International Scholarly Research Network
ISRN Applied Mathematics
Volume 2011, Article ID 807486, 16 pages
doi:10.5402/2011/807486

Research Article

A Mathematical Study of a Predator-Prey Dynamics with Disease in Predator

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Received 24 March 2011; Accepted 19 April 2011

Academic Editor: M. Langthjem

We consider a predator-prey model where parasitic infection is spread in only predator population. We work out the local stability analysis of equilibrium point by the help of basic reproduction numbers. We also analyze the community structure of model system by the help of ecological as well as disease basic reproduction numbers. We derive Hopf bifurcation condition and permanence and impermanence of model system. We perform a numerical experiment and observe that parasitic infection in predator population stabilizes predator-prey oscillations.

1. Introduction

The effect of disease in ecological system is an important issue from mathematical as well as ecological point of view. So, in recent time ecologists and researchers are paying more and more attention to the development of important tool along with experimental ecology and describe how ecological species are infected. However, the first breakthrough in modern mathematical ecology was done by Lotka and Volterra for a predator-prey competing species. On the other hand, most models for the transmission of infectious diseases originated from the classic work of Kermack and Mc Kendrick [1]. After these pioneering works in two different fields, lots of research works have been done both in theoretical ecology and epidemiology. Anderson and May [2] 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 authors [3–7] proposed and studied different predator-prey models in presence of disease.

Microparasites may be thought of as those parasites which have direct reproduction—usually at very high rates—within the host [8]. They tend to be characterized by small size and a short generation time. Hosts that recover from infection usually acquire immunity

against reinfection for some time, and often for life. Although there are important exceptions, the duration of infection is typically short relative to the expected life span of the host. This feature, combined with acquired immunity, means that for individual hosts microparasitic infection is typically of a transient nature. Most viral and bacterial parasites, and many protozoan and fungal parasites, fall broadly into the microparasitic category [9]. In the natural world, no species can survive alone. While species spreads the disease, also competes with the other species for space or food, or is predated by other species. Predator-prey relationship can be important in regulating the number of prey and predators. For example, when a bounty was placed on natural predators such as cougars, wolves, and coyotes in the Kaibab Plateau in Arizona, the deer population increased beyond the food supply, and then over half of the deer died of starvation in 1923–1925 [10]. But predator control may be very important and essential in many situations. For example, one may consider the interaction between plant (prey) and pest (predator). Many pest species caused a fearful toll for human life by destroying plants like tea, potato, and maize. However, an infection in the predator (pest) may control the predator (pest) and lead the prey (plant) population to increase. This guess arises from the promising results of different experimental findings on plant-pest systems. For example, the pest *Cydia pomonella* in orchards [11] is controlled when granulosis virus is sprayed against *C. pomonella*. Successful pest control using virus is also reported by Caballero et al. [12] (by using granulosis virus against the pest *Agrotis segetum* in maize), Laarif et al. [13] (by using granulovirus (PoGV) against the pest *Phthorimaea operculella* in potato). The literature abounds with such evidences. In the last few decades, mathematical models have become extremely important tools in understanding and analyzing the spread and control of infectious diseases. On the other hand, a number of sophisticated predator-prey models are introduced and extensively studied in ecological literature. But a little attention had been paid on the effect of transmissible diseases when two or more species are in an ecological relationship between them. To the best of our knowledge, the influence of predation on epidemics has not yet been studied considerably, except the works of Anderson and May, [14] Hadeler and Freedman, [15] Hochberg [16], Venturino, [6, 17] Chattopadhyay and Arino [3], Han et al. [18], Xiao and Chen [7], Hethcote et al. [5], Greenhalgh and Haque [19], and Haque and Venturino [20, 21]. Most of these works have dealt with predator-prey models with disease in the prey (except Venturino [6], Haque and Venturino [20, 21]). Recently Auger et al. have studied the effects of a disease affecting a predator on the dynamics of a predator-prey system, and they have observed two possible asymptotic behaviours: either the predator population dies out and the prey tends to its carrying capacity, or the predator and prey coexist. In this latter case, the predator population tends either to a disease-free or to a disease-endemic state. Russell et al. have created a model of long-lived aged-structured shared prey and explored the nonequilibrium dynamics of the system and concluded that the superpredator can impact all prey life stages (adult survival and reproductive success) where the smaller mesopredator can only impact early life stages (reproductive success). They have also tested with data from a closed oceanic island system where eradication of introduced intraguild predators is possible for conservation of threatened birds. But the study of the dynamics of a predator-prey system with an infected predator has a great importance so long as the question of predator control is concerned. To the best of our knowledge, mathematical epidemiology almost remained silent in this issue.

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 [14, 21, 22] and indirect life cycles [23, 24]. Macroparasitic models generally have a tendency to unstable dynamics, because they

consider the parasite burden in the host in an additional equation [23, 25]. Here we show that the scenario of destabilization does not always hold true. The effect of disease introduction can be quite the opposite, namely, to stabilize oscillatory predator-prey dynamics. We analyze the community structure of our model system with the help of ecological and disease basic reproduction numbers.

The paper is organized as follows. In the Section 2, we outline the mathematical model with some basic assumption. In Section 3 we study the stability of the equilibrium points and Hopf bifurcation and the permanence and impermanence of the system in Section 4. We give numerical results and discussion in Section 5. The paper ends with a conclusion.

2. Mathematical Model

In formulation of mathematical model we assume the following basic assumptions.

(1) Let X denote the population density of the prey, Y the population density of the susceptible predator, and Z the density of the infected predator, respectively, in time T.

(2) We assume that in the absence of the predators the prey population density grows according to a logistic curve with carrying capacity K ($K > 0$) and with an intrinsic growth rate constant r ($r > 0$).

(3) 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_1 following the mass action law.

From the above assumptions we can write the following set of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dX}{dT} &= rX\left(1 - \frac{X}{k}\right) - \frac{c_1 X(Y + fZ)}{a_1 + X},\\
\frac{dY}{dT} &= \frac{m_1 X(Y + fZ)}{a_1 + X} - d_1 Y - \lambda_1 YZ,\\
\frac{dZ}{dT} &= \lambda_1 YZ - (d_1 + \alpha_1)Z.
\end{aligned} \tag{2.1}$$

Here c_1 is the predation rate of susceptible predator, $c_1 f$ is the predation rate of infected predator, λ_1 is the infection rate, and a_1 is the half saturation constant. The infected predator is less able to hunt or to capture a prey than a susceptible predator, that is, the parasite has negative effect on the predation rate. Since microparasites affect the internal mechanisms of their hosts, therefore, the net gain from the consumption of preys must be different for susceptible and infected predators. From this viewpoint, we have chosen the different predation rates and conversion rates for susceptible and infected predators. The constant m_1 is the conversion factor for the susceptible predator, and $m_1 f$ is the conversion factor for the infected predator. The constant d_1 is the parasite-independent mortality rate of predator. α_1 denotes additional mortality rate of predator due to infection.

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

$$x = \frac{X}{K}, \qquad y = \frac{Y}{K}, \qquad z = \frac{Z}{K}, \qquad t = rT. \tag{2.2}$$

Then the system (2.1) takes the form

$$\begin{aligned} \frac{dx}{dt} &= x(1-x) - \frac{ax(y+z)}{1+bx}, \\ \frac{dy}{dt} &= \frac{cx(y+fz)}{1+bx} - dy - \beta yz, \\ \frac{dz}{dt} &= \beta yz - ez, \end{aligned} \tag{2.3}$$

where

$$a = \frac{c_1 K}{r a_1}, \qquad b = \frac{K}{a_1}, \qquad c = \frac{m_1 K}{r a_1}, \qquad d = \frac{d_1}{r}, \qquad \beta = \frac{\lambda_1}{r}, \qquad e = \frac{d_1 + \alpha_1}{r}. \tag{2.4}$$

System (2.3) has to be analyzed with the following initial conditions:

$$x(0) > 0, \qquad y(0) > 0, \qquad z(0) > 0. \tag{2.5}$$

3. Qualitative Analysis of Model System

3.1. Equilibria and Their Local Stability

The system has four equilibrium points. The trivial equilibrium point $E_0(0,0,0)$ and the axial equilibrium point $E_1(1,0,0)$ exist for all parametric values. Disease-free equilibrium point is $E_2(\overline{x}, \overline{y}, 0)$, where

$$\overline{x} = \frac{d}{c - bd}, \qquad \overline{y} = \frac{c(c - bd - d)}{a(c - bd)}. \tag{3.1}$$

The existence conditions of disease-free equilibrium point is $c - bd - d > 0$, that is, $R_{01} = (1/d)(c/(1+b)) > 1$.

The interior equilibrium point is given by $E^*(x^*, y^*, z^*)$, where x^* is the positive root of the equation

$$Q_1 x^3 + Q_2 x^2 + Q_3 x + Q_4 = 0, \tag{3.2}$$

where

$$
\begin{aligned}
Q_1 &= b\beta(cf - be),\\
Q_2 &= \beta(cf - be)(1 - b) - ebb\beta,\\
Q_3 &= e\beta(b - 1) - ae(c - bd) - (cf - be)(\beta - ea),\\
Q_4 &= aed + e(\beta - ea),\\
y^* &= \frac{e}{\beta},\\
z^* &= \frac{\beta(1 - x^*)(1 + bx^*) - ea}{a\beta}.
\end{aligned}
\tag{3.3}
$$

The Jacobian matrix J of the system (2.3) at any arbitrary point (x, y, z) is given by

$$
\begin{bmatrix}
1 - 2x - \dfrac{a(y + z)}{(1 + bx)^2} & \dfrac{-ax}{1 + bx} & \dfrac{-ax}{1 + bx}\\
\dfrac{c(y + fz)}{(1 + bx)^2} & \dfrac{cx}{1 + bx} - d - \beta z & \dfrac{cfx}{1 + bx} - \beta y\\
0 & \beta z & \beta y - e
\end{bmatrix}.
\tag{3.4}
$$

Theorem 3.1. *The trivial equilibrium point E_0 is always unstable. The axial equilibrium point E_1 is locally stable if $R_{01} < 1$, where $R_{01} = (1/d)(c/(1 + b))$. The disease-free equilibrium point E_2 is locally asymptotically stable if $(1 + b\overline{x})^2 > ab\overline{y}$ and $R_{02} < 1$ where $R_{02} = \beta\overline{y}/e$.*

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

The Jacobian matrix at axial equilibrium point E_1 is given by

$$
J_1 = \begin{bmatrix}
-1 & 0 & 0\\
0 & \dfrac{c}{1 + b} - d & \dfrac{cf}{1 + b}\\
0 & 0 & -e
\end{bmatrix}.
\tag{3.5}
$$

The characteristic roots of the Jacobian matrix J_1 are $c/(1 + b) - d$ and $-e$.

Hence E_1 is stable if $c/(1 + b) - d < 0$ which implies $R_{01} < 1$ and unstable if $R_{01} > 1$, where $R_{01} = (1/d)(c/(1 + b))$.

The Jacobian matrix at disease-free equilibrium point E_2 is given by

$$
J_2 = \begin{bmatrix}
-\overline{x} + \dfrac{a\overline{x}\overline{y}b}{(1 + b\overline{x})^2} & \dfrac{-a\overline{x}}{1 + b\overline{x}} & \dfrac{-a\overline{x}}{1 + b\overline{x}}\\
\dfrac{c\overline{y}}{(1 + b\overline{x})^2} & 0 & \dfrac{cf\overline{x}}{1 + b\overline{x}} - \beta\overline{y}\\
0 & 0 & \beta\overline{y} - e
\end{bmatrix}.
\tag{3.6}
$$

The characteristic roots of the Jacobian matrix J_2 are $\beta\overline{y}-e$, and the roots of the equation

$$\lambda^2 + \overline{x}\left(1 - \frac{a\overline{y}b}{(1+b\overline{x})^2}\right)\lambda + \frac{ca\overline{x}\overline{y}}{(1+b\overline{x})^3} = 0. \tag{3.7}$$

It is clear that E_2 is stable if $1 - a\overline{y}b/(1+b\overline{x})^2 > 0$, that is, $(1+b\overline{x})^2 > a\overline{y}b$ and $\beta\overline{y} - e < 0$, that is, $R_{02} = \beta\overline{y}/e < 1$ and unstable for $R_{02} = \beta\overline{y}/e > 1$. □

3.2. *Biological Significance of Threshold Parameters and Community Structure*

We discuss here the biological significance of two threshold parameters obtained from stability analysis of equilibria points, each of which has clear and distinct biological meaning. We also discuss the community structure of model system with the help of these ecological and disease threshold parameters. We first define the ecological threshold parameter by

$$R_{01} = \frac{1}{d}\left(\frac{c}{1+b}\right) \tag{3.8}$$

which determines the local stability of $E_1(1,0,0)$. Here $c/(1+b)$ is the birth rate of predator at E_1, and $1/d$ is the mean lifespan of predator. Subsequently their product gives the mean number of newborn predators by a predator which can be interpreted as the ecological basic reproduction number at E_1. We note that this term, first formulated and explained by Pielou [26], is the average number of prey converted to predator biomass in a course of the predator's life span [5]. Here R_{01} is denoted by ecological basic reproduction numbers according to Hsiesh and Hsiao [27]. $R_{01} < 1$ implies that the predators will become extinct and consequently there will be no chance of infection in predator population. Hence this condition results in E_1 being locally asymptotically stable.

We define disease threshold parameter by

$$R_{02} = \frac{\beta\overline{y}}{e} \tag{3.9}$$

which necessarily determines the local stability of disease-free equilibrium point $E_2(\overline{x},\overline{y},0)$. Here $\beta\overline{y}$ is the infection rate of a new infective predator appearing in a totally susceptible predator population, and $1/e$ is the duration of infectivity of an infective predator. Their product, that is, R_{02}, gives the disease basic reproduction number of system. Here R_{02} is denoted by disease basic reproduction numbers according to Hsiesh and Hsiao [27]. It 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 [28]. $R_{02} < 1$ implies the infected predators will become extinct and consequently disease will be eradicated from the system. Actually $R_{02} < 1$ is the necessary condition for local stability of E_2. Here we have observed that disease-free equilibrium (DFE) is stable if $R_{02} < 1$ and unstable if $R_{02} > 1$. So entire community composition, that is, the persistence of (i) prey alone, (ii) prey and predator, and (iii) prey, predator, and disease, can be predicted by biologically meaningful reproduction numbers.

3.3. Local Stability of Interior Equilibrium Point and Hopf Bifurcation

Theorem 3.2. *The interior point $E^*(x^*, y^*, z^*)$ of the system (2.1) exists, then E^* is locally asymptotically stable if the following conditions hold:*

$$(1 + bx^*)^2 > ay^*b, \qquad \frac{cx^*}{1 + bx^*} < \beta z^* + d. \tag{3.10}$$

Proof. The Jacobian matrix at the interior point $E^*(x^*, y^*, z^*)$ is

$$V = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{bmatrix}, \tag{3.11}$$

where

$$\begin{gathered} A_{11} = -x^* + \frac{abx^*(y^* + z^*)}{(1 + bx^*)^2}, \qquad A_{12} = \frac{-ax^*}{1 + bx^*}, \qquad A_{13} = \frac{-ax^*}{1 + bx^*}, \\ A_{21} = \frac{c(y^* + fz^*)}{(1 + bx^*)^2}, \qquad A_{22} = \frac{cx^*}{1 + bx^*} - \beta z^* - d, \qquad A_{23} = \frac{cfx^*}{1 + bx^*} - \beta y^*, \\ A_{31} = 0, \qquad A_{32} = \beta z^*, \qquad A_{33} = 0. \end{gathered} \tag{3.12}$$

The characteristic equation of the Jacobian matrix is given by

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0, \tag{3.13}$$

where

$$\begin{gathered} \sigma_1 = -(A_{11} + A_{22}), \\ \sigma_2 = A_{11}A_{22} - A_{23}A_{32} - A_{12}A_{21}, \\ \sigma_3 = A_{23}A_{32}A_{11} - A_{13}A_{21}A_{32}, \\ \sigma_1\sigma_2 - \sigma_3 = -A_{11}^2A_{22} + A_{11}A_{12}A_{21} - A_{11}A_{22}^2 + A22A_{23}A_{32} + A_{22}A_{12}A_{21} + A_{13}A_{21}A_{32}. \end{gathered} \tag{3.14}$$

□

The sufficient conditions for $\sigma_1 > 0$, $\sigma_3 > 0$, and $\sigma_1\sigma_2 - \sigma_3 > 0$ are as follows:

$$A_{11} \le 0, \qquad A_{22} \le 0 \tag{3.15}$$

which implies the conditions

$$(1 + bx^*)^2 > ay^*b, \qquad \frac{cx^*}{1 + bx^*} < \beta z^* + d. \tag{3.16}$$

Thus, if the condition stated in the theorem holds, then all the Routh-Hurwitz criteria (i) $\sigma_1 > 0$, (ii) $\sigma_1\sigma_2 - \sigma_3 > 0$, (iii) $\sigma_3 > 0$ are satisfied, and the system (2.3) is locally asymptotically stable around the positive equilibrium point.

Theorem 3.3. *The rate of infection β crosses a critical value β^*, and the system enters into Hopfbifurcation around the positive equilibrium E^* if the following conditions hold:*

(i) $\sigma_1(\beta^*) > 0$;

(ii) $\sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0$;

(iii) $[\sigma_1(\beta^*)\sigma_2(\beta^*)]' < \sigma_3'(\beta^*)$.

Proof. We assume that the steady state E^* is asymptotically stable; we would like to know if E^* will lose its stability when one of the parameters changes. We choose β, the force of infection, as the bifurcation parameter; we can see that if there exists a critical value β^* such that

$$\sigma_1(\beta^*) > 0, \quad \sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0, \quad [\sigma_1(\beta^*)\sigma_2(\beta^*)]' < \sigma_3'(\beta^*), \tag{3.17}$$

for the Hopfbifurcation to occur at $\beta = \beta^*$, the characteristic equation must be of the form

$$\left(\lambda^2(\beta^*) + \sigma_2(\beta^*)\right)(\lambda(\beta^*) + \sigma_1(\beta^*)) = 0, \tag{3.18}$$

which has three roots $\lambda_1(\beta^*) = i\sqrt{\sigma_2(\beta^*)}$, $\lambda_2 = -i\sqrt{\sigma_2(\beta^*)}$, $\lambda_3 = -\sigma_1(\beta^*) < 0$.

To see if Hopf bifurcation occurs at $\beta = \beta^*$, we need to verify the transversality condition

$$\left[\frac{d\,\mathrm{Re}(\lambda(\beta))}{d\beta}\right]_{\beta=\beta^*} \neq 0. \tag{3.19}$$

For all β, the roots are in general of the form

$$\begin{aligned} \lambda_1(\beta) &= \mu(\beta) + i\nu(\beta), \\ \lambda_2(\beta) &= \mu(\beta) - i\nu(\beta), \\ \lambda_3(\beta) &= -\sigma_1(\beta). \end{aligned} \tag{3.20}$$

Now, we will verify the transversality condition

$$\left[\frac{d\,\mathrm{Re}(\lambda_j(\beta))}{d\beta}\right]_{\beta=\beta^*} \neq 0, \quad j = 1, 2. \tag{3.21}$$

Substituting $\lambda_j(\beta) = \mu(\beta) \pm i\nu(\beta)$, into (3.18) and calculating the derivative, we have

$$\begin{aligned} K(\beta)\mu'(\beta) - L(\beta)\nu'(\beta) + M(\beta) &= 0, \\ K(\beta)\mu'(\beta) + L(\beta)\nu'(\beta) + N(\beta) &= 0, \end{aligned} \tag{3.22}$$

where

$$K(\beta) = 3\mu^2(\beta) + 2\sigma_1(\beta)\mu(\beta) + \sigma_2(\beta) - 3\nu^2(\beta),$$
$$L(\beta) = 6\mu(\beta)\nu(\beta) + 2\sigma_1(\beta)\nu(\beta),$$
$$M(\beta) = \mu^2(\beta)\sigma_1'(\beta) + \sigma_2'(\beta)\mu(\beta) + \sigma_3'(\beta) - \sigma_1'(\beta)\nu^2(\beta), \tag{3.23}$$
$$N(\beta) = 2\mu(\beta)\nu(\beta)\sigma_1'(\beta) + \sigma_2'(\beta)\nu(\beta).$$

Noticing that $\mu(\beta^*) = 0$, $\nu(\beta^*) = \sqrt{\sigma_2(\beta^*)}$, we have

$$K(\beta) = -2\sigma_2(\beta^*), \qquad L(\beta^*) = 2\sigma_1(\beta^*)\sqrt{\sigma_2(\beta^*)},$$
$$M(\lambda^*) = \sigma_3'(\beta^*) - \sigma_1'(\beta^*)\sigma_2(\beta^*), \qquad N(\beta^*) = \sigma_2'(\beta^*)\sqrt{\sigma_2(\beta^*)}. \tag{3.24}$$

Solving for $\mu'(\beta^*)$ from system (3.22) we have

$$\left[\frac{dRe(\lambda_j(\beta))}{d\beta}\right]_{\beta=\beta^*} = \mu'(\beta)_{\beta=\beta^*} = -\frac{L(\beta^*)N(\beta^*) + K(\beta^*)M(\beta^*)}{K^2(\beta^*) + L^2(\beta^*)}$$
$$= \frac{\sigma_3'(\beta^*) - \sigma_1'(\beta^*)\sigma_2(\beta^*) - \sigma_1(\beta^*)\sigma_2'(\beta^*)}{\sigma_1^2(\beta^*) + \sigma_2(\beta^*)} > 0, \tag{3.25}$$

if $[\sigma_1(\beta^*)\sigma_2(\beta^*)]' < \sigma_3'(\beta^*)$ and

$$\lambda_3(\beta^*) = -\sigma_1(\beta^*) < 0. \tag{3.26}$$

Thus the transversality conditions hold, and hence Hopf bifurcation occurs at $\beta = \beta^*$. Hence the theorem. □

Remark 3.4. If there exists a critical value of force infection β^* such that $\sigma_1(\beta^*) > 0$, $\sigma_1(\beta^*)\sigma_2(\beta^*) - \sigma_3(\beta^*) = 0$, and $[\sigma_1(\beta^*)\sigma_2(\beta^*)]' < \sigma_3'(\beta^*)$, then when $\beta > \beta^*$, the steady state E^* is stable; when $\beta = \beta^*$, E^* loses its stability and the Hopf bifurcation occurs at E^*, and when $\beta < \beta^*$, E^* becomes unstable and a family of periodic solutions bifurcates from E^*.

4. Permanence and Impermanence

From biological point of view, permanence of a system means the survival of all populations of the system in future time. Mathematically, permanence of a system means that strictly positive solutions do not have omega limit points on the boundary of the nonnegative cone.

Theorem 4.1. *If the condition $R_{01} > 1$ is satisfied and further if there exists a finite number of periodic solutions $x = \phi_r(t)$, $y = \psi_r(t)$, $r = 1, 2, \ldots, n$, in the $x - y$ plane, then system* (2.3) *is uniformly persistent provided for each periodic solutions of period T,*

$$\eta_r = -e + \frac{1}{T}\int_0^T \beta\psi_r dt > 0, \tag{4.1}$$

$r = 1, 2, \ldots, n$.

Proof. Let p be a point in the positive cone, $o(p)$ orbit through p, and Ω the omega limit set of the orbit through p. Note that $\Omega(p)$ is bounded.

We claim that $E_0 \notin \Omega(p)$. If $E_0 \in \Omega(p)$ then by the Butler-McGehee lemma [29] there exists a point q in $\Omega(p) \bigcap W^s(E_0)$ where $W^s(E_0)$ denotes the stable manifold of E_0. Since $o(q)$ lies in $\Omega(p)$ and $W^s(E_0)$ is the $y - z$ plane, we conclude that $o(q)$ is unbounded, which is a contradiction.

Next $E_1 \notin \Omega(x)$; for otherwise, since E_1 is a saddle point which follows from the condition $R_{01} > 1$ by the Butler-McGehee lemma [29] there exists a point q in $\Omega(p) \bigcap W^s(E_1)$. Now $W^s(E_1)$ is the $x - z$ plane which implies that an unbounded orbit lies in $\Omega(p)$, a contradiction.

Lastly we show that no periodic orbit in the $x - y$ plane or $E_2 \in \Omega(p)$. Let r_i $i = 1, 2, \ldots, n$ denote the closed orbit of the periodic solution $(\phi_r(t), \psi_r(t))$ in $x - y$ plane such that r_i lies inside r_{i-1}. Let, the Jacobian matrix J given in (3.4) corresponding to r_i be denoted by $J_r(\phi_r(t), \psi_r(t), 0)$. Computing the fundamental matrix of the linear periodic system,

$$X' = J_r(t)X, \qquad X(0) = I. \tag{4.2}$$

We find that its Floquet multiplier in the z direction is $e^{\eta_r T}$. Then proceeding in an analogous manner like Kumar and Freedman [30], we conclude that no r_i lies on $\Omega(x)$. Thus, $\Omega(x)$ lies in the positive cone and system (2.1) is persistent. Finally, only the closed orbits and the equilibria from the omega limit set of the solutions are on the boundary of R^3_+, and system (2.3) is dissipative. Now using a theorem of Butler et al. [29], we conclude that system (2.3) is uniformly persistent. □

Theorem 4.2. *If the conditions $R_{01} > 1$ and $R_{02} > 1$ are satisfied and if there exists no limit cycle in the $x - y$ plane, then system* (2.3) *is uniformly persistent.*

Proof. Proof is obvious and hence omitted. □

Before obtaining the conditions for impermanence of system (2.3), we briefly define the impermanence of a system. Let $x = (x_1, x_2, x_3)$ be the population vector, let $D = \{x : x_1, x_2, x_3 > 0\}$, and ∂D is the boundary of D. $\mu(\cdot, \cdot)$ is the distance in R^3_+.

Let us consider the system of equations

$$\dot{x} = f_i(x), \qquad i = 1, 2, 3, \tag{4.3}$$

where $f_i : \mathrm{R}^3_+ \to R$ and $f_i \in C^1$.

The semiorbit γ^+ is defined by the set $\{x(t) : t > 0\}$, where $x(t)$ is the solution with initial value $x(0) = x_0$.

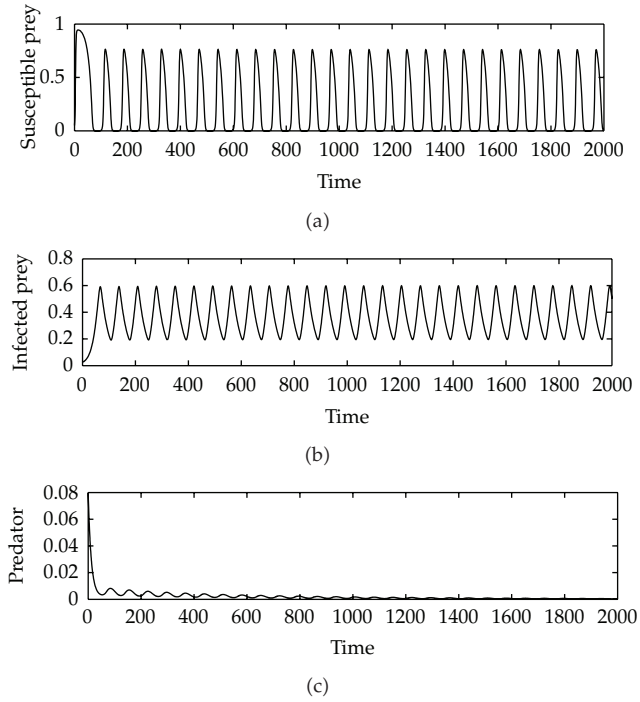

Figure 1: The figure depicts the extinction of infected predator and oscillation of other two species for $\beta = 0.24$ and $a = 2.8$, $b = 2.8$, $c = 0.12 \times a$, $d = 0.03$, $e = 0.09$, $f = 0.01$.

The above system is said to be impermanent [31] if and only if there is an $x \in D$ such that $\lim_{t\to\infty}\mu(x(t), \partial D) = 0$. Thus a community is impermanent if there is at least one semiorbit which tends to the boundary.

Theorem 4.3. *If the condition $R_{01} < 1$ or $R_{02} < 1$ holds, then the system* (2.1) *is impermanent.*

Proof. The given condition $R_{01} < 1$ implies that E_1 is a stable equilibrium point on the boundary. Similarly $R_{02} < 1$ implies that E_1 is a saturated equilibrium point on the boundary. Hence, there exists at least one orbit in the interior that converges to the boundary [32]. Consequently the system (2.1) is impermanent [31]. □

5. Numerical Results and Discussion

We know that the infectious disease plays important roles in the dynamics of a predator-prey system with infection in prey [5, 33]. But in our model system infection in predator (β) plays an important role since the inclusion of disease in predator population in our model is vital modification of most of the earlier models. So, we have focused our study in observing the role of infection rate upon predator-prey dynamics. We have taken a set of hypothetical parameter values $a = 2.8$, $b = 2.8$, $c = 0.12 \times a$, $d = 0.03$, $e = 0.09$, $f = 0.01$. We will now observe the dynamical behavior of the system (2.3) for the above set of parameter values. We observe from Figure 1 that disease in predator population cannot propagate for $\beta = 0.24$ and

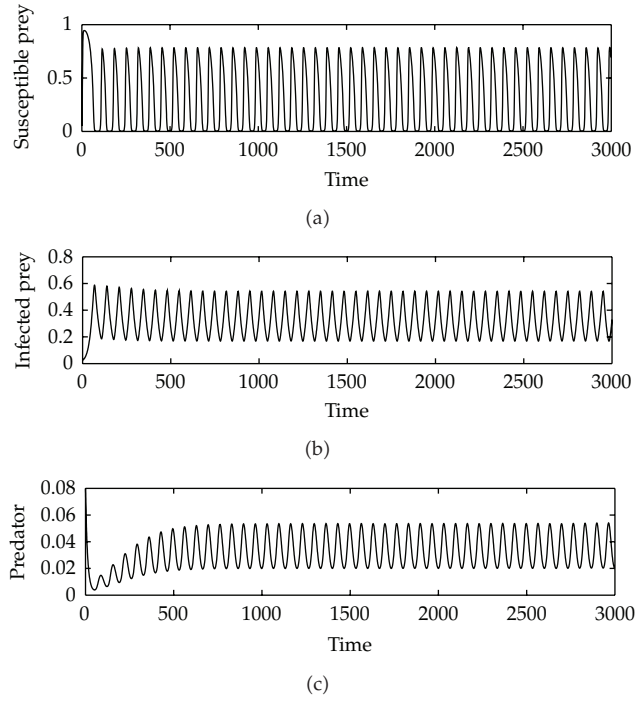

Figure 2: The figure depicts that all three species coexist in oscillating position (limit cycle) for $\beta = 0.27$ and other parameter values given in Figure 1.

predator and prey species coexist in oscillatory position. If we increase the infection rate β, we observe that all three species coexist in oscillatory position and this observation is clear from Figure 2. Figure 3 illustrates that oscillations settle down into stable situation and all three species persist in stable position for $\beta = 0.32$. A clear dynamics of predator-prey system for variation of infection rate β, we draw a bifurcation diagram. From Figure 4 it is clear that oscillatory coexistence of all three species is found for $0.25 \leq \beta \leq 0.3$ and all species will be stable for $\beta > 0.3$. In our proposed model we get an interesting result that disease in predator population has stabilizing effect on predator-prey oscillation. Nonlinear interactions between predators and prey are wellknown to generate endogenous oscillations. We have shown, to our knowledge, that these fluctuations can be stabilized by an infectious disease spreading within the predator population. This challenges the current view of destabilizing disease impacts [14, 15, 21–25], which also similarly exists for disease infecting prey populations [14, 20, 34, 35]. Moreover, our results appear to contradict the observation of de Castro and Bolker [36] that parasite-induced cycles are more likely to occur in larger communities. 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 multispecies 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) [37–39].

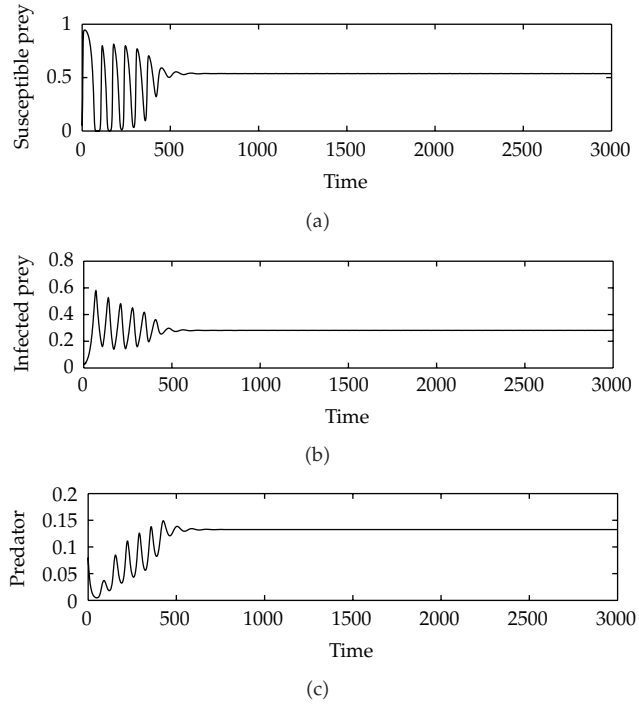

Figure 3: The figure depicts that all three species coexist in stable position for $\beta = 0.32$ and other parameter values given in Figure 1.

We now explain the stability mechanism in our model system. The effect of the disease is only to increase predator mortality, which decreases predator population size and the predation pressure on the prey. This, in turn, increases prey population size and the density dependence felt by the prey population, which is a stabilizing factor. Infection thus indirectly couples predator mortality with prey population size. A similar inhibition of the predator population by high densities of the prey occurs in the presence of toxic prey species [40].

We also analyze the community structure of our model system with the help of ecological and disease basic reproduction number. It 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. We use reproduction numbers as helpful tools in determining the persistence (if they are larger than one) or extinction (if they are smaller than one) of a species. This allows 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 ecoepidemiological models [5, 7, 15, 18].

6. Conclusion

In the present paper we consider a predator-prey system where predator is infected by parasitic attack. The main objective of this paper is to observe the effect of parasitic

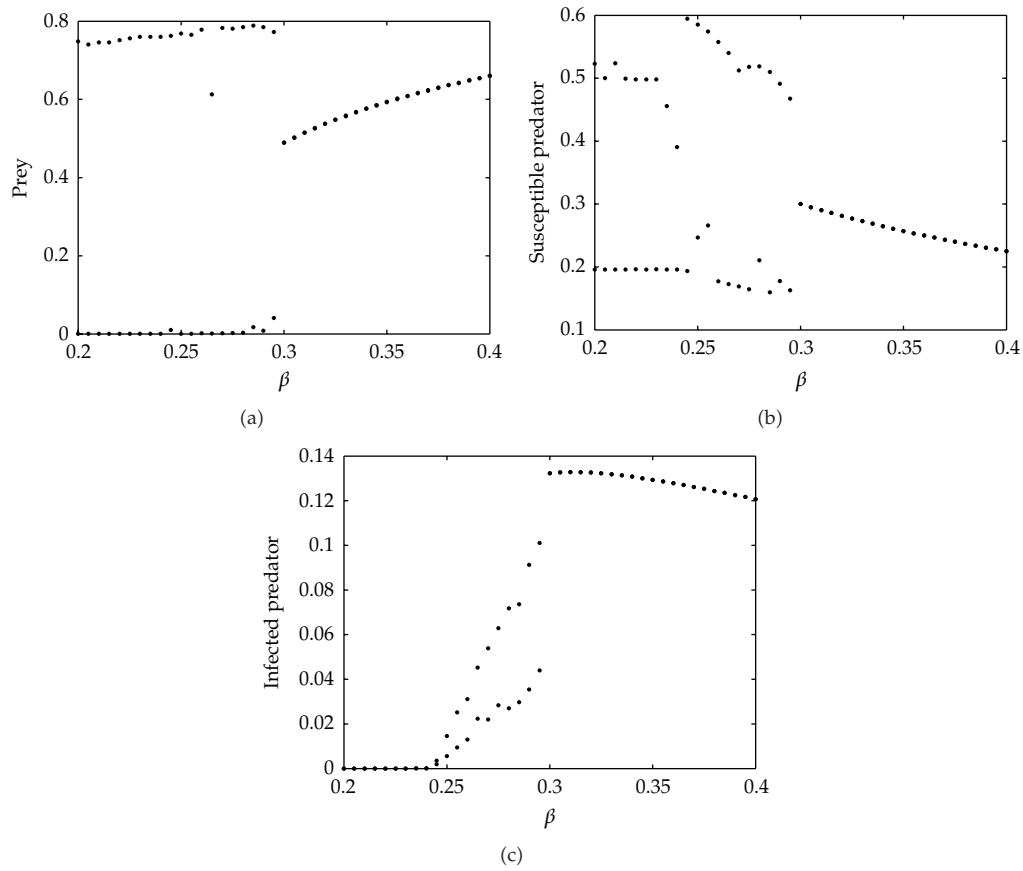

Figure 4: The figure indicates the bifurcation diagram for $\beta \in [0.2, 0.4]$ and also indicates that all three species coexist in stable position for $\beta > 0.3$ and other parameter values given in the Figure 1.

infection in predator population. We analyze the local stability of equilibrium points and community structure of model system by the help of ecological and disease basic reproduction numbers. This study provides insightful ecological and disease reproduction numbers for understanding how parasites structure community composition. Moreover, this study indicates that two very different outcomes are possible upon disease introduction: (1) the host population can either be driven to extinction, or (2) an otherwise unstable resident community can be stabilized. Adding or removing parasites from food webs might therefore has unexpected and dramatic consequences, possibly leading to extinctions or outbreaks on more than one trophic level. This highlights the importance of including infectious disease agents in food webs, which has begun to be recognized only recently [41].

We perform extensive numerical experiment and get an important result that the introduction of disease in predator population stabilizes predator-prey oscillations. Disease introduction in our model does not reverse the paradox of enrichment; it offers another potential explanation for why natural populations tend to be stable. Many species have a plethora of parasites and pathogens, making it possible that inherently cyclic behavior can be stabilized. In practice, however, it will be difficult to distinguish whether a particular system is stabilized due to disease or any other factor.

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