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Effect of delay on globally stable prey–predator system

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ABSTRACT

The present paper deals with an eco-epidemiological prey–predator model with delay. It is assumed that infection floats in predator species only. Both the susceptible and infected predator species are subjected to harvesting at different harvesting rates. Differential predation rates for susceptible and infected predators are considered. It is shown that the time delay can even destabilize the otherwise globally stable non-zero equilibrium state. It is observed that coexistence of all the three species is possible through periodic solutions due to Hopf bifurcation. With the help of normal form theory and central manifold arguments, stability of bifurcating periodic orbits is determined. Numerical simulations have been carried out to justify the theoretical results obtained.

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1. Introduction

Most of the ecological studies depend upon the population size of species and are under the influence of ecological and epidemiological factors. The ecological factors include species interactions in the form of competition and predation whereas epidemiological factors include the spread of infectious diseases [1,2]. It is becoming biologically relevant to study the effect of disease on the dynamics of ecological system. Some of the researchers deal with infection in prey species only [3–5]. On the other hand, some of them considered the situation where predator is infected with some disease [6–10]. Few studies deals with the situation where both the prey and predator species have got infected with some disease [11–14].

In recent years, several researchers [15–22] consider delay induced prey–predator model to analyze the stability of the system. Some authors like [23] described the effects of time lag in a prey–predator model incorporating parasite infection for the prey population. The combined effects of harvesting and delay on the dynamics of prey–predator systems has also been investigated by Wang and Pei [24], Pal et al. [25], Kar [26], Martin and Ruan [27] and Kar and Ghorai [28]. Xiao and Chen [8] proposed a predator–prey model with disease in prey. Their model shows that the introduction of a time delay in the coefficient of converting prey into predators has both stabilizing and destabilizing effects on the positive steady state.

Agnihotri and Juneja [29] proposed a harvested prey–predator SI (Susceptible-Infected) model with disease in predator. Many of such situations are outlined in [30]. In this paper, the role of differential predation in controlling the spread of disease in predator population has been shown. It has been proved that the interior equilibrium point is globally stable whenever it is locally stable. The role of controlled harvesting in controlling the spread of disease is also shown, but in this paper, the effect of time delay has not been taken into consideration. We know that almost all the processes in ecology involve time delays, so we may not get more realistic model without including time lags. In particular, Kuang [31] observes that animals take time to digest their feed, and this in fact delay their further activities. Keeping in view this fact, an eco-epidemiological delay model is proposed and analyzed in this paper. Also a constant time delay is incorporated in the logistic growth of the prey. The reason behind the consideration of delay in prey species is that the prey species take some time τ_1 to convert the food into its growth. It is assumed that the viral disease is spreading only among the predator species. Also it is considered that the reproduction of predators after predating the prey population is not instantaneous; thus it will be incorporated by taking some time lag τ_2 required for the gestation of predators.

The paper is structured in the following manner. In the next section, we present the model. Section 3 deals with the stability analysis of interior equilibrium point. The criteria for the existence of Hopf bifurcation around non zero equilibrium point is given in Section 4. In Section 5, stability of periodic solutions is discussed.

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Table 2.1
Variables and parameters used in the system (2.1).

Variable/parameters	Biological meaning
k	carrying capacity of the environment for prey
r	intrinsic growth rate of prey
l	disease transmission coefficient in predator
μ, μ'	mortality rates of susceptible and infected predators respectively with $\mu' > \mu$
γ, γ'	catchability coefficients of susceptible and infected predators respectively with $\gamma' > \gamma$
γ_1	catchability coefficient of prey species
E_1	harvesting effort rates for prey species
E	harvesting effort rates for predator species (both susceptible and infected)
α, α'	predation rates of susceptible and infected predators respectively with $\alpha' < \alpha$
β	numerical response

Numerical simulations are carried out in Section 6, to validate the theoretical results obtained. Finally, a brief conclusion is given in Section 7.

2. Formulation of Model

After considering the time delays in model by Agnihotri and Juneja, our delayed prey–predator model is as follows

$$\begin{aligned}\frac{dN_1}{dt} &= rN_1(t)\left(1-\frac{N_1(t-\tau_1)}{k}\right)-\alpha N_1(t)N_2(t)-\alpha' N_1(t)N_3(t)\\&\quad-\gamma_1E_1N_1(t)\\\frac{dN_2}{dt} &= \alpha\beta N_1(t-\tau_2)N_2(t)-\mu N_2(t)-\gamma EN_2(t)-lN_2(t)N_3(t)\\\frac{dN_3}{dt} &= \alpha'\beta N_1(t-\tau_2)N_3(t)-\mu' N_3(t)-\gamma' EN_3(t)+lN_2(t)N_3(t)\end{aligned}\tag{2.1}$$

$(0 \le N_1(0) \le k,\ 0 \le N_2(0),\ 0 \le N_3(0))$

The variables and parameters used in system (2.1) are given in Table 1.

Variable $N_1(t)$ denotes the density of prey species at any time t and $N_2(t)$, $N_3(t)$ denote the respective densities of susceptible and infected predator species. Also all the parameters in the model are assumed to be positive.

Suppose $N_1^{(0)}, N_2^{(0)}, N_3^{(0)}$ denote the initial functions associated with system (2.1), then

$$\begin{aligned}N_1^{(0)}(\theta) &= \phi_1(\theta) \ge 0\\N_2^{(0)}(\theta) &= \phi_2(\theta) \ge 0\\N_3^{(0)}(\theta) &= \phi_3(\theta) \ge 0\end{aligned}\tag{2.2}$$

where $\theta \in [-\tau, 0]$, $\phi = (\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], \mathbb{R}^3_{+0})$, $\mathbb{R}^3_{+0} = \{(N_1, N_2, N_3) : N_1 \ge 0, N_2 \ge 0, N_3 \ge 0\}$

2.1. Positivity and boundedness of the solution

Lemma 2.1. *All the solutions of the system (2.1) which initiate in R^3_{+0} are uniformly bounded.*

Proof. The proof of the lemma is obvious. See [17]. □

Lemma 2.2. *For given non negative initial functions (2.2), the solutions of the system (2.1) are non negative.*

Proof. The first equation of the system (2.1) gives

$$\frac{dN_1}{dt} = N_1(t)\left[r\left(1-\frac{N_1(t-\tau_1)}{k}\right)-\alpha N_2(t)-\alpha' N_3(t)-\gamma_1E_1\right]$$

which can be integrated to give

$$N_1(t) = N_1^{(0)}exp\left[\int_0^\infty\left\{r\left(1-\frac{N_1(t-\tau_1)}{k}\right)-\alpha N_2(t)-\alpha' N_3(t)-\gamma_1E_1\right\}dt\right]\tag{2.3}$$

Similarly from second and third equation of (2.1), we get

$$N_2(t) = N_2^{(0)}exp\left[\int_0^\infty\{\alpha\beta N_1(t-\tau_2)-\mu-\gamma E-lN_3(t)\}dt\right]\tag{2.4}$$

$$N_3(t) = N_3^{(0)}exp\left[\int_0^\infty\{\alpha'\beta N_1(t-\tau_2)-\mu'-\gamma' E+lN_2(t)\}dt\right]\tag{2.5}$$

Hence, Eqs. (2.3)–(2.5) proves the non negativity of the variables $N_1(t)$, $N_2(t)$, $N_3(t)$ [32]. □

3. Existence and stability of interior equilibrium point

Case 1. $\tau = 0$ we are only interested with endemic equilibrium point $E^*(N_1^*, N_2^*, N_3^*)$ of the system where all the populations coexists.

$$N_1^* = k\left(1-\frac{(\alpha\mu'-\alpha'\mu)+(\alpha\gamma'-\alpha'\gamma)E+l\gamma_1E_1}{rl}\right)$$

$$N_2^* = -\frac{\alpha'\beta N_1^*-\mu'-\gamma' E}{l}$$

$$N_3^* = \frac{\alpha\beta N_1^*-\mu-\gamma E}{l}$$

which exists provided

$$r-\gamma_1E_1 > (\alpha\mu'-\alpha'\mu)+(\alpha\gamma'-\alpha'\gamma)E\tag{3.1}$$

and

$$\frac{\alpha'\beta N_1^*-\mu'}{\gamma'} < E < \frac{\alpha\beta N_1^*-\mu}{\gamma}\tag{3.2}$$

i.e. a reasonable harvesting effort is required for the existence of all the three populations.

In [29] it is already proved that this interior equilibrium point become globally stable whenever it is locally stable.

Case 2. $\tau \ne 0$

To simplify the analysis, both the delays are assumed to be of equal magnitude i.e. $\tau_1 = \tau_2 = \tau$. However the model has been simulated extensively for unequal delays. The Characteristic roots corresponding to the equilibrium $E^*(N_1^*, N_2^*, N_3^*)$ are given by the equation

$$\begin{vmatrix}\lambda+\frac{rN_1^*}{k}e^{-\tau\lambda} & \alpha N_1^* & \alpha' N_1^*\\-\alpha\beta N_2^*e^{-\tau\lambda} & \lambda & lN_2^*\\-\alpha'\beta N_3^*e^{-\tau\lambda} & -lN_3^* & \lambda\end{vmatrix} = 0$$

The characteristic equation becomes

$$\lambda^3+B_1\lambda+e^{-\tau\lambda}(A\lambda^2+B\lambda+C) = 0\tag{3.3}$$

where $A = \frac{rN_1^*}{k}$, $B = \alpha^2\beta N_1^*N_2^*+\alpha'^2\beta N_1^*N_3^*$ and $C = \frac{l^2rN_1^*N_2^*N_3^*}{k}$, $B_1 = l^2N_2^*N_3^*$.

For stability of $E^*(N_1^*, N_2^*, N_3^*)$, all the eigenvalues of characteristic Eq. (3.3) should have negative real parts. It is difficult to establish the conditions under which the Eq. (3.3) has all roots with negative real parts. Therefore, the method of stability change has been adopted to discuss the stability and Hopf bifurcation of the system (2.1). In the following analysis, the parameter delay

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