Applied Mathematics and Computation 284 (2016) 268–285

Contents lists available at ScienceDirect

Applied Mathematics and Computation

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

Modeling of hunting strategies of the predators in susceptible and infected prey

Yang Lu, Dan Li, Shengqiang Liu*

The Academy of Fundamental and Interdisciplinary Science, Harbin Institute of Technology, #3026, 2 Yi-Kuang Street, Harbin 150080, China

ARTICLE INFO

MSC:
34K11
37N25
92D25

Keywords:
Predator–prey
Infected prey
Staged structure
Global dynamics
Hunting strategies

ABSTRACT

A predator–prey model with epidemic prey and staged structure in the predators, where the predators are assumed to keep constant preference probability of predation on susceptible/infected prey, is formulated to study the hunting strategies of predators for maximum surviving rate as well as maximum density. Given that the disease is endemic among prey before the invasion of predators, global dynamics of the model are obtained and threshold dynamics determined by the predator's net reproductive number R_H are established: the predators go extinct if $R_H < 1$; and predators persist if $R_H > 1$. As an application, the hunting strategies of the predators for the maximum R_H are studied, and it is shown that the predators should only hunt the susceptible prey when the disease is just slightly endemic, while if the disease is seriously endemic, they should only hunt the infected prey. Numerical simulations are performed to verify/support the theoretical results and to consider the hunting strategies of predators for their maximum density, for which it is shown that the predators should keep some proper preference probability on both susceptible and infected prey.

1. Introduction

It is a central goal for theoretical ecologists to understand the relationship between predators and their prey, and one of the main components of the predator–prey relationship is the influence of disease on the ecosystem, which is mainly reflected in disease-related mortality, disease-reduced reproduction, change in population size and induced oscillation of population states [1,18,25]. In particular, the disease can change the behavioral patterns of prey, which makes them more vulnerable to predation [6]. For instance, in Salton Sea of California, fish population infected by a vibrio class of bacteria may become weaker and susceptible to be caught by piscivorous birds [8,9]. Peterson and Page [10] observed that most Isle Royale wolves, in Lake Superior, are more successful to capture moose infected with lungworm. In addition, predators like lions or wolves have a natural tendency to select prey that are weakened by a disease [11,12], and to consume disproportionately infected prey [7]. Therefore, it is necessary to study the interaction between the predators and their prey with the existence of infected prey.

In the following decades, the literature in the field of eco-epidemiology has grown enormously [3–5,13,14,18–20,25,26]. Anderson and May [2] did theoretically pioneering work in the epidemiology and ecology, and formulated a predator–prey model where prey species were infected by some disease. Niu et al. [13] discussed and modified a nonautonomous

* Corresponding author. Tel.: +86045186402848.
E-mail address: sqliu@hit.edu.cn, lsqmath@gmail.com (S. Liu).

http://dx.doi.org/10.1016/j.amc.2016.03.005
0096-3003/

eco-epidemic model with disease in the prey, where they assumed that the predators only hunted the infected prey. Hethcote et al. [25] incorporated the *SIS* parasitic infection in the prey, with infected prey being more vulnerable to predation, into a predator–prey model with logistic growth in the prey, and they concluded that the predation on the infected prey can help eradicate the disease. By introducing predator switching in an eco-epidemiological model with disease in the prey, Mukhopadhyay and Bhattacharyya [23] investigated the role of predator switching on disease dominated ecological populations; They have identified two important predator characteristics (i.e., predator mortality and predator efficiency) and obtained some threshold values of these parameters that are crucial for controlling disease eradication and species coexistence. However, the global asymptotical behaviors of the model are not discussed, and optimal hunting strategies of the predators based on different prey carrying capacities, and infection rates and hunting rates are not analyzed and discussed.

Motivated by the above work, in this paper we will propose and consider a predator–prey model with infected prey and stage-structured predator. Our main purpose is to study the optimal hunting strategies of predators for maximum surviving probability as well as for maximum population density, to do that, we will obtain the global asymptotical behaviors of the model by applying the uniform persistence theory (for reference, see [31, p. 16] and [35]) and constructing suitable Lyapunov functionals.

This paper is organized as follows. In the next section, we present our model and introduce some preliminaries used in the later parts. In Section 3, we investigate dynamics of the model in the cases of $\beta K < c$ and $\beta K > c$, respectively. In the following section, we perform numerical simulations to verify our analytical results. At the end of the paper, we give a summary of the results.

2. Model description and preliminaries

2.1. Model formulating

Our basic model consists of two populations: (i) the prey whose population density is denoted by $N(t)$ and (ii) the predators which are divided into the mature $P(t)$ and immature $P_j(t)$ subpopulations. Before giving our mathematical model, we make the following assumptions:

- In the absence of the disease, the prey population obey the logistic growth, which is mathematically described by $dN/dt = rN(1 - N/K)$ with the intrinsic growth rate r and the carrying capacity of the environment K.
- Suppose a microparasite only infects the prey population and divides it into two classes: the susceptible prey $S(t)$ and the infected prey $I(t)$. Then the total population of the prey at any time t is $N(t) = S(t) + I(t)$. It is assumed that the infected population dies before having the capability of reproduction, i.e., only susceptible individuals are capable of reproducing. However, the infective individuals also consume resources and contribute with susceptible prey to population growth towards the carrying capacity [15–17,20].
- It is assumed that the disease is not genetically inherited and the infected population does not recover or become immune. And the disease transmission rate is assumed to be the law of mass action with β as the transmission rate, where β is the probability of transmission per contact (a positive constant). Indeed, for eco-epidemiology modeling, both mass action transmission rate [22,23] and standard transmission rate [25] exist. Because the main purpose of this paper is to understand the hunting strategies of predator, we consider only one of them for the mathematical simplicity.
- Predators consume both the susceptible and infected preys, and we denote by p_S, p_I $(0 \le p_S, p_I \le 1)$ the probability that a predator will attack the prey S or I [37]; We assume $p_S + p_I = 1$, i.e., the predators choose either to attack prey S or to attack prey I.
  We denote by H_S, H_I the predation rate on prey S, I, respectively, and let $n = p_S \cdot H_S$, $m = p_I \cdot H_I$; we assume that $H_I \ge H_S$ because the infected prey I is more vulnerable to the predation than the uninfected prey S [21].
- The predator is assume to be stage-structured, i.e., to have diversities during the life histories: (1) Immature predators reach maturity after surviving the immature stage of fixed time lag; (2) immature predators are raised by their parents or dependent on the nutrition, hence they do not feed on prey; (3) only mature predator has the ability to reproduce. For references of stage-structured population models, we refer to [29,33,38,43,44].
- The functional response of the predation is assumed to take the predator-dependent Beddington–DeAngelis type, regarded in many literatures [29,39–41] to provide good descriptions of predator feeding over a range of predator–prey abundances, and in some cases by Skalski and Gilliam [39]. Indeed, the Beddington–DeAngelis response can be generated by a number of natural mechanisms [40,41] and it admits rich but biologically reasonable dynamics [42].

Based on the preceding assumptions, our model is formulated as follows

$$\begin{cases} S'(t) = rS(t)\left(1 - \dfrac{S(t)+I(t)}{K}\right) - \beta I(t)S(t) - \dfrac{nS(t)P(t)}{1 + k_1 S(t) + k_2 P(t)}, \\ I'(t) = \beta I(t)S(t) - \dfrac{mI(t)P(t)}{1 + k_1 I(t) + k_2 P(t)} - cI(t), \\ P'(t) = \dfrac{\alpha n P(t-\tau)S(t-\tau)e^{-d_j\tau}}{1 + k_1 S(t-\tau) + k_2 P(t-\tau)} + \dfrac{\alpha m P(t-\tau)I(t-\tau)e^{-d_j\tau}}{1 + k_1 I(t-\tau) + k_2 P(t-\tau)} - dP(t), \end{cases} \tag{1}$$