^2k + 1 - 2b_1 + (r - d_1)a_1b_1k)N_3 + a_1k(r - d_1) - 1 = 0 \quad (4)$$

and $S_3 = \frac{(1 + b1N3)(1-N3)}{a1}$.

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 $b_1(e_2a_2 - b_2d_3)N_4^2 + (e_2a_2 - b_2d_3)(1 - b_1)N_4 + a_1d_3 - (e_2a_2 - b_2d_3) = 0$. $S_4 = \frac{d3}{e2a2 - b2d3}$ and $P_4 = \frac{(1 + b_2S_4)}{a_2}\left[(r - d_1 - \frac{S_4}{k}) + \frac{e_1a_1N_4}{1 + b_1N_4}\right]$.

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 $I_5 = \frac{\beta(1 - N5)(1 + b1N5) - a1d2}{a1\beta}$.

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 Equation (3) at any arbitrary point (N, S, I, P) is given by $(J_{ij})_{4\times4}$ where, $J_{11} = 1 - 2N - \frac{a_1(S + I)}{(1 + b_1N)^2}$, $J_{12} = J_{13} = \frac{-a1N}{1 + b1N}$, $J_{14} = 0$, $J_{21} = \frac{e_1a_1(S + I)}{(1 + b_1N)^2}$, $J_{22} = r - d_1 - \frac{2S}{k} + \frac{e_1a_1N}{1 + b_1N} - \frac{a_2P}{(1 + b_2S)^2} - \beta I$, $J_{23} = \frac{e_1a_1N}{1 + b_1N} - \beta S$, $J_{24} = \frac{- a2S}{1 + b2S}$, $J_{31} = 0$, $J_{32} = \beta I$, $J_{33} = \beta S - \frac{a_2P}{(1 + b_2I)^2} - d_2$, $J_{34} = \frac{- a2I}{1 + b2I}$, $J_{41} = 0$, $J_{42} = \frac{e_2a_2P}{(1 + b_2S)^2}$, $J_{43} = \frac{e_3a_2P}{(1 + b_2I)^2}$, $J_{44} = \frac{e_2a_2S}{1 + b_2S} + \frac{e_3a_2I}{1 + b_2I} - d_3$.

Theorem 3. *The trivial equilibrium point E_0 is always unstable. The axial equilibrium point E_1 is locally stable if $R_1 < 1$, where $R_1 = (\frac{1}{d1 - r})(\frac{e_1a_1}{1 + b_1})$. 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 eigenvalues associated with the Jacobian matrix computed around E_0 is $1 > 0$, 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 $R_1 = (\frac{1}{d1 - r})(\frac{e_1a_1}{1 + b_1})$.

The Jacobian matrix J_2 at predator free equilibrium point E_2 is given by $J_2 = (C_{ij})_{4\times4}$ where $C_{11} = 1 - a_1\theta$, $C_{12} = C_{13} = C_{14} = 0$, $C_{21} = e_1a_1\theta$, $C_{22} = -\frac{\theta}{k}$, $C_{23} = -\beta\theta$, $C_{24} = \frac{-a2\theta}{1 + b2\theta}$, $C_{31} = C_{32} = 0$, $C_{33} = \beta\theta - d_2$, $C_{34} = C_{41} = C_{42} = C_{43} = 0$, $C_{44} = \frac{e_2a_2\theta}{1 + b_2\theta} - d_3$.

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 $D_{11} = -N_3 + \frac{a_1b_1N_3S_3}{(1 + b_1N_3)^2}$, $D_{12} = D_{13} = -\frac{a_1N_3}{1+b_1N_3}$, $D_{14} = 0$, $D_{21} = \frac{e_1a_1S_3}{(1 + b_1N_3)^2}$, $D_{22} = -\frac{S_3}{k}$, $D_{23} = \frac{e_1\,a_1N_3}{1+b_1N_3} - \beta S_3$, $D_{24} = -\frac{a_2S_3}{1+b_2S_3}$, $D_{31} = D_{32} = 0$, $D_{33} = \beta S_3 - d_2$, $D_{34} = D_{41} = D_{42} = D_{43} = 0$, $D_{44} = \frac{e_2a_2S_3}{1+b_2S_3} - d_3$.

The characteristics 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

$$\lambda^2 + (N_3 - \frac{a_1b_1N_3S_3}{(1+b_1N_3)^2} + \frac{S_3}{k})\lambda + (N_3 - \frac{a_1b_1N_3S_3}{(1+b_1N_3)^2})\frac{S_3}{k} + (\frac{a_1N_3}{1+b_1N_3})(\frac{e_1\,a_1S_3}{(1+b_1S_3)^2}) = 0.$$

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., $(1+b_1N_3)^2 > a_1b_1\,S_3$, 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 4. *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 $H_{44} < 0$) hold, 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. □

3.3. 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,$V_{11} = \frac{a_1b_1N_6(S_6 + I_6)}{(1 + b_1N_6)^2} - N_6$, $V_{12} = V_{13} = -\frac{a_1N_6}{1+b_1N_6}$, $V_{14} = 0$, $V_{21} = \frac{e_1a_1(S_6 + I_6)}{(1 + b_1N_6)^2}$, $V_{22} = -\frac{S_6}{k} - \frac{e_1a_1N_6I_6}{S_6(1+b_1N_6)} + \frac{a_2b_2S_6P_6}{(1 + b_2S_6)^2}$, $V_{23} = \frac{e_1a_1N_6}{1+b_1N_6} - \beta S_6$, $V_{24} = -\frac{a_2S_6}{1+b_2S_6}$, $V_{31} = 0$, $V_{32} = \beta I_6$, $V_{33} = \frac{a_2b_2I_6P_6}{(1 + b_2I_6)^2}$, $V_{34} = -\frac{a_2I_6}{1+b_2I_6}$, $V_{41} = 0$, $V_{42} = \frac{e_2a_2P_6}{(1 + b_2S_6)^2}$, $V_{43} = \frac{e_3a_2P_6}{(1 + b_2I_6)^2}$, $V_{44} = 0$.

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 eigenvalues(β^*), $\bar{\lambda}(\beta^*) \in \sigma(\beta)$ is such that Re $\lambda(\beta^*) = 0$, Im $\lambda(\beta^*) = \omega_0 > 0$, mand the transversality condition $\left[\frac{d\mathrm{Re}\lambda(\beta)}{d\beta}\right]_{at(\beta^*)} \neq 0$;

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

Now we present a theorem for Hopf bifurcation.

Theorem 5. *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 eigenvalues 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

$$(\lambda^2 + \frac{\sigma_3}{\sigma_1})(\lambda^2 + \sigma_1\lambda + \frac{\sigma_1\sigma_4}{\sigma_3}) = 0 \quad (5)$$

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

$$\lambda_3 + \lambda_4 = -\sigma_1 \quad (6)$$

$$\omega_0^2 + \lambda_3\lambda_4 = \sigma_2 \quad (7)$$

$$\omega_0^2(\lambda_3 + \lambda_4) = -\sigma_3 \quad (8)$$

$$\omega_0^2\lambda_3\lambda_4 = \sigma_4 \quad (9)$$

where $\omega_0 = \mathrm{Im}\ \lambda_1(\beta^*)$. By above $\omega_0 = \frac{\sigma_3}{\sigma_1}$. Now, if λ_3 and λ_4 are complex conjugates, then from Equation (5), it follows that $2\mathrm{Re}\lambda_3 = -\sigma_1$; if they are real roots, then by Equations (8) and (9) $\lambda_3 < 0$ 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 conjugates for β. Suppose, their general forms in this neighborhood 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 (5) 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 various mathematical experiments to evaluate the global behavior of the model. In this study, the disease rate β in the mesopredator group is a new change from most previous studies. a1 = 4.9, a2 = 0.1, b1 = 2.9, b2 = 2.0, d1 = 0.4, d2 = 0.41, d3 = 0.01, e1 = 0.98, e2 = 0.6, d3 = 0.9 r = 0.01, we get the parameter set k = 0.5, b = 8.2. Throughout the numerical experiments, we fixed the above method of measurement parameters, mainly taken from Hastings and Powels[18]. We first considered the evolution of our claim to understanding changes in the transmission of disease between animals. Finally, we look at the role of other foods. The implementation of disease by host animals is a critical issue in tri-trophic food systems.

We observed chaotic dynamics for process parameter values in our scheme (**Figure 1**). We make a diagram of the chaotic dynamics (**Figure 2**) and see that this will lead to the shape of the teacup attractor. Now let's analyze the motion of the teacup puller. The dynamics in the tractor are roughly as follows. The system starts from the handle of the tea glass, moves towards the tip of the tea glass, then draws spirals along the tea glass, moving towards the narrow end and back into the handle. In terms of species behavior, the top animal Z collapses, causing large population level changes in X and Y1 + Y2.

As the value of Z increases, the changes in X and Y1+Y2 weaken until Z collapses at the Y1+Y2 level.

This causes Z to explode and X to explode, restarting the process. The sequence of events always follows a general pattern depending on the number of species. What is unpredictable is time. One way to express this is that the duration of the Z type collapse is variable.

In addition, the peak of type Y is different from the crack size, and the population size of the peak is also different. The delicate dependence of future dynamics in the present case, i.e., chaotic character, is due to the fact that all trajectories of the teacup handle are very close.

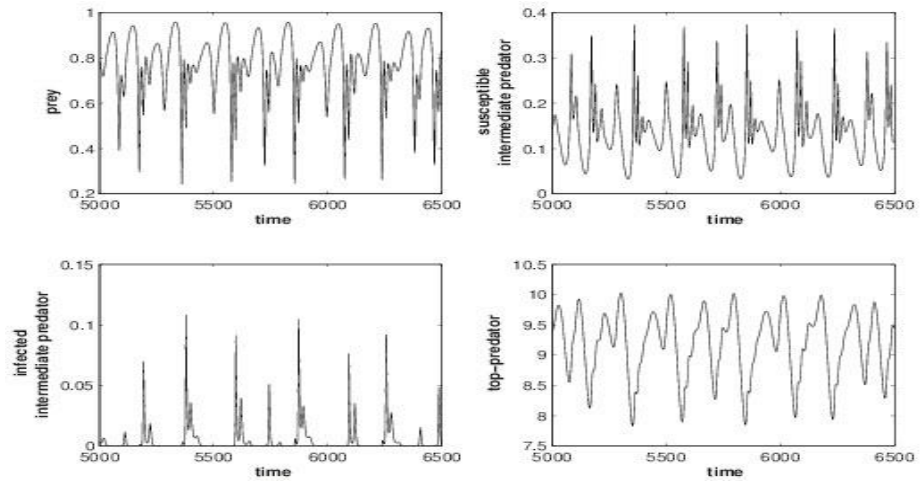

Figure 1. The time series solution of the model Equation (3) for a1 = 4.9, a2 = 0.1, b2 = 2.0, d1 = 0.4, d2 = 0.41, d3 = 0.01, e1 = 0.98, e2 = 0.95, e3 = 0.6, b1 = 2.9, r = 0.01, k = 0.5 and β = 8.2.

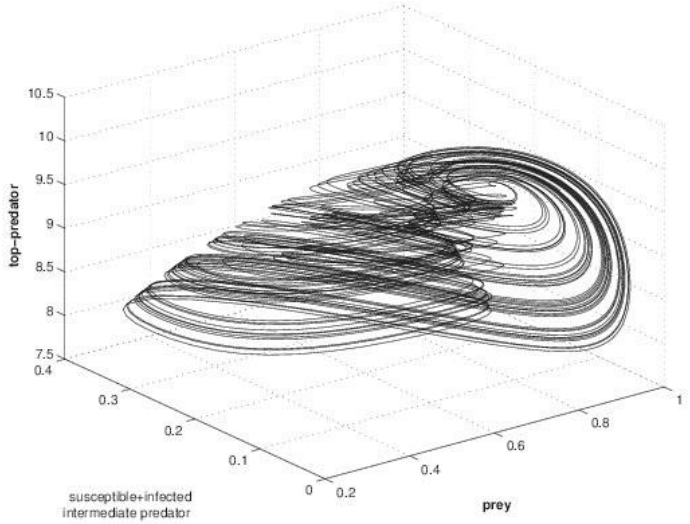

Figure 2. The phase plane of the model Equation (3) for a1 = 4.9, a2 = 0.1, b2 = 2.0, d1 = 0.4, d2 = 0.41, d3 = 0.01, e1 = 0.98, e2 = 0.95, e3 = 0.6, b1 = 2.9, r = 0.01, k = 0.5 and β = 8.2.

If we increase the β value from 8.2 to 9.6, we observe quasiperiodic dynamics (**Figure 3**). If we add the β voltage, we see that the quasiperiodic dynamics decreases to avoid period oscillations (**Figure 4**). Finally, we see that the system oscillates from its limit cycle to a steady state (**Figure 5**). To better understand the behavior of the system, we draw a bifurcation diagram (**Figure 6**), from which we see that the system moves from chaotic dynamics to a steady state, causing diffusion. It is seen that the system transitions from a chaotic state to a stable state, which increases the transmission power (β). We observe that the system enters quasiperiodic chaos; It limits the loop oscillations with quasiperiodic dynamics and finally provides a stable environment by limiting the loop oscillations for further conduction. Therefore, it is clear that when the infection level is low, the system exhibits a chaotic dynamic, while when the infection level is high, the chaos decreases to a constant level. Current mathematical models show that the introduction of pathogens into animal populations tends to affect livestock producing communities. This has been proposed for microparasites with direct life[3,11,33], and

indirect life[8,34]. Macroscopic parasite models generally favor instability because they account for the parasite in the host in additional equations[1,8,35]. Here we show that chaotic dynamics reduce to a stable analysis to have transfer.

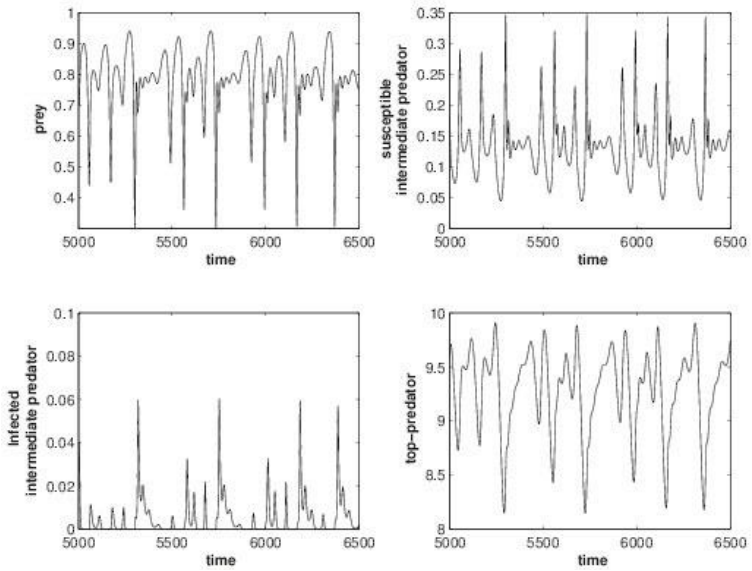

Figure 3. The time series quasi-periodic dynamics of the Equation (3) for β = 9.6 and other parameter values given in the **Figure 1**.

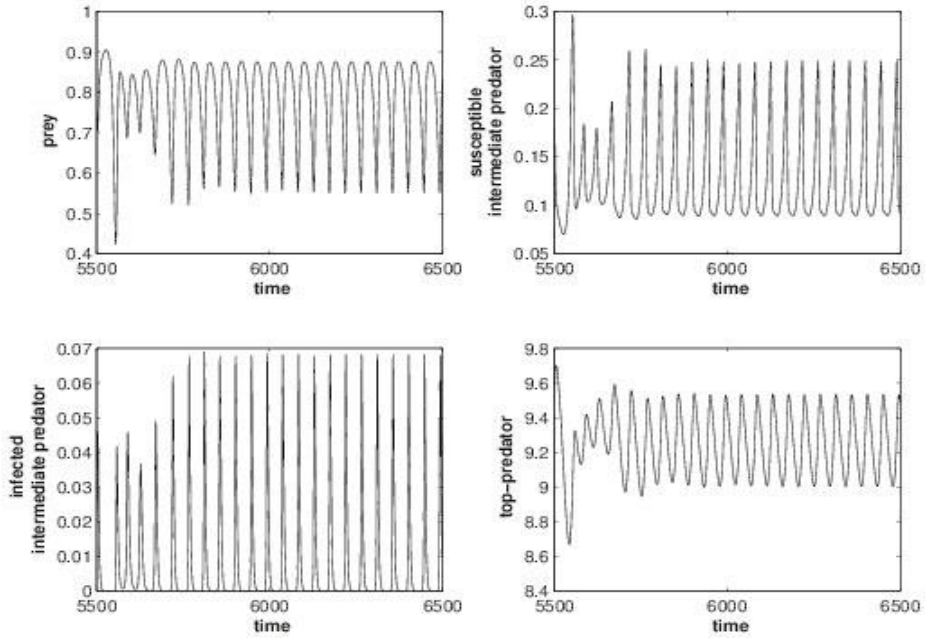

Figure 4. The time series limit cycle oscillation of the Equation (3) for β = 10.2 and other parameter values given in the **Figure 1**.

Introduction of the virus into the central predator of the tritrophic food model may have a direct effect of increasing the stress of the predator. We demonstrate this in a threetrophic food model in which mesopredators are infected. Thus, our model brings together two fields of ecology and epidemiology as it expands the spread of infectious diseases through population interactions and interventions. From the above discussion, we see that when the disease is at a low level, the system exhibits chaotic dynamics. We now want to analyze the role of other nutrients in chaotic dynamics. To do this, we will change the carrying capacity of the mesopredator k. As can be seen from **Figure 7**, the

system exhibits chaotic dynamics at k = 0.5. Now if we reduce the value of k from 0.5 to 0.2, the system enters a quasiperiodic process (**Figure 4**). If we further reduce the value of k, we observe a quasiperiodic extinction with the extinction of intermediate predators (**Figure 8**). We also prepare the bifurcation graph (**Figure 9**) to observe the real dynamic behavior of k change. As a result, when other foods are reduced, stress disappears and infections disappear from the body, but when other foods are increased, the chaos in our body remains unchanged. Therefore, other nutrients can be used as preservatives in our body.

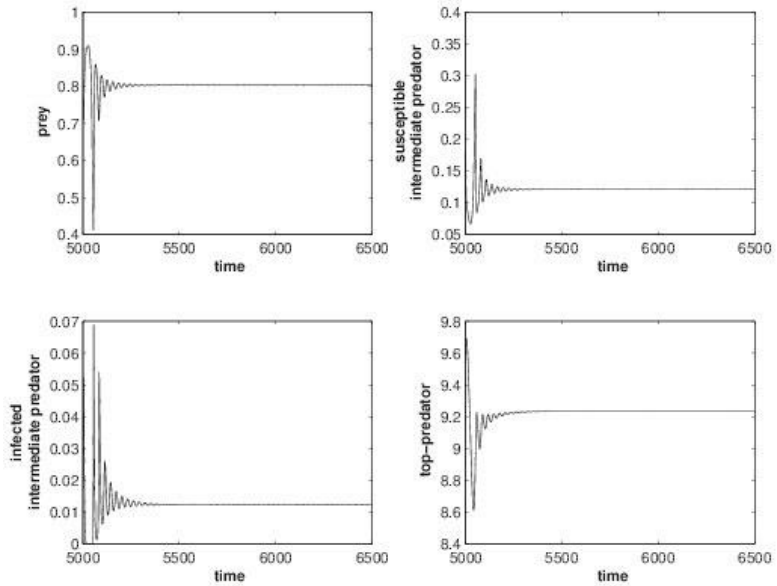

Figure 5. The time series stable solution of the Equation (3) for β = 10.5 and other parameter values given in the **Figure 1**.

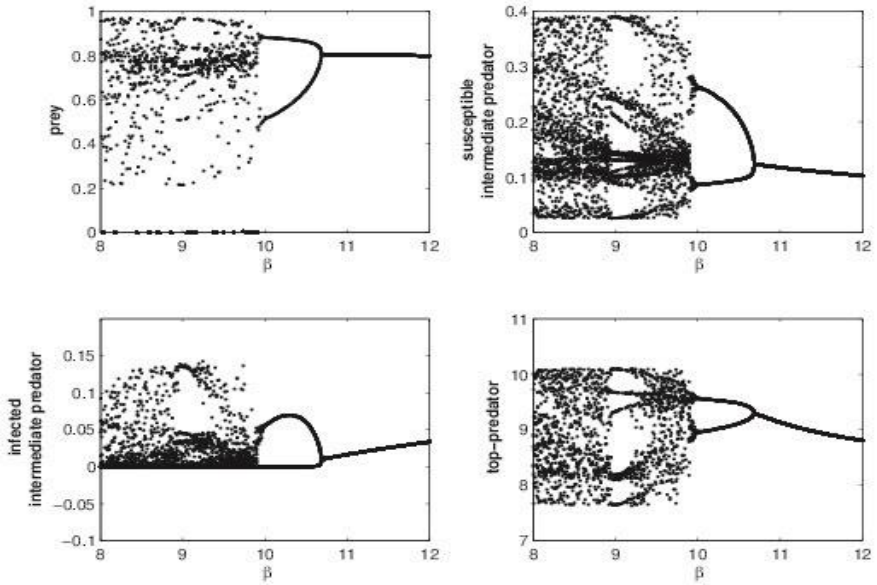

Figure 6. Figure shows the bifurcation diagram for β ϵ [8.0, 11.0] and other parameter values given in **Figure 1**.

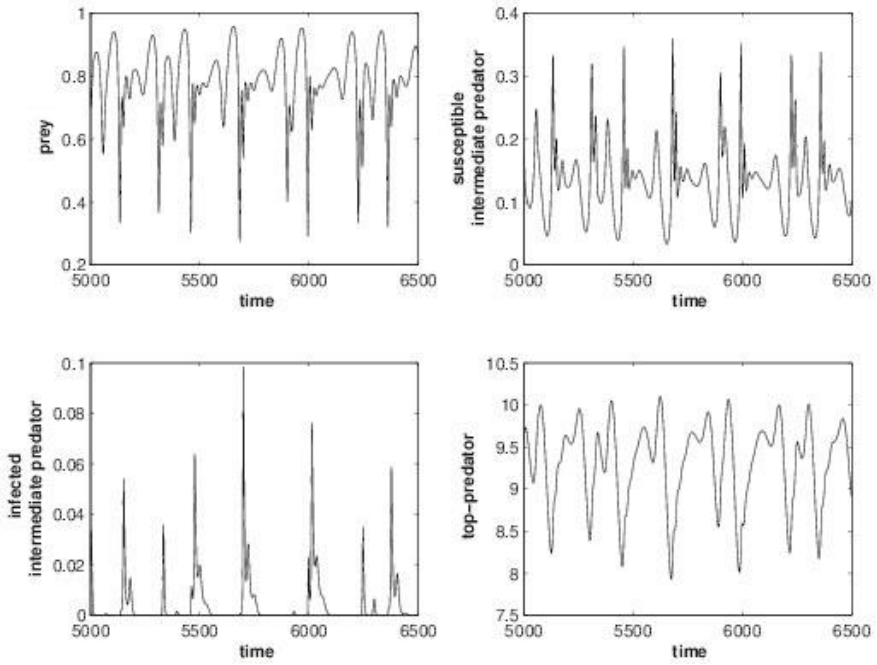

Figure 7. The time series chaotic dynamics of the Equation (3) for k = 0.2, r = 0.01, β = 9.0 and other parameter values given in the **Figure 1**.

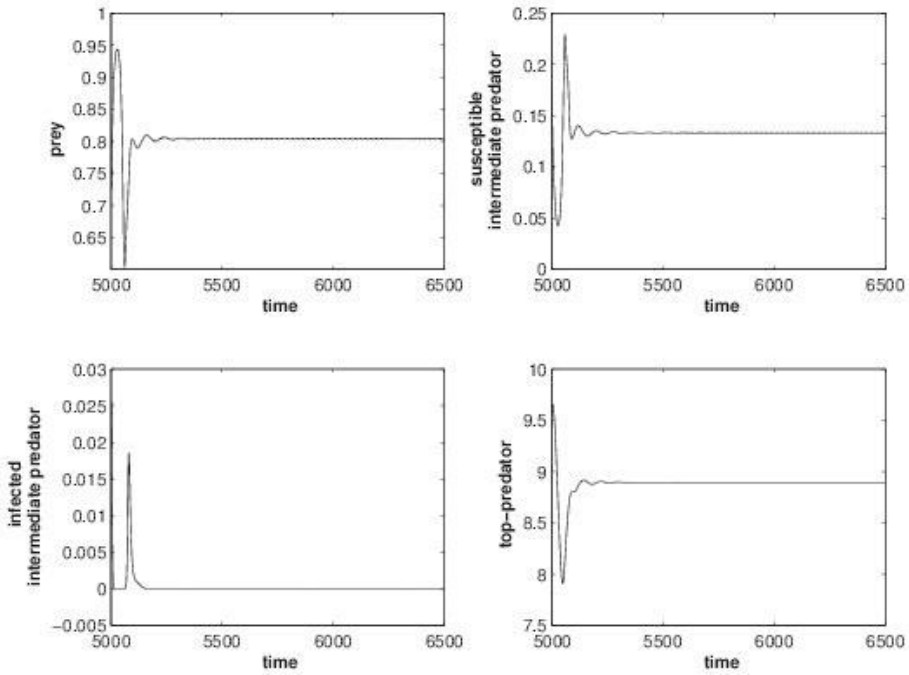

Figure 8. The time series chaotic dynamics of the Equation (3) for k = 0.02, r = 0.01, β = 9.0 and other parameter values given in the **Figure 4**.

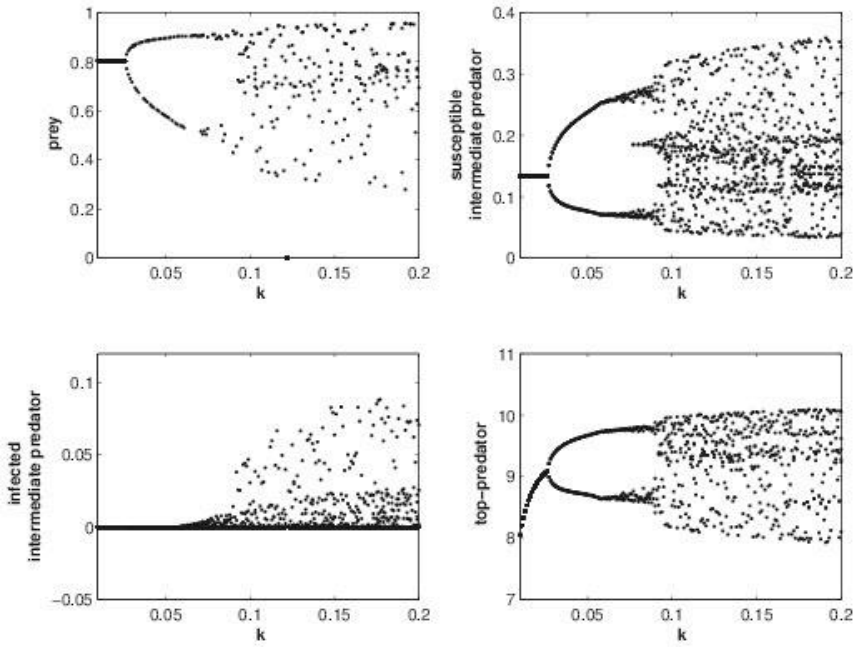

Figure 9. The bifurcation diagram of the Equation (3) for k ϵ [0.02, 0.2], r = 0.01 and other parameter values given in **Figure 1**.

We will now describe the new information obtained from this study. We see that at low infection levels, the system exhibits chaotic dynamics, while at high infection levels, chaos becomes the target of stability. We have also seen some negative events leading to the transmission of intermediate species, from chaos to quasi-periodicity; quasi-periodic to limit cycle; Limit the cycle to stability. We also look at the effects of other foods on chaotic dynamics. It has been shown that when other nutrients are at low levels, stress disappears and animalborne diseases are eliminated from the body, but when other nutrients are at high levels, the stress pressure in the body does not change. We have shown that, to our knowledge, the conflict observed in the system can be stabilized by the spread of disease in the mesopredator population. This contradicts existing theory regarding the instability of the affected organism[3,5,8,11,33–35]. Our findings also have implications for health management, as infectious diseases can be used as antidotes against harmful species such as invaders. Interestingly, this research shows that parasites can affect different trophic levels and can be used for management in different systems. Disease control not only controls or eliminates livestock, but also allows the animal to recover. For example, bacteria can be used to control domestic animals such as feral domestic cats (predators) on oceanic islands, which have a significant impact on wild animals such as seabirds[36–38].

Other foods are also control agents in our model. This study provides information on establishing ecological and epidemiological systems to help understand how diseases affect society. We also present a comparison with most previous studies. Hadeler and Freedman[5] evaluated, developed and analyzed a predatorprey model in which two species are affected by parasitism. They also suggested that animals could become infected by eating livestock, and that animals could become infected by parasites transmitted from livestock to the environment. There is no fire. It also shows that the parasite can increase the risk to livestock in cases where the animal cannot survive on livestock alone in a diseasefree environment, as it can only survive on livestock if some animals are infected with certain diseases. It is easier to catch the disease. In Hsieh and Hsiao's study[39], animals could become infected when they approached or were near animals during hunting, but animals could not become infected from each other. They showed that infected animals played a minor and indirect role in the spread of the disease; this was mainly due to the assumption that animals could not infect other members of the

population. Recently, Hilker and Schmitz[40] evaluated the impact of the prey population on infectious diseases frequently transmitted in the predator population. These are provided by music and consensus (population numbers and epidemics) that allow them to determine the entire society. Their findings contradict predictions from previous models showing a negative effect of the parasite and suggest that the predator prevents the beneficial effect. In this study, a threetrophic food model in which only the intermediate predator is an insect and the intermediate predator has other foods is considered. We see chaotic dynamics becoming a stable environment for infection. We also look at the effects of other foods on chaotic dynamics. It has been found that at low levels of other foods, clutter disappears and disease among animals is eliminated from the body, but at high levels of other foods, clutter is still unchanged in our bodies. Recently, Mandal et al.[41] Consider a predatorprey system in which victims are affected by a disease. They observed changes in this system under the influence of severe and invisible diseases and other food predators. They believe that hunters only choose infected animals for their food because these animals are more dangerous. The results show that when the disease is severe, the animal population prefers other foods over infected foods. However, if the parasite attack is not obvious, the strategy is reversed. In this study, we considered the tritrophic food model, in which disease spreads between humans and animals, and other food is found among animals. We see that when the infection level is low, the system exhibits stress, while at high levels, stress becomes the key factor in maintaining stability. We also look at the effects of other foods on chaotic dynamics. When other nutrients increase, the stress in the body remains constant, but when other nutrients decrease, the stress disappears and intermediate organisms are eliminated from the body.

5. Conclusion

Infectious diseases control not only their own populations but also other species to which their hosts belong[11]. In this study, we consider a tritrophic food model that results from bacterial infection of a medium containing other nutrients. We track the local security of our system models closely into the performance equation. We report the ecology and disease of a significant number of children and identify community patterns in the sample. We performed several simulations to evaluate the global behavior of the model system. We saw stress when the infection rate was low, doubling the duration and limited cycles when the infection was high. Finally, we see that the crisis has become the main element of stability during the epidemic period. Nonlinear interactions between predators and prey are known to produce endogenous oscillations. As we know, random changes in the three trophic trophic structures can be offset by the spread of diseases among animals. This contrasts with current theory regarding the instability of associated diseases[11,42]. We also looked at the effect of other foods on chaos dynamics, finding that when other foods increase, the stress in the body does not change, but when other foods decrease, the stress leads to stability and diseases among animals are eliminated from the body.

Author contributions

Conceptualization, KPD and PR; methodology, AS; software, AS; validation, PR, AS and KDP; formal analysis, PR; investigation, AS; resources, KPD; data curation, AS; writing—original draft preparation, AS; writing—review and editing, KA and KPD; visualization, KA; supervision, KA; project administration, KPD; funding acquisition, PR. All authors have read and agreed to the published version of the manuscript.

Conflict of interest

The authors declare no conflict of interest.

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