Applied Mathematical Modelling 34 (2010) 1861–1872

Contents lists available at ScienceDirect

Applied Mathematical Modelling

journal homepage: www.elsevier.com/locate/apm

Modelling a predator–prey system with infected prey in polluted environment

Sudipa Sinha [a,*], O.P. Misra [a], J. Dhar [b]

[a] School of Mathematics and Allied Sciences, Jiwaji University, Gwalior 474 011, Madhya Pradesh, India
[b] Department of Applied Sciences, ABV-Indian Institute of Information Technology and Management, Gwalior 474 010, Madhya Pradesh, India

ARTICLE INFO

Article history:
Received 1 April 2008
Received in revised form 20 September 2009
Accepted 8 October 2009
Available online 28 October 2009

Keywords:
Predator
Prey
SIS model
Disease
Toxicant
Persistence

ABSTRACT

A predator–prey model with logistic growth in prey is modified by introducing an SIS parasite infection in the prey. We have studied the combined effect of environmental toxicant and disease on prey–predator system. It is assumed in this paper that the environmental toxicant affects both prey and predator population and the infected prey is assumed to be more vulnerable to the toxicant and predation compared to the sound prey individuals. Thresholds are identified which determine when system persists and disease remains endemic.

1. Introduction

In modern era, the most threatening problem to society is the change in the environment caused by pollution, affecting the long term survival of species, human life style and bio diversity of the habitat. A great quantity of the toxicant and contaminants enter into the ecosystem one after another which seriously threaten the survival of the exposed population including human. In order to use and regulate toxic substance wisely, we must asses the risk of the populations exposed to toxicant. The problem of estimating qualitatively the effect of a toxicant on a population by mathematical models is a relatively new field that began only in the early 1980s. For a general class of single population models with toxicant stress, Ma et al. [1] obtained a survival threshold distinguishing between persistence in the mean end extinction of a single population under the hypothesis that the capacity of the environment is large relative to the population biomass, and that the exogenous input of toxicant into the environment is bounded. In 1987, Ma and Hallam [2] studied two dimensional non-autonomous Lotka–Volterra model by the average method and obtained sufficient conditions for persistence and for extinction of the population. The threshold of the survival for a system of two species in a polluted environment was studied by Huaping and Ma [3]. Population toxicant coupling has been applied in several contexts including Lotka–Volterra and chemostat like environments, resulting in ordinary, integro-differential and stochastic models. All these studies rely on the hypothesis of a complete spatially homogeneous environment. Recently, a spatial structure has been carried out by Zhan Li et al. [4] when a diffusive–convective model is proposed to describe the dynamics of a population in a polluted

* Corresponding author.
E-mail addresses: sudeepasinha@yahoo.com (S. Sinha), misra_op58@yahoo.co.in (O.P. Misra), jdhar@iiitm.ac.in (J. Dhar).

0307-904X/$ - see front matter © 2009 Elsevier Inc. All rights reserved.
doi:10.1016/j.apm.2009.10.003

environment and the sufficient criteria on persistence and extinction of the population are derived. Another serious problem for society is the spread of infectious disease. But it can also be a factor in regulating human and animal population sizes. For example, the black death in Europe in 14th century killed up to one fourth of the human population. European disease such as small pox brought by cortex and entered to Mexico decimated the native population over there in the 16th century. In complex ecosystem predator–prey relationship can also be important in regulating the numbers of prey and predators. For example, when a boundary was placed on natural predators such as wolves and coyotes in the Kaibab plateau in Arizona, the deer increased beyond the food supply, and then over half deer died of starvation in 1923–1925, [5]. However, predator control does not always cause the prey population increase. Sih et al. [6] found that predator removal decreased the prey population in 54 of the 135 system examined. The review of the Holmes and Bethel [7] contains many examples in which the parasite changes the external features or behavior of the prey, so that infected prey are more vulnerable to predation. Infected prey may live in location that are more accessible to predators; for example, fish or aquatic snails may live close to the water surface or snails may live on the top of the vegetation rather than under protective plant cover. Similarly, infected prey may be weaker or less active, so that they are caught more easily [8,9]. As species do not exists alone in nature, it is of more biological significance to study the persistence-extinction threshold of each population in system of two or more interacting species. In SI disease model in which susceptible prey grow logistically and predators eat only infected prey individuals, Chattopadhyay and Arino [10] found persistence and extinction conditions for the population and also determined conditions for Hopf bifurcation to periodic solutions. Here, we investigate the epidemiological, ecotoxicological and demographic effects in a predator–prey system in which the infected prey are more vulnerable to predation and toxicant. The application of the model can be seen in the context of a study made by [11]. In their study they have shown that juvenile salmon *Oncorhynchus* spp. and their prey bioaccumulate chlorinated hydrocarbons and aromatic hydrocarbons-important class of toxic xenobiotics. Furthermore, they have shown the exposure to these pollutants can lead to immunosuppression and increased disease susceptibility in juvenile salmon. Recent studies of natural fish populations have demonstrated that infectious disease-induced mortality can significantly reduce the size of the host population. By creating adverse environments e.g. pollutant which alter the susceptibility of the host to pathogens that are integral and ubiquitous components of the habitat, pollution increases the probability of disease-related impacts on fish population.

Keeping in the view above discussion, in this paper, we have proposed a predator–prey system with combined effect of disease and toxicant. It is assumed that the epidemiological interaction is SIS type and both prey and predator populations are affected by environmental toxicity. In analysis, threshold criteria are developed which determined when the predator population persist and when the disease remains endemic in the prey population in polluted environment. Section 2, describes demographic predator–prey system, epidemiological single species model with SIS interaction and a predator–prey model with infected prey in fresh (pollution free) and polluted environment. Section 3, identifies the boundary equilibria, interior equilibria and their stability thresholds for the predator–prey system in polluted environment. Section 4, contains results on persistence of predator–prey model with infected prey in polluted environment. Finally, a brief discussion has also been included in Section 5.

2. The mathematical formulation

Let $H(t)$ and $P(t)$ be the sizes of prey population and predator population, respectively. We first consider, the predator–prey model of the form:

$$\dot{H}(t) = r\left(1 - \frac{H}{K}\right) - aHP, \tag{1}$$

$$\dot{P}(t) = kaHP - c_1 P. \tag{2}$$

This is a modified Lotka–Volterra predator–prey model with density dependent logistic growth of the prey. The initial per capita growth rate in prey population is r and prey carrying capacity of the environment is K. The per capita death rate of the predator is c_1. The predation rate is a and the feeding efficiency in turning predation into new predator is k, so the birth rate of the new predator is $akHP$.

Study of the infectious disease in natural animal population has been a subject of great interest among the ecologists. When the infection does not lead to immunity, so that infectives become susceptible again after recovery the disease is called SIS disease. When infectives have permanent immunity after recovery, the disease is called SIR disease. Bacterial infections tend to be SIS, while viral infections correspond to SIR disease. Animals are called susceptible if they can become infected, are called infectious if they are infected and can transmit the infection. Thus the total size of the prey population is $H = X + Y$, where X is the number of susceptible prey and Y is the number of infected prey. Then $S = X/H$ is the fraction of the prey that are susceptible and $I = Y/H$ is the fraction that are infectious. If β is the average number of the contacts of an animal per unit time then $\beta X/H = \beta S$ is the average number of contacts per unit time of one infectious animal. Since there are $Y = IH$ infectives then number of new cases per unit time in the X susceptible is equal to the standard incidence $(\beta X/H)Y = \beta ISH$. The standard incidence is the preferred formulation for animal populations by many researchers [12–15]. The movement out of the infectious class due to recovery is given by γY in the epidemic model. Moreover there is no disease-related deaths so we get the following SIS disease model [16]: