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Harvesting as a disease control measure in an eco-epidemiological system – A theoretical study

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ABSTRACT

Epidemiology and ecology are traditionally treated as independent research areas, but there are many commonalities between these two fields. It is frequently observed in nature that the former has an encroachment into the later and changes the system dynamics significantly. In population ecology, in particular, the predator–prey interaction in presence of parasites can produce more complex dynamics including switching of stability, extinction and oscillations. On the other hand, harvesting practices may play a crucial role in a host–parasite system. Reasonable harvesting can remove a parasite, in principle, from their host. In this paper, we study theoretically the role of harvesting in a predator–prey–parasite system. Our study shows that, using impulsive harvesting effort as control parameter, it is not only possible to control the cyclic behavior of the system populations leading to the persistence of all species, but other desired stable equilibrium including disease-free can also be obtained.

1. Introduction

Both the theoretical ecology and the theoretical epidemiology are developed research fields and are treated separately. However, there are some common features between these two systems and merging the two areas may show interesting dynamics. Eco-epidemiology is a branch in mathematical biology which considers both the ecological and epidemiological issues simultaneously. 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 researchers have proposed and studied different predator–prey models in presence of disease. The literature in the field of eco-epidemiology has grown enormously in the last two decades and we are mentioning few of them [2,3,7,9,19,20,24,25,27,42,45–47].

Reasonable harvesting policy is indisputably one of the major and interesting problems in ecology and economics and have been studied for a long time. The exploitation of biological resources and harvest of population species are commonly practiced in fishery, forestry, agriculture and wildlife management. Harvesting has sometimes been considered as a stabilizing factor [22], a destabilizing factor [14] or even oscillation-inducing factor [15,31]. The problem of predator–prey interactions under constant rate of harvesting or constant quota of harvesting has been studied by many authors [5,6,11–13,22,30,38,40,41]. Parasites may reduce both abundance and yield by increasing mortality, reducing fecundity, affecting the size structure of the population or by reducing the marketability of harvested stocks [18]. It is, therefore, important in both fishery and conservative biology [39]. In many cases, over-exploitation has resulted in stressed populations of many species across the globe [37], and parasites that decrease host density have the potential to aggravate mortality in harvested stocks. Also, there are several examples in the literature that parasites are being eliminated locally from a population by reduction of their hosts' density [1,17]. As far as knowledge goes, nobody has explicitly put a harvested parameter in a predator–prey–parasite model and studied its effect on the system (but see [8]).

It is well known that the predator–prey interaction exhibits oscillations and these phenomena are also carried over to a host–parasite–predator interaction [19,24,25,29,43]. Oscillatory population may be driven to extinction in presence of environmental stochasticity when the population density is very low [19,32]. Therefore, the question is – how to control these oscillations if it arises in such eco-epidemiological situations? Here we study the role of harvesting in an eco-epidemiological system where the susceptible and infected prey are subjected to combined harvesting. The objectives of this study are the following:

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- Can harvesting regulate the cyclic behavior, if it exists, of the system populations?
- Can parasite wipe out host population in presence of harvesting?
- Under what conditions, harvesting can eliminate parasites in an eco-epidemiological system?
- What are the overall effects of harvesting in a predator–prey–parasite system?

The organization of the paper is as follows: Section 2 deals with the model formulation. In Sections 3 and 4 mathematical and numerical studies of the proposed model have been given. Finally, discussion is presented in Section 5.

2. Formulation of the model

The following assumptions are made in formulating the basic microparasitic eco-epidemiological model.

A predator–prey interaction is considered where the prey species follow the logistic dynamics in absence of predator and the predator consumes prey following type II response function. Logistic growth is mathematically represented by $f(N) = rN(1 - \frac{N}{K})$, r being the intrinsic growth rate, K being the carrying capacity of the environment and N being the prey density. The type II response function is represented by $\frac{gN}{h+N}$, where g is the prey capture rate and h is the half-saturation constant. Suppose a microparasite infects the prey population and divides it into two disjoint classes, viz. susceptible (S) and infected (I) populations, so that the total population at any time t is $N(t) = S(t) + I(t)$. It is assumed that only susceptible population is capable of reproducing and the infected population dies before having the capability of reproduction. However, the infective population consumes resources and contributes with susceptible prey to population growth towards the carrying capacity [7,19,25,47]. The disease is not genetically inherited and the infected population does not recover or become immune. Disease transmission is assumed to follow the law of mass action with λ as the transmission rate. Predators consume both the susceptible and infected preys. However, the predation rate (m) on infected prey may be high compared to that on susceptible prey (n) [36]. Predators may have to pay a cost in terms of extra mortality in the trade-off between the easier predation and the parasitized prey acquisition, but the benefit is assumed to be greater than the cost [28,35]. So we assume that consumption of infected prey also contributes positive growth to the predator population, contrary to Bairagi et al. [3].

From the above assumptions, we formulate the following basic eco-epidemiological model:

$$\begin{aligned}\frac{dS}{dt} &= rS\left(1-\frac{S+I}{K}\right) - \lambda IS - \frac{nSP}{a+S},\\ \frac{dI}{dt} &= \lambda IS - \frac{mIP}{a+I} - \mu I,\\ \frac{dP}{dt} &= \frac{n\alpha SP}{a+S} + \frac{m\alpha IP}{a+I} - dP.\end{aligned} \tag{1}$$

Variables and parameters used to describe the system have been defined in Table 1.

Let q_1 and q_2 be the catchability coefficients of the susceptible and infected prey, respectively, and E be the combined external effort devoted to non-selective harvesting of both the susceptible and infected preys by the external harvester (not by predator). The terms q_1ES and q_2EI thus represent the catch of the respective species. It is to be noted that q_1, the catchability coefficient of the susceptible prey, may be less than q_2, the catchability coefficient of the infected prey. The reason is that the infected prey is less active than their healthy counterpart; therefore, for the same effort E, the number of infected prey caught per unit time may be much higher than that of non-infected prey. All parameters are assumed to be positive. Thus, the dynamics of a host–parasite–predator interaction that includes non-selective prey harvesting can be described by the following set of three coupled differential equations:

$$\begin{aligned}\frac{dS}{dt} &= rS\left(1-\frac{S+I}{K}\right) - \lambda IS - \frac{nSP}{a+S} - q_1ES,\\ \frac{dI}{dt} &= \lambda IS - \frac{mIP}{a+I} - \mu I - q_2EI,\\ \frac{dP}{dt} &= \frac{n\alpha SP}{a+S} + \frac{m\alpha IP}{a+I} - dP.\end{aligned} \tag{2}$$

Lafferty and Morris [36] observed experimentally that the predation rates of piscivorous birds on infected fish is, on an average, 31 times higher than the predation rates on susceptible fish. Based on the above experimental observation, it is quite reasonable to assume that predator consumes infected prey only, that is $n = 0$. The above assumption is not only realistic but also simplifies the model significantly. Thus, the model (2) becomes

$$\begin{aligned}\frac{dS}{dt} &= rS\left(1-\frac{S+I}{K}\right) - \lambda IS - q_1ES,\\ \frac{dI}{dt} &= \lambda IS - \frac{mIP}{a+I} - \mu I - q_2EI,\\ \frac{dP}{dt} &= \frac{m\alpha IP}{a+I} - dP.\end{aligned} \tag{3}$$

System (3) has to be analyzed with the initial conditions

$$S(0) > 0, \quad I(0) > 0, \quad P(0) > 0.$$

We also assume, throughout the paper, that $S(0) + I(0) \leqslant K$.

It is to be mentioned that the model (3) is appropriate when disease persists heavily in the system so that there exists sufficient number of infected preys for the predators. This is possible only if λ is large; but in this case, the disease may have impact on the reproductive period and we need S individuals in order to replenish the prey population. So we assume that specific growth rate, r, of prey population is high enough so that continuous replenishment

Table 1
Notations used to denote variables and parameters[a].

Variable/ parameter	Units	Description	Default value
S	Number per unit area	Susceptible prey population	Variable
I	Number per unit area	Infected prey population	Variable
P	Number per unit area	Predator population	Variable
r	Per day	Intrinsic birth rate constant	3
K	Number per unit area	Environmental carrying capacity	45
λ	Per day	Transmission rate	–
n	Per day	Search rate of the susceptible prey	–
m	Per day	Search rate of the infected prey	–
a	Number per unit area	Half-saturation constant	15
μ[b]	Per day	Death rate of infected prey not due to predation	0.24
α	Per day	Conversion efficiency	0.4
d	Per day	Food independent predator death rate	0.09
q_1	Per day	Catchability coefficient of the susceptible prey	0.2
q_2	Per day	Catchability coefficient of the infected prey	0.5
E	Per day	Constant harvesting effort	–

[a] Most of the parameter values have been taken from Bairagi et al. [3].
[b] μ is natural death rate + virulence of the disease.