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# Effects of additional food on an ecoepidemic model with time delay on infection

Banshidhar Sahoo *, Swarup Poria

*Department of Applied Mathematics, University of Calcutta, Kolkata, West Bengal, India*

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ABSTRACT

We propose a predator–prey ecoepidemic model with parasitic infection in the prey. We assume infection time delay as the time of transmission of disease from susceptible to infectious prey. We examine the effects of supplying additional food to predator in the proposed model. The essential theoretical properties of the model such as local and global stability and in addition bifurcation analysis is done. The parameter thresholds at which the system admits a Hopf bifurcation are investigated in presence of additional food with non-zero time lag. The conditions for permanence of the system are also determined in this paper. Theoretical analysis results are verified through numerical simulations. By supplying additional food we can control predator population in the model. Most important observation is that we can control parasitic infection of prey species by supplying additional food to predator. Eliminating the most infectious individuals from the prey population, predator quarantine the infected prey and prevent the spreading of disease.

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## 1. Introduction

Over the last 50 years, disease control has relied heavily on the use of chemical fungicides, bactericides and soil fumigants. Use of chemical to control disease has proved to have long term side effects on ecosystem viz. infected individuals contaminate the environment, further they get absorbed and remain as residue in the biomass of coexisting healthy species in the ecosystem. Therefore, non-chemical methods of disease control become a topic of great attention for many scientists. It is well-known that the non-prey food sources has long been recognised and attempts have been made to manipulate these non-prey sources (viz., nectar, pollen etc.,) in agricultural lands to enhance levels of biological disease control [1,2]. Several experiments [3,4] reported the benefits of using additional or alternative food supplements in biological control programs. Wäckers and van Rijn [3] pointed out that additional food in plant–herbivore–carnivore interactions is not only an important topic in basic ecology, but also directly applied for biological pest control. It is observed that quality and quantity of additional food play a vital role in the controllability of the system. These findings agree with the observations made in the recent review [4] dealing with the effects of artificial food sprays on conservational biological control wherein it is emphasised that the success of biological control much depends on quantity and quality of food sprays.

Additional foods (to predator) help to increase predators [5]. Harwood and Obrycki [6] pointed out that provision of alternative food to a generalist predator is twofold: on one hand these nutritious food items improve the predator population and on the other results reduction of prey consumption per individual predator. Thus studying the effects of additional food on a diseased predator–prey system when the predators are provided with additional food is important, which is the focus of the

* Corresponding author.
*E-mail addresses:* banshivu@gmail.com (B. Sahoo), swarup_p@yahoo.com (S. Poria).

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current study. In recent decades significant progress has occurred in the theory and application of epidemiology modelling of predator–prey systems [7,8] in absence of alternative food resource. The first eco-epidemic model with alternative food for the predator has been introduced by Haque and Greenhalgh [9]. Recently, Sahoo and Poria [10] reported the effects of additional food to predator in a diseased predator–prey system. They reported that increasing predator population by supplying additional food, one can remove infection from the system. But in the considered system [10] the infection process is instantaneous. Actually, in reality, an infected prey contacts a susceptible prey, the latter becomes infectious and therefore there is an infection time [11]. In the present paper, we investigate the effects of additional food on a epidemic predator–prey model with infection time delay.

Habitat complexity is the structural complexity of habitats. Habitat complexity can strongly mediate predator–prey interactions, affecting not only total predation rates, but also modifying selectivities for different prey species or size classes [12–14]. It is well established that habitat complexity reduces encounter rates of predators with prey [15,16]. Aquatic habitat becomes structurally complex in presence of submerged vegetation or aquatic weeds. It is observed that structural complexity of the habitat stabilizes the predator–prey interaction between piscivorous perch (predator) and juvenile perch and roach (prey) by reducing predator foraging efficiency. Therefore, it is important to incorporate the effect of habitat complexity when predator–prey interaction is studied by means of theoretical models.

Some field experiments reported that predator removal is one of the most important cause of disease spreading in prey populations [17,18]. Sih et al. [17] documented predator-removal experiments and found 54 out of 135 systems in which predator removal reduced the prey population. Similarly, Cote and Sutherland [18] found that predator removal reduced prey populations in 3 of 11 laboratory studies. If predators eliminate the most infectious individuals from the prey population, they will have an outcome equivalent to quarantine-whereby infectious individuals are removed from the healthy population and thereby prevented from spreading disease [19]. The above facts motivate us to investigate the effects of predator control on a diseased predator–prey model. Predator population is controlled by supplying additional food to predator in our model.

The paper is organised as follows: In the next section, an infected predator–prey model with habitat complexity in presence of additional food to predator is proposed. The effects of time delay in infection is incorporated. Section 3 contains the preliminary properties of the model. The conditions for the existence of various equilibria, local stability and conditions for existence of Hopf bifurcations are investigated in Section 4. The conditions for permanence of the system is presented in Section 5. Section 6 illustrates some of the key findings through numerical simulations. Section 7 highlights the consequence of providing additional food to predator for disease control. Finally, we draw conclusions in Section 8.

## 2. The model

The most commonly used functional response in a predator–prey model is the Holling Type II functional response [20]. The Holling type-II functional response is defined as

$$f(X) = \frac{A_1 X}{B_1 + X},$$

where $f(X)$ is the amount of food consumed, $X$ is the amount of food offered, $A_1$ is a proportionality constant related to the attack rate. Since the existence of habitat complexity reduces the probability of capturing a prey by reducing the searching efficiency of predator and habitat complexity affects the attack coefficient [21]. Therefore, the attack coefficient $A_1$ has to be replaced by $A_1(1-c)$, where $c$ $(0 < c < 1)$ is a dimension less parameter that measures the strength of habitat complexity. We assume that the complexity is homogeneous throughout the habitat. Then following Kot [22], the total number of prey caught(V), is given by

$V = A_1(1-c)T_v X$, where $T_v = T - hV$.

Here $T$ is the total time, $T_v$ is the available searching time. Solving for $V$ we get the modified Holling type-II response function as

$$V = \frac{TA_1(1-c)X}{B_1 + (1-c)X}.$$

Since, predator's functional response is defined as the number of prey caught by a predator at unit time, so the functional response in presence of habitat complexity is given by

$$f(X) = \frac{A_1(1-c)X}{B_1 + (1-c)X}.$$

Notice that for $c = 0$, there is no complexity, we get the original Holling Type II response function. Assuming density-dependent logistic growth of prey with intrinsic growth rate $R_0$, a predator–prey model in presence of habitat complexity is given by:

$$\begin{aligned} \frac{dX}{dT} &= R_0 X\left(1 - \frac{X}{K_0}\right) - \frac{A_1(1-c)XY}{B_1 + (1-c)X}, \\ \frac{dY}{dT} &= \frac{\epsilon A_1(1-c)XY}{B_1 + (1-c)X} - D_2 Y. \end{aligned} \tag{1}$$

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