Disease control in a food chain model supplying alternative food

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ABSTRACT

Necessity to find a non-chemical method of disease control is being increasingly felt due to its eco-friendly nature. In this paper the role of alternative food as a disease controller in a disease induced predator–prey system is studied. Stability criteria and the persistence conditions for the system are derived. Bifurcation analysis is done with respect to rate of infection. The main goal of this study is to show the non-trivial consequences of providing alternative food in a disease induced predator–prey system. Numerical simulation results illustrate that there exists a critical infection rate above which disease free system cannot be reached in absence of alternative food whereas supply of suitable alternative food makes the system disease free up to certain infection level. We have computed the disease free regions in various parametric planes. This study is aimed to introduce a new non-chemical method for controlling disease in a predator–prey system.

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1. Introduction

Mathematical models are increasingly used to guide public health policy decisions and for controlling infectious disease. Epidemic dynamics is an important method of studying the spread of infectious disease qualitatively and quantitatively. It is based on the specific property of population growth, the spread rules of infectious diseases, and the related social factors to construct mathematical models reflecting the dynamic properties of infectious diseases, to analyse the dynamical behaviour and to do some simulations. The research results are helpful to predict the developing tendency of the infectious disease, to determine the key factors of the spread of infectious disease and to seek the optimum strategies for preventing and controlling the spread of infectious diseases. Mathematical models have a long history in infectious disease ecology starting with Bernoulli's [1] modelling of smallpox and including Ross's [2] analysis of malaria. The earliest attempt to provide a quantitative understanding of the dynamics of malaria transmission was that of Ross (1911). Ross models consisted of a few differential equations to describe changes in densities of susceptible and infected people, and susceptible and infected mosquitoes. Based on his modelling, Ross introduced the concept of a threshold density and concluded that 'in order to counteract malaria anywhere we need not banish Anopheles there entirely we need only to reduce their numbers below a certain figure [3]. Classical papers of mathematical modelling of infectious disease was by Kermack and McKendrick (1927 [4], 1932 [5], and 1933 [6]). These papers had a major influence on the development of mathematical models for disease spread and are still relevant in many epidemic situations. Aim of ecological modelling is to understand the prevalence and distribution of a species, together with the factors that determine incidence, spread, and persistence (Anderson and May [7]; May and Anderson [8]; Bascompte and Rodriguez-Trelles [9]). We now have models for many of the most important human emerging infectious diseases e.g., HIV (May and Anderson [10]), malaria (Aron and May [11]; Macdonald [12]), SARS-coronavirus (Anderson et al. [13]), rabies (Murray and Seward [14]), and influenza (Ferguson and Anderson [15]). Mathematical models

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are also being used to explore wildlife disease dynamics (Grenfell and Dobson [16]; Hudson et al.[17]) and possible routes of zoonotic disease emergence. Understanding disease dynamics across hosts is an essential first step in understanding and articulating those conditions under which new diseases can emerge from wildlife reservoirs [18]. A predator–prey system with infected prey in polluted environment is proposed by Sinha et al. [19]. Anderson and May [20] were probably the first who considered the disease factor in a predator–prey dynamics and found that the pathogen tends to destabilize the predator–prey interaction. In Rosenzweig predator–prey model, Hadeler and Freedman [21] determined a threshold above which an infected equilibrium or an infected periodic solution appear. Chattopadhyay and Arino [22] considered a three species ecoepidemiological model and studied local stability of equilibrium points, extinction criteria of species and found condition for Hopf-bifurcation in an equivalent two-dimensional model. Haque and Chattopadhyay [23] studied the role of transmissible diseases in a prey dependent predator–prey system with prey infection. Bhattacharyya et al. [24] proposed a epidemiological model with nonlinear infection incidence. In 2012, scientists [25,26] investigated infectious disease model and suggested disease control strategies.

Over the last 50 years, disease control has relied heavily on the use of chemical fungicides, bacteriocides and soil fumigants. However, there are now many problems associated with their continued deployment including increasing pressure to reduce chemical use in the environment in general, development of pesticide resistance in many pathogens, and decreasing availability of active ingredients through stricter registration and difficulty in finding novel active compounds. Consequently, the search for alternative non-chemical methods of disease control continues to gain significance. Some investigations [27–29] were done for population control in a predator–prey model providing alternative food to predator. Disease controlling aspects of predator–prey model providing alternative food to predator is unexplored. In this paper we formulate a diseased predator–prey model in presence of alternative food for predator. This is a totally new research area which has applications in many important practical fields. This paper aims to analyse the role of alternative food for controlling disease in the system.

To model the disease induced predator–prey system with alternative food to predator we do the following assumptions:

(a) In the presence of disease, the prey population consists of two sub classes, namely, the susceptible prey $S(t)$ and infected prey $I(t)$ and the density of the predator is denoted by $P(t)$ at time t.
(b) In presence of disease, the susceptible prey population grows logistically with intrinsic growth rate R_0 and environmental carrying capacity K_0.
(c) The susceptible prey population become infected at a rate of W, following the law of mass action.
(d) Infected prey population is not in a state of reproduction and does not compete for the resource.
(e) The interaction between predator and susceptible prey is of Holling type-II and that between predator and infected prey is of Holling type-I (mass action law) in presence of alternative food. This combination of functional forms are taken because the capturing of infected prey is easier than the susceptible prey.
(f) Predators are provided with alternative food of constant biomass A which is distributed uniformly in the habitat. The constant biomass assumption is valid for many arthropod predators because they can feed on plant-provided alternative food sources such as pollen or nectar which approximately remains constant.
(g) The number of encounters per predator with the alternative food is proportional to the density of the alternative food.
(h) The proportionality constant characterizes the ability of the predator to identify the alternative food.

With the above assumptions, we formulate the following model:

$$\frac{dS}{dT} = R_0 S\left(1 - \frac{S}{K_0}\right) - WSI - A_1 \frac{SP}{B_1 + \alpha\mu A + S},$$
$$\frac{dI}{dT} = WSI - A_2 \frac{IP}{B_1 + \alpha\mu A} - D_1 I,$$
$$\frac{dP}{dT} = A_1 C_1 \frac{(S + \mu A)P}{B_1 + \alpha\mu A + S} + C_2 A_2 \frac{IP}{B_1 + \alpha\mu A} - D_2 P. \tag{1}$$

The constants A_1, A_2 are maximal predation rate of predator for susceptible prey and infected prey, respectively. The terms C_1, C_2 are conversion rates of susceptible prey and infected prey to predator, respectively; B_1 is the half saturation constant for predator; D_1 and D_2 are constant death rates for I and P, respectively. If h_1 represents the handling time of the predator per prey and h_2 represents the handling time of the predator per unit quantity of alternative food, then $\alpha = h_2/h_1$. If the constant e_1, e_2 represent ability of the predator to detect the prey item and to detect alternative food, respectively, then $\mu = e_2/e_1$. The term μA represents effectual alternative food for the predator.

We nondimensionalize the system (1) using $s = \frac{S}{K_0}, i = \frac{I}{K_0}, p = \frac{P}{K_0}$ and $t = R_0 T$ and obtain the following system

$$\frac{ds}{dt} = s(1 - s) - \gamma is - \frac{asp}{1 + \alpha\xi + bs},$$
$$\frac{di}{dt} = \gamma is - \frac{\beta ip}{1 + \alpha\xi} - di,$$
$$\frac{dp}{dt} = \frac{\epsilon(s + c\xi)p}{1 + \alpha\xi + bs} + \frac{\eta ip}{1 + \alpha\xi} - ep, \tag{2}$$

where $\gamma = \frac{WK_0}{R_0}, a = \frac{A_1 K_0}{R_0 B_1}, \xi = \frac{\mu A}{B_1}, b = \frac{K_0}{B_1}, \beta = \frac{A_2 K_0}{R_0 B_1}, \epsilon = \frac{C_1 A_1 K_0}{R_0 B_1}, c = \frac{B_1}{K_0}, \eta = \frac{C_2 A_2 K_0}{R_0 B_1}, d = \frac{D_1}{R_0}$ and $e = \frac{D_2}{R_0}$. We analyse the system dynamics assuming $s(t) \geqslant 0, i(t) \geqslant 0$ and $p(t) \geqslant 0$.

The constant γ represents the infection rate of the prey. The terms α and ξ are the parameters which characterize the alternative food. From the relation $\alpha = h_2/h_1$, it can be inferred that α is directly proportional to the handling time h_2 of the alternative food. Hence the parameter α is proportionally related to the "quality" of the alternative food. If the relation $h_2 < h_1$ holds, then the predator can easily capture alternative food than prey species which implies that the alternative food is of high quality. Therefore for high quality of alternative food α is less than 1. Again, from the relation $\xi = \frac{\mu A}{B_1}$, it can be inferred that ξ is directly proportional to the biomass of the alternative food (A) and thus ξ is a representative of the "quantity" of the alternative food that is supplied to predator [28].

This paper is organized as follows: in Section 2, the existence condition for equilibrium points of the system are derived. The stability analysis of equilibrium points are done in Section 3. Persistence conditions of the system are derived in Section 4. We have analysed the dynamics of this model through bifurcation analysis with respect to the rate of infection and quantity of alternative food in Section 5. Finally, we draw conclusions in Section 6.

2. Theoretical study

In this section, positivity and boundedness for the system (2) are established. Since the state variables s, i and p represent population size, positivity implies that they never become negative. The boundedness may be interpreted as a natural restriction to growth as a consequence of limited resources.

2.1. Positive invariance

The system (2) can be written as $\dot{\overline{X}} = F(\overline{X})$ with $\overline{X}(0) = \overline{X}_0 \in R_+^3$, where $\overline{X} = (s, i, p)^T \in R_+^3$ and $F(\overline{X})$ is given by

$$F = F(\overline{X}) = \begin{pmatrix} s(1-s) - \gamma is - \frac{asp}{1+\alpha\xi+bs} \\ \gamma is - \frac{\beta ip}{1+\alpha\xi} - di \\ \frac{\epsilon(s+c\xi)p}{1+\alpha\xi+bs} + \frac{\eta ip}{1+\alpha\xi} - ep \end{pmatrix},$$

where $F : C_+ \to R^3$ and $F \in C^\infty(R^3)$.

It can be shown that whenever $\overline{X}(0) \in R_+^3$ such that $X_i = 0$ then $F_i(\overline{X})|_{X_i=0} \geqslant 0$ (for i = 1, 2, 3). Now any solution of $\dot{\overline{X}} = F(\overline{X})$ with $\overline{X}_0 \in R_+^3$, say $\overline{X}(t) = \overline{X}(t, \overline{X}_0)$, is such that $\overline{X}(t) \in R_+^3$ for all $t > 0$ (Nagumo [30]).

2.2. Boundedness

Theorem 1. *All solutions of the system (2) which initiated in R_+^3 are uniformly bounded.*

Proof. Let $(s(t), i(t), p(t))$ be any solution of the system (2) with positive initial condition.
Let us consider that, $w = s + i + p$

$$\frac{dw}{dt} = \frac{ds}{dt} + \frac{di}{dt} + \frac{dp}{dt}.$$

Using Eq. (2), we have

$$\frac{dw}{dt} = s(1-s) - \frac{(a-\epsilon)sp}{1+\alpha\xi+bs} + \frac{\epsilon c\xi p}{1+\alpha\xi+bs} - \frac{(\beta-\eta)ip}{1+\alpha\xi} - di - ep.$$

Therefore, $\frac{dw}{dt} \leqslant -\theta(s+i+p) - (1-s)^2 + 1$, where θ = min $\{1, d, e - \epsilon c\xi\}$.

Thus, $\frac{dw}{dt} + \theta w \leqslant 1 - (1-s)^2$.

That is, $\frac{dw}{dt} + \theta w \leqslant 1$, since $(1-s)^2 \geqslant 0$.

Applying the theory of differential inequality we obtain

$$0 < w < \frac{1 - e^{-\theta t}}{\theta} + w(s(0), i(0), p(0))e^{-\theta t}.$$

For $t \to \infty$, we have $0 < w < \frac{1}{\theta}$.

Hence all the solutions of (2) that initiate in R_+^3 are confined in the region.
$B = \{(s, i, p) \in R_+^3 : w = \frac{1}{\theta} + \tau, \text{ for any } \tau > 0\}$. This proves the theorem.

2.3. Extinction criterion of s and i

Lemma 1. *If* $1 \leqslant \gamma i(t)$, *then* $\lim_{t\to\infty} s(t) = 0$. *If* $\gamma s(t) \leqslant \frac{\beta p(t)}{1+\alpha\xi}$, *then* $\lim_{t\to\infty} i(t) = 0$.

Proof. $\frac{ds}{dt} = s(1-s) - \gamma is - \frac{asp}{1+\alpha\xi+bs} \leqslant s(1-\gamma i)$.

Therefore, $s(t) \leqslant s(t_0) exp(\int_{t_0}^{t}(1-\gamma i(t))dr)$.

Hence $\lim_{t\to\infty} s(t) = 0$, provided $1 \leqslant \gamma i(t)$.

$\frac{di}{dt} = \gamma is - \frac{\beta ip}{1+\alpha\xi} - di \leqslant i(\gamma s - \frac{\beta p}{1+\alpha\xi})$.

Thus, $i(t) \leqslant i(t_0) exp(\int_{t_0}^{t}(\gamma s(t) - \frac{\beta p(t)}{1+\alpha\xi})dr)$.

Hence $\lim_{t\to\infty} i(t) = 0$, provided $\gamma s(t) \leqslant \frac{\beta p(t)}{1+\alpha\xi}$, i.e., $0 \leqslant \gamma \leqslant \frac{\beta p(t)}{s(t)(1+\alpha\xi)}$.

2.4. Existence of equilibrium points

The system (2) possesses the following equilibrium points.

(i) The trivial equilibrium point $E_T = (0,0,0)$.
(ii) The axial equilibrium point $E_A = (1,0,0)$.
(iii) The disease free boundary equilibrium point is $E_D = (\bar{s}, 0, \bar{p})$, where $\bar{s} = \frac{e+\xi(e\alpha-\epsilon c)}{\epsilon-be}$, $\bar{p} = \frac{\epsilon}{a}[\frac{\epsilon-e-be-\xi(\epsilon c+e\alpha)}{(\epsilon-be)^2}](1+\alpha\xi-bc\xi)$. E_D exists if $e+\xi(e\alpha-\epsilon c) > 0$ and $\epsilon-e-be > \xi(\epsilon c+e\alpha)$. Therefore, the existence criteria of disease free boundary equilibrium point is $\frac{e}{\epsilon c-e\alpha} < \xi < \frac{\epsilon-e-be}{\epsilon c+e\alpha}$.
(iv) The predator free boundary equilibrium point $E_P = (\hat{s}, \hat{i}, 0)$, where $\hat{s} = \frac{d}{\gamma}$ and $\hat{i} = \gamma - d$. The predator free equilibrium point E_P exists if $\gamma > d$, i.e., if infection rate is greater than the death rate of infected prey.
(v) The steady state of coexistence $E^* = (s^*, i^*, p^*)$, where $p^* = \frac{(1+\alpha\xi)(d-\gamma s)}{\beta}$, $i^* = \frac{1+\alpha\xi}{\eta}[e - \frac{\epsilon(s+c\xi)}{1+\alpha\xi+bs}]$ and s^* is the positive root of the equation

$$T_1 s^{*2} + T_2 s^* + T_3 = 0,$$

where

$$T_1 = \eta\beta b,$$

$$T_2 = -[(1+\alpha\xi)\{a\eta\gamma - \eta\beta(1+eb-\epsilon)\} + \eta\beta b],$$

$$T_3 = -(1+\alpha\xi)[\eta\beta - a\eta d - \gamma\beta\{e(1+\alpha\xi) - \epsilon c\xi\}].$$

The sufficient conditions of the existence of E^* in the interior of the first octant are easily obtained as follows: $T_2^2 - 4T_1 T_3 \geqslant 0, d > \gamma s$ and $e > \frac{\epsilon(s+c\xi)}{1+\alpha\xi+bs}$.

Therefore, the values of E_D, E_P and E^* depend on the quality (α) and quantity (ξ) of alternative food. The existence conditions of E_D, E_P and E^* are also depend on α and ξ.

3. Local stability analysis

Theorem 2. *The trivial equilibrium point E_T is always unstable. The axial equilibrium point E_A is unstable if $\gamma > d$ and $e < \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b}$.*

Proof. The Jacobian matrix $J(E_T)$ at E_T is given by

$$J(E_T) = \begin{pmatrix} 1 & 0 & 0 \\ 0 & -d & 0 \\ 0 & 0 & \frac{\epsilon c\xi}{1+\alpha\xi} - e \end{pmatrix}.$$

We observe that $J(E_T)$ has one positive eigenvalue 1, therefore E_T is always unstable.

The Jacobian matrix $J(E_A)$ at E_A is given by

$$J(E_A) = \begin{pmatrix} -1 & -\gamma & \frac{-a}{1+\alpha\xi+b} \\ 0 & \gamma-d & 0 \\ 0 & 0 & \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b} - e \end{pmatrix}.$$

From the Jacobian matrix $J(E_A)$, it is observed that it has one negative eigenvalue -1 and two positive eigenvalues if $\gamma > d$ and $e < \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b}$. That means it has stable and unstable manifold in the neighbourhood of E_A. Hence the axial equilibrium point is unstable for $\gamma > d$ or $e < \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b}$.

Theorem 3. *The disease free equilibrium point E_D for the system (2) is locally stable if the following conditions hold: $\Theta_1 > 0, \Theta_3 > 0$ and $\Theta_1\Theta_2 - \Theta_3 > 0$, where Θ's are given in the proof of the theorem.*

Proof. The Jacobian matrix $J(E_D)$ at disease free equilibrium point E_D is given by

$$J(E_D) = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix},$$

where, $a_{11} = 1 - 2\bar{s} - \frac{a\bar{p}(1+\alpha\xi)}{(1+\alpha\xi+b\bar{s})^2}, a_{12} = -\gamma\bar{s}, a_{13} = \frac{-a\bar{s}}{1+\alpha\xi+b\bar{s}}, a_{21} = 0, a_{22} = \gamma\bar{s} - \frac{\beta\bar{p}}{1+\alpha\xi} - d, a_{23} = 0, a_{31} = \frac{\epsilon\bar{p}(1+\alpha\xi-bc\xi)}{(1+\alpha\xi+b\bar{s})^2}, a_{32} = \frac{\eta\bar{p}}{1+\alpha\xi}, a_{33} = \frac{\epsilon(\bar{s}+c\xi)}{1+\alpha\xi+b\bar{s}} - e.$

The characteristic equation of the Jacobian matrix $J(E_D)$ is given by

$$\lambda^3 + \Theta_1\lambda^2 + \Theta_2\lambda + \Theta_3 = 0,$$

where,

$$\Theta_1 = -(a_{11} + a_{22} + a_{33}),$$

$$\Theta_2 = (a_{22}a_{33} + a_{11}a_{33} - a_{31}a_{13} + a_{11}a_{22}),$$

and $\Theta_3 = a_{22}(a_{11}a_{33} - a_{13}a_{31})$.

If $a_{11} + a_{33} < 0, a_{22} < 0$ and $a_{11}a_{33} < a_{13}a_{31}$ then it is easy to see that $\Theta_1 > 0, \Theta_3 > 0$ and $\Theta_1\Theta_2 - \Theta_3 > 0$. Using the Routh–Hurwitz criteria [31] we observe that the system (2) is stable at the positive equilibrium point E_D if the conditions $\Theta_1 > 0, \Theta_3 > 0$ and $\Theta_1\Theta_2 - \Theta_3 > 0$ hold. Hence the disease free system is locally stable under this conditions. But, the disease free equilibrium E_D is unstable if at least one of these conditions is violated.

Theorem 4. *The predator free equilibrium point E_P is locally stable if $\frac{\epsilon(\hat{s}+c\xi)}{1+\alpha\xi+b\hat{s}} + \frac{\eta\hat{i}}{1+\alpha\xi} < e$ and $(\gamma - 2)\hat{s} < \gamma\hat{i} + d - 1$.*

Proof. The Jacobian matrix $J(E_P)$ at $E_P(\hat{s}, \hat{i}, 0)$ is given by

$$J(E_P) = \begin{pmatrix} 1 - 2\hat{s} - \gamma\hat{i} & -\hat{s}\gamma & -\frac{a\hat{s}}{1+\alpha\xi+b\hat{s}} \\ \gamma\hat{i} & \gamma\hat{s} - d & -\frac{\beta\hat{i}}{1+\alpha\xi} \\ 0 & 0 & \frac{\epsilon(\hat{s}+c\xi)}{1+\alpha\xi+b\hat{s}} + \frac{\eta\hat{i}}{1+\alpha\xi} - e \end{pmatrix}.$$

The characteristic roots of the Jacobian matrix $J(E_P)$ are $\frac{\epsilon(\hat{s}+c\xi)}{1+\alpha\xi+b\hat{s}} + \frac{\eta\hat{i}}{1+\alpha\xi} - e$ and the roots of the equation $\lambda^2 - [(\gamma - 2)\hat{s} - \gamma\hat{i} - d + 1]\lambda + \gamma^2\hat{s}\hat{i} + (\gamma\hat{s} - d)(1 - 2\hat{s} - \gamma\hat{i}) = 0$.

Hence E_P is locally stable if the conditions given in the theorem are satisfied.

Theorem 5. *The interior equilibrium point E^* for the system (2) is locally stable if the conditions $\Omega_1 > 0, \Omega_3 > 0$ and $\Omega_1\Omega_2 - \Omega_3 > 0$ hold, where Ω's are given in the proof of the theorem.*

Proof. The Jacobian matrix $J(E^*)$ at the interior point $E^*(s^*, i^*, p^*)$ is

$$J(E^*) = \begin{pmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{pmatrix},$$

where, $A_{11} = 1 - 2s^* - \gamma i^* - \frac{ap^*(1+\alpha\xi)}{(1+\alpha\xi+bs^*)^2}, A_{12} = -\gamma s^*, A_{13} = \frac{-as^*}{1+\alpha\xi+bs^*}, A_{21} = \gamma i^*, A_{22} = \gamma s^* - \frac{\beta p^*}{1+\alpha\xi} - d, A_{23} = -\frac{\beta i^*}{1+\alpha\xi}, A_{31} = \frac{\epsilon p^*(1+\alpha\xi-bc\xi)}{(1+\alpha\xi+bs^*)^2}, A_{32} = \frac{\eta p^*}{1+\alpha\xi}, A_{33} = \frac{\epsilon(s^*+c\xi)}{1+\alpha\xi+bs^*} + \frac{\eta i^*}{1+\alpha\xi} - e.$

The characteristic equation of the Jacobian matrix $J(E^*)$ is given by

$$\lambda^3 + \Omega_1\lambda^2 + \Omega_2\lambda + \Omega_3 = 0,$$

where,

$$\Omega_1 = -(A_{11} + A_{22} + A_{33}),$$

$$\Omega_2 = (A_{22}A_{33} - A_{23}A_{32}) + (A_{11}A_{33} - A_{31}A_{13}) + (A_{11}A_{22} - A_{12}A_{21}),$$

and $\Omega_3 = A_{11}(A_{22}A_{33} - A_{23}A_{32}) - A_{12}(A_{21}A_{33} - A_{23}A_{31}) + A_{13}(A_{21}A_{32} - A_{22}A_{31}).$

Using the Routh–Hurwitz criteria [31] we observe that the system (2) is locally stable at the equilibrium point E^* if the conditions $\Omega_1 > 0, \Omega_3 > 0$ and $\Omega_1\Omega_2 - \Omega_3 > 0$ hold.

Therefore, we observe that the stability conditions for all the equilibrium points depend on the parameters ξ and α.

4. Persistence of the system

Butler et al. [32], Freedman and Waltman [31,33] developed the following definition of persistence:
If a population $N(t)$ is such that $N(t) > 0$, then.

(i) $N(t)$ is said to be *weakly persistent* if $\lim_{t\to\infty} \sup N(t) > 0$.
(ii) $N(t)$ is said to be *(strongly) persistent* if $\lim_{t\to\infty} \inf N(t) > 0$.
    Further, if $N(t) \in C$, where C is a certain class of function, then
(iii) $N(t)$ is said to be *weakly uniformly persistent* if there exists $\delta > 0$ such that $\lim_{t\to\infty} \sup N(t) > \delta$ for all $N(t) \in C$.
(iv) $N(t)$ is said to be *uniformly persistent* if there exists $\delta > 0$ such that $\lim_{t\to\infty} \inf N(t) > \delta$ for all $N(t) \in C$.

From biological point of view, persistence of a system means the long term survival of all populations of the system, no matter what the initial populations are. Mathematically, persistence of a system means that strictly positive solutions do not have omega (Ω) limit points on the boundary of the non-negative cone.

Theorem 6. *Let the following conditions are satisfied*

(i) $\gamma > d$,
(ii) $e < \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b}$,
(iii) $\frac{\epsilon(\hat{s}+c\xi)}{1+\alpha\xi+b\hat{s}} + \frac{\eta\hat{i}}{1+\alpha\xi} > e$,

and if there exists a finite number (say, n) of periodic solutions $s = \phi_r(t), p = \psi_r(t), r = 1, 2, 3, \ldots, n$. in the $s - p$plane, then system (2) is uniformly persistent provided for each periodic solutions of period $T, \zeta_r = -d + \frac{1}{T}\int_0^T \left(\gamma\phi_r(t) - \frac{\beta\psi_r(t)}{1+\alpha\xi}\right)dt > 0, r = 1, 2, \ldots, n.$

Proof. Let $X = (s, i, p)$ be a point in the positive quadrant and $O(X)$ be orbit through X and Ω be the omega limit set of the orbit through X. Note that $\Omega(X)$ is bounded.

We claim that $E_T \notin \Omega(X)$. If $E_T \in \Omega(X)$ then by the Butler-McGehee lemma [31] there exist a point P in $\Omega(X) \bigcap W^s(E_T)$ where $W^s(E_T)$ denotes the stable manifold of E_T. Since $O(P)$ lies in $\Omega(X)$ and $W^s(E_T)$ is the $i - p$ plane, we conclude that $O(P)$ is unbounded, which is contrary to the boundedness of the system.

Next $E_A \notin \Omega(X)$, for otherwise, since E_A is a saddle point which follows from the conditions $\gamma > d$ and $e < \frac{\epsilon(1+c\xi)}{1+\alpha\xi+b}$, by the Butler-McGehee lemma [31] there exist a point P in $\Omega(X) \bigcap W^s(E_A)$. Now $W^s(E_A)$ is the s-axis implies that an unbounded orbit lies in $\Omega(X)$, a contradiction.

The condition $\frac{\epsilon(\hat{s}+c\xi)}{1+\alpha\xi+b\hat{s}} + \frac{\eta\hat{i}}{1+\alpha\xi} > e$ implies that E_P is unstable and therefore $E_P \notin \Omega(X)$.

Lastly, we show that no periodic orbits in the $s - p$ plane or $E_D \in \Omega(X)$. Let $r_i, i = 1, 2, \ldots, n$ denote the closed orbit of the periodic solution $(\phi_r(t), \psi_r(t))$ in $s - p$ plane such that r_i lies inside r_{i-1}. Let the Jacobian matrix $J_r(\phi_r(t), 0, \psi_r(t))$ corresponding to r_i is given by

$$J_r = \begin{pmatrix} F_{1s}(\phi_r(t), 0, \psi_r(t)) & F_{1i}(\phi_r(t), 0, \psi_r(t)) & F_{1p}(\phi_r(t), 0, \psi_r(t)) \\ 0 & F_{2i}(\phi_r(t), 0, \psi_r(t)) & 0 \\ F_{3s}(\phi_r(t), 0, \psi_r(t)) & F_{3i}(\phi_r(t), 0, \psi_r(t)) & F_{3p}(\phi_r(t), 0, \psi_r(t)) \end{pmatrix}. \tag{3}$$

Here $F_{2i} = -d + \gamma\phi_r(t) - \frac{\beta\psi_r(t)}{1+\alpha\xi}$. Computing the fundamental matrix of the linear periodic system

$$M' = J_r(t)M, M(0) = M_0,$$

we find that its Floquet multiplier in the i-direction is $e^{\zeta_r(t)}$. Then proceeding in an analogous manner like Kumar and Freedman [34], we conclude that no r_i lies on $\Omega(X)$. Thus, $\Omega(X)$ lies in the positive quadrant and system (2) is persistent. Finally,

since only the closed orbits and the equilibria from the omega limit set of the solutions on boundary of R_+^3 and the system (2) is dissipative. Now using a theorem of Butler et al. [32], we conclude that system (2) is uniformly persistent.

Note that the persistence condition of the system depends on infection rate, death rate of infected prey, death rate of predator, capturing rate of predator on infected prey and quality and quantity of alternative food.

5. Results and discussions

In this section, we perform numerical simulations with the help of parameter values taken from data of field works and experimental data (see, Table 1) which remains fixed for all numerical simulations. In our numerical simulations we have varied the infection rate γ, quality of alternative food α and quantity of alternative food ξ.
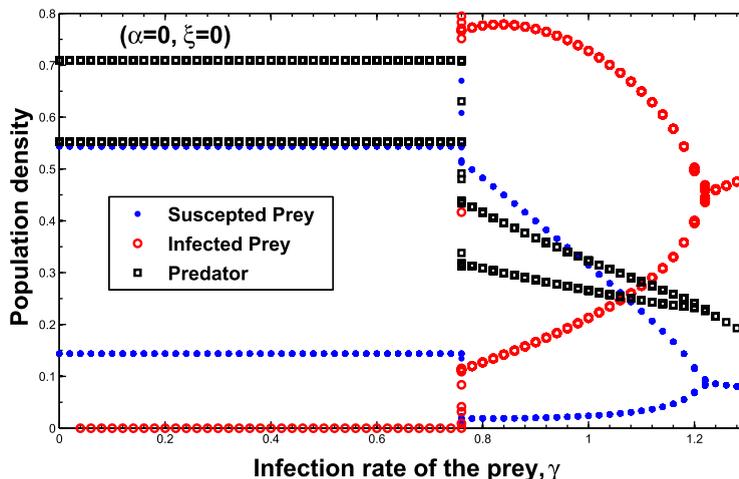
The bifurcation diagram of the system (2) with respect to infection rate of prey in the range $0 \leqslant \gamma \leqslant 1.34$ in absence of alternative food (i.e., $\alpha = 0, \xi = 0$) to predator is presented in Fig. 1. Fig. 1 shows that within $0 \leqslant \gamma \leqslant 0.76$, there is no infected prey species in the system and the susceptible prey and predator species have periodic behaviour. But, for $0.76 < \gamma \leqslant 1.21$, the susceptible prey, infected prey and predator species have limit cycle oscillations. All prey and predator species settle down to their respective steady states after $\gamma > 1.21$. Notice that the average density of the infected prey species becomes higher than the average density of susceptible prey for $\gamma > 0.76$. With the increase of infection rate susceptible prey becomes infected at higher rate and as a result the density of susceptible prey population decreases and the density of infected prey population increases.

The bifurcation diagram of the system (2) with respect to infection rate γ in the range $0 \leqslant \gamma \leqslant 1.74$ in presence of alternative food ($\alpha = 0.85, \xi = 0.3$) to predator is plotted in the Fig. 2. It is evident from Fig. 2 that the system becomes disease free within $0 \leqslant \gamma \leqslant 0.9$ in presence of alternative food. Therefore, supply of alternative food to predator makes the system disease free in $0.76 \leqslant \gamma \leqslant 0.9$ which is impossible in absence of alternative food. The infected prey population oscillates within $0.9 < \gamma < 1.6$ and it reaches steady state after $\gamma > 1.6$. The susceptible prey and predator species have periodic behaviour for $0 \leqslant \gamma \leqslant 1.6$ and finally they settle down to their respective steady state after $\gamma > 1.6$.

We plot dynamical behaviours of the infected prey population in absence of alternative food as well as in presence of alternative food in Fig. 3. Time evolution of infected prey is plotted in Fig. 3(a) in absence of alternative food taking $\gamma = 0.8$, on the other hand Fig. 3(b) represents time evolution of the infected prey species for same infection rate in presence of alternative food ($\alpha = 0.85, \xi = 0.6$). Fig. 3(c) depicts the time evolution of infected prey without alternative food with infection rate $\gamma = 1$ whereas Fig. 3(d) represents the disease free dynamics for ($\alpha = 0.85, \xi = 0.7$) with same infection rate.

Table 1
Ranges of parameters with their sources.

Parameter	Description	Range	Default value
a	Attack rate of susceptible prey	0.61–6.087 [35]	2.0
β	Attack rate of infected prey	0–0.5 [36]	0.12
b	Half saturation constant	2–6.2 [37]	3
ϵ	Conversion rate of susceptible prey	0.05–0.33 [38]	0.25
η	Conversion rate of infected prey	0.05–0.33 [38]	0.05
c	Conversion rate of additional food	$0 < c < 1$	0.3
d	Infected prey mortality rate	0.04–0.16 [39,40]	0.08
e	Predator mortality rate	0.04–0.16 [39,40]	0.04

Fig. 1. Bifurcation diagram of the system (2) with respect to infection rate of the prey in absence alternative food i.e., for $\alpha = 0$ and $\xi = 0$.
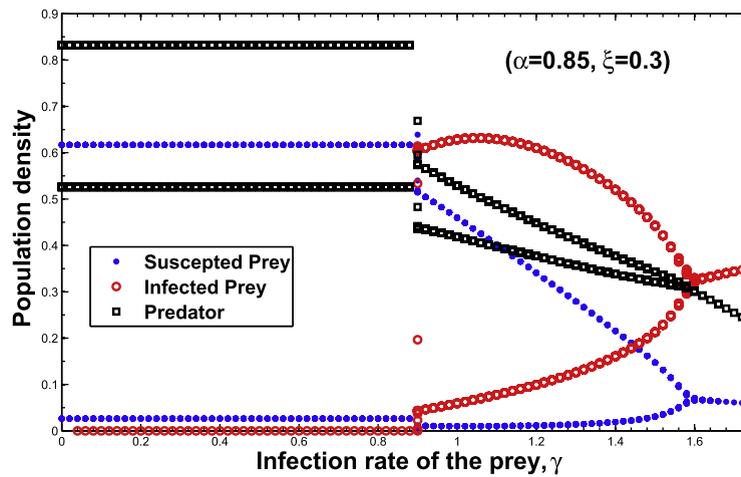
Fig. 2. Bifurcation diagram of the system (2) with respect to infection rate of the prey in presence of alternative food for $\alpha = 0.85$ and $\xi = 0.3$.
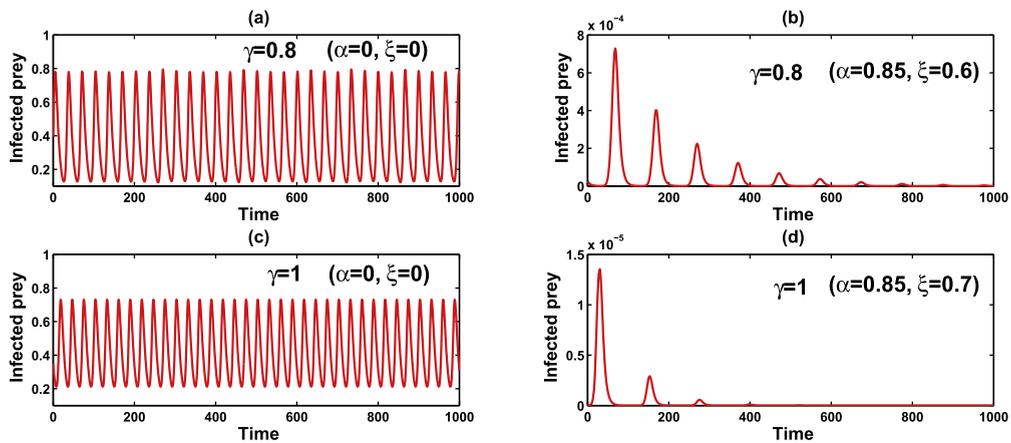

Fig. 3. Behaviour of infected prey. (a) oscillatory behaviour for $\gamma = 0.8$ with $\alpha = 0$ and $\xi = 0$, (b) extinction for $\gamma = 0.8$ with $\alpha = 0.85$ and $\xi = 0.6$, (c) oscillatory behaviour for $\gamma = 1$ with $\alpha = 0$ and $\xi = 0$, (d) extinction for $\gamma = 1$ with $\alpha = 0.85$ and $\xi = 0.7$.

From the Fig. 3, it is clear that the system becomes disease free under suitable supply of alternative food even if the infection rate is high which is impossible in absence of alternative food.

In Fig. 4, the $\alpha\xi$ plane is divided into infected and uninfected regions for fixed infection rate γ. We have determined the regions for $\gamma = 0.8$ (Fig. 4(a)) and for $\gamma = 1$ (Fig. 4(b)) considering other parameters values are as in Table 1. Fig. 4(a) is plotted in the $\alpha\xi$ parametric plane for $0 \leqslant \alpha \leqslant 2.5$ and $0 \leqslant \xi \leqslant 6.3$ with constant infection rate $\gamma = 0.8$, whereas Fig. 4(b) divides the $\alpha\xi$ plane into infected and uninfected regions for $0 \leqslant \alpha \leqslant 2.5$ and $0 \leqslant \xi \leqslant 7.7$ with constant infection rate $\gamma = 1$. We observe from Fig. 4 that it is impossible to make the system disease free in absence of alternative food for higher infection rate $\gamma \, (> 0.76)$ but disease free system can be achieved by suitable supply of alternative food. It is obvious that disease free solution exists for high quality of alternative food even if it is supplied in small quantity. On the other hand if alternative food is of low quality then high quantity of alternative food may not efficient to make the system disease free. Therefore high quality of alternative food supply is very efficient for disease control.

The $\gamma\xi$ and $\gamma\alpha$-planes are divided into infected and uninfected regions which are presented in Fig. 5(a) for $\alpha = 0.85$ and in Fig. 5(b) for $\xi = 0.62$, respectively. Fig. 5(a) is the parametric plane $\gamma\xi$ for $0 \leqslant \gamma \leqslant 2.5$ and $0 \leqslant \xi \leqslant 1$ fixing $\alpha = 0.85$. The Fig. 5(a) shows that the system becomes disease free for small quantity of alternative food for lower infection rate $\gamma(< 0.5)$. But for higher infection rate, the supply level ξ increases depending on the increase of infection rate. There exists a critical infection rate above which alternative food may not be useful to make the system disease free. It is clear from Fig. 5(b) that disease free state can be reached for higher infection rate $(\gamma > 0.76)$ supplying high quality of alternative food. It is evident from Fig. 5(b) that for lower infection rate quality of alternative is not so important for obtaining disease free system.
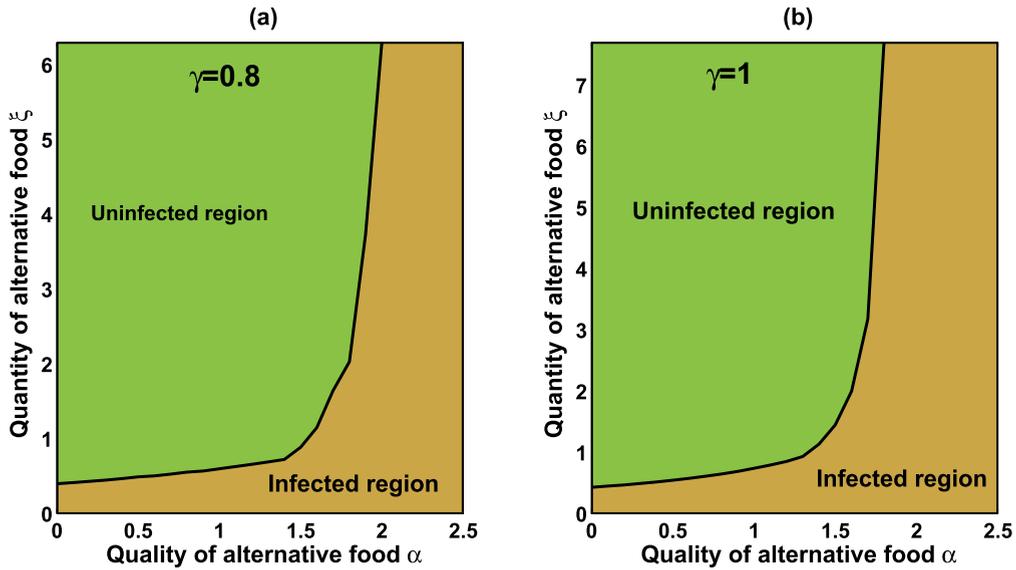
Fig. 4. Graph of α versus ξ for fixed (a) γ = 0.8 and (b) γ = 1. Parameters are as in Table 1. The αξ-plane is divided into infected and uninfected regions. This figure indicates that for high quality of alternative food, small quantity of it is sufficient to make the system disease free.
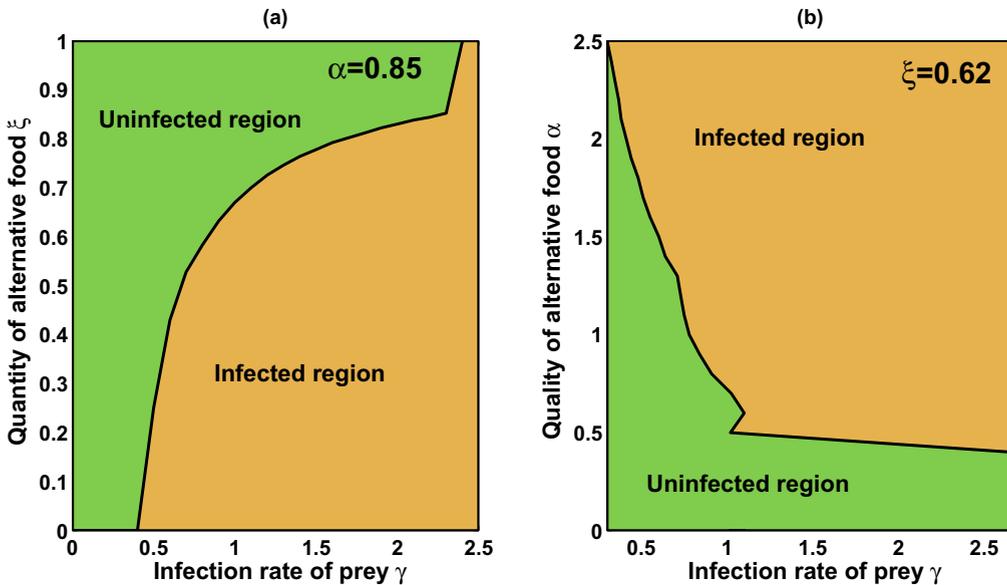

Fig. 5. Graph of (a) γ versus ξ for fixed α = 0.85 and (b) γ versus α for fixed ξ = 0.62. Parameters are as in Table 1. The γξ and γα-planes are divided into infected and uninfected regions. The figure (a) gives the minimum values of ξ for different infection rate γ and (b) depicts the values of α for different infection rate γ to make the system disease free.

6. Conclusions

We examine some nontrivial consequences of alternative food to predator in a infected predator–prey model. We have studied stability and persistence criterion of the system in presence of alternative food. One parameter bifurcation analysis is done with respect to infection rate using experimental and field data. Numerical simulation results predict that the system becomes disease free for lower infection rate ($0 \leqslant \gamma \leqslant 0.76$) in absence of alternative food (Fig. 1). This is happening because infected prey is weaker than susceptible prey and is easily captured by predator. This will reduce the number of infected prey and ultimately the system may becomes disease free without alternative food at certain stage for lower infection rate. However, for higher infection rate ($\gamma > 0.76$), disease free system cannot be obtained without alternative food

(Fig. 3). Whereas for higher infection rate disease free state may be reached by supplying suitable alternative food (Figs. 4 and 5). It is evident from our study that high quality of alternative food ($\alpha < 1$) has the capability of making the system disease free for higher infection rate. Quantity of alternative food supply increases with the increase of infection rate for fixed quality to obtain a disease free system. Possible reason behind it is that with the supply of high quality or high quantity of alternative food, the predator catches the alternative food as well as infected prey at higher rate compare to susceptible prey. As a result, the growth rate of predator species will increase with the supply of high quality or high quantity of alternative food and it captures the infected prey population at faster rate than susceptible prey. Due to this, infected prey population become very very small at certain stage and consequently infection cannot spread at that stage and the system becomes disease free. Notice that there is a critical infection rate above which the alternative food may not be useful to make the system disease free. Results presented in the present paper provided a useful platform to understand the role of alternative food as disease controller in a diseased food chain model. A possible interesting aspects for future developments are to study the role of alternative food as a disease controller in different types of disease induced food chain models and also the study the effects of supplying alternative food to a network of infected food chain models. This new non-chemical method of disease control will be very useful for biological conservation of prey species in real world biological systems.

Acknowledgements

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