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Revealing the role of predator interference in a predator–prey system with disease in prey population

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ABSTRACT

Predation on a species subjected to an infectious disease can affect both the infection level and the population dynamics. There is an ongoing debate about the act of managing disease in natural populations through predation. Recent theoretical and empirical evidence shows that predation on infected populations can have both positive and negative influences on disease in prey populations. Here, we present a predator–prey system where the prey population is subjected to an infectious disease to explore the impact of predator on disease dynamics. Specifically, we investigate how the interference among predators affects the dynamics and structure of the predator–prey community. We perform a detailed numerical bifurcation analysis and find an unusually large variety of complex dynamics, such as, bistability, torus and chaos, in the presence of predators. We show that, depending on the strength of interference among predators, predators enhance or control disease outbreaks and population persistence. Moreover, the presence of multistable regimes makes the system very sensitive to perturbations and facilitates a number of regime shifts. Since, the habitat structure and the choice of predators deeply influence the interference among predators, thus before applying predators to control disease in prey populations or applying predator control strategy for wildlife management, it is essential to carefully investigate how these predators interact with each other in that specific habitat; otherwise it may lead to ecological disaster.

1. Introduction

The relationship between predation and infection in prey populations is complex. There is evidence showing both increase and decrease of infection in prey populations in response to predation (Packer et al., 2003; Holt and Roy, 2007; Cáceres et al., 2009). According to "healthy herds" hypothesis (Packer et al., 2003), selective predation by the predator on infected prey helps to eliminate infectious individuals from the healthy population and thereby prevents the spread of disease. Evidence from different other fields also support this hypothesis (Pulkkinen and Dieter, 2006). Various programs for the management of disease in natural populations also suggest the control of the diseased population through predation (Hudson et al., 1998; Choisy and Rohani, 2006; Greenman and Hoyle, 2010; Hawlena et al., 2010). On the other hand, there are also studies showing an increase in infection in prey populations due to the presence of predators (Holt and Roy, 2007; Bate and Hilker, 2013a). Recently, Cáceres et al. (2009) presented an example using field patterns, experiments and a model study to show that the release of infective spores of fungal parasite by the predator facilitates epidemics in *Daphnia* population. Predators can also affect the persistence of prey populations that are regulated by infectious diseases (Chattopadhyay and Arino, 1999; Roy and Chattopadhyay, 2005). Therefore, how predators affect the disease dynamics in prey populations is still not clear and, thus, an interesting topic of research.

In the presence of a predator, a system with disease in a prey population can show different complex dynamical behaviors, like bistability, quasi-periodicity and chaos. Previously, there are studies showing some of these complex dynamics. For example, Upadhyay et al. (2008) found the existence of chaos via a period-doubling route

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in a predator–prey system with disease in a prey population. After the addition of a free-living virus stage in a predator–prey model with disease in a prey population, Siekmann et al. (2010) found bistability where depending on the initial conditions, the system can be made disease-free. Hilker and Malchow (2006) found strange periodic attractors with complicated, long lasting transient dynamics in a predator–prey model with disease transmission in a prey population. Furthermore, Sieber and Hilker (2011) demonstrated the occurrence of chaos, bistability and attractor crisis. The existence of such complexity makes the disease dynamics more complicated and difficult to predict. Thus, to know how a predator population affects disease dynamics, first we need a more thorough study on how the predator population affects the complexity of the system and then analyzing those complex results we can get information regarding the disease dynamics.

In most of the studies, it is assumed that predators do not interfere with each other's activities; thus the competition among predators occurs only via depletion of prey abundance. In reality, there are several situations when predators have to encounter with other predators, especially when predators have to search for food (and therefore, have to share or compete for food). In fact, predator interference has been found to occur quite frequently in laboratory and natural systems (Kratina et al., 2009; Skalski and Gilliam, 2001; Salt, 1974). There is many significant evidence of predator interference in predator–prey systems involving herbivore-plant, snail–barnacle, parasite–host, mite–mite and beetle–cricket interactions (Arditi and Ginzburg, 1989; Salt, 1974). Analyzing published data on eight predator–prey and seven host–parasitoid systems, Arditi and Akakaya (1990) evidenced strong predator interference in twelve out of fifteen cases. Predator interference is also important at very low and high prey and predator densities (Kratina et al., 2009; Skalski and Gilliam, 2001). Moreover, previous studies have shown that interference among predators is a dominant driver of food-web stability (Chakraborty and Chattopadhyay, 2011; Rall et al., 2008; van Voorn et al., 2008; Huisman and De Boer, 1997) and also has the ability to generate patchiness in a homogeneous environment (Alonso et al., 2002). In spite of such huge importance, the effects of predator interference on the predator–prey-disease interactions have never been thoroughly investigated. This paper is aimed to bridge the existing gap.

There are different ways of incorporating predator interference in a mathematical model, e.g., by considering ratio dependent functional response (Arditi and Ginzburg, 1989), including predator interference in a Holling type I functional response (Seo and De Angelis, 2011), including predator interference in a Holling type II functional response (Beddington, 1975; DeAngelis et al., 1975), density dependent mortality of predators (Holt, 1977). However, several previous researchers have suggested in favor of using Beddington–DeAngelis functional response which is similar to Holling type II functional response, but contains an extra term describing mutual interference among predators (Kratina et al., 2009; Skalski and Gilliam, 2001; Huisman and De Boer, 1997).

In the present study, we consider a predator–prey system in which the prey population is subjected to an infectious disease. We assume that the disease is transmitted via both vertically and horizontally. For horizontal transmission, we consider the density dependent disease transmission among the prey population, whereas due to vertical transmission, an infected prey produces only infected individuals (Sieber et al., 2014). This kind of vertical transmission occurs in the case of lysogenic infection where viruses enter and integrate their genome into the host's genome and start reproducing as the host reproduces and duplicates its genome (Malchow et al., 2004). For example, plankton system is very prone to lysogenic infection (Fuhrman and Suttle, 1993). Previously, there are several mathematical modeling studies dealt with lysogenic infection in prey populations (Sieber et al., 2014; Malchow et al., 2004, 2005; Hilker et al., 2006). We further assume that the growth rate of susceptible prey is higher than that of the infected one (Hilker and Malchow, 2006; Hilker et al., 2006). The predator can consume both infected and healthy preys; however, the attack rates on infected and healthy preys are different (Chattopadhyay and Arino, 1999; Malchow et al., 2004; Hilker and Malchow, 2006; Hilker et al., 2006). We consider that predators interfere with each other and we represent this interference by considering Beddington–DeAngelis functional response (Beddington, 1975; DeAngelis et al., 1975). In order to study the long-term dynamics of the model we use numerical analysis techniques and perform a detailed numerical bifurcation analysis using AUTO (Doedel and Oldeman, 2009).

Here, we focus on investigating the role of predator interference on the disease dynamics in a prey population. For this reason, first we observe how interference among predators affects the system dynamics, especially, the complexity of the system by varying the interference strength and carrying capacity. From there, we comment on how different predator populations with different interference strengths regulate disease outbreaks and the persistence of the prey population.

The paper is organized as follows: in Section 2, we introduce the model for our investigation and mention about the possible equilibrium points. In Section 3, we examine how predator interference affects system dynamics by performing a rigorous bifurcation analysis on the model system. Finally, the paper ends with a discussion given in Section 4.

2. Basic model structure

We build an eco-epidemiological model that tracks population dynamics of susceptible prey $S(t)$, infected prey $I(t)$ and predator population $P(t)$ at time t. We construct the model based on the following assumptions:

(A1) In the absence of infected (susceptible) prey and predation, the susceptible (infected) prey population follows logistic growth (Malchow et al., 2004; Hilker and Malchow, 2006).

(A2) In the absence of predation, the susceptible and infected prey populations compete which is described by the classical Lotka–Volterra competition model. The interaction is weak–weak so that an interior stable equilibrium exists (Kot, 2001). Both susceptible and infected preys have a common carrying capacity K (Sieber et al., 2014). Moreover, the susceptible population becomes infected following the simple law of mass-action. We consider that the growth rate of infected prey is reduced due to infection (Hilker and Malchow, 2006; Hilker et al., 2006). We also assume that susceptible and infected individuals produce only susceptible and infected individuals, respectively.

(A3) The infected prey does not recover or become immune but are removed by a constant death rate.

(A4) The Beddington–DeAngelis functional response is chosen to represent predator's per capita feeding rate on susceptible and infected preys as $\beta_1 S/(1 + T_h(S + I) + T_i P)$ and $\beta_2 I/(1 + T_h(S + I) + T_i P)$ respectively, where β_1/T_h and β_2/T_h are the maximum uptake rates of susceptible and infected prey, respectively, and T_i is the constant positive parameter representing the interference among predators. It is to be mentioned here that the handling times for infected and susceptible prey are assumed to be same.

(A5) Disease is spreading among the prey population only and the predator population is not directly affected by disease due to the predation of infected prey.