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Stabilization and complex dynamics in a predator–prey model with predator suffering from an infectious disease

Bob W. Kooi [a,*], George A.K. van Voorn [b], Krishna pada Das [c]

[a] Department of Theoretical Biology, Faculty of Earth and Life Sciences, Vrije Universiteit, de Boelelaan 1087, 1081 HV Amsterdam, The Netherlands
[b] Wageningen University and Research Centre, Biometris, Building 107, Droevendaalsesteeg 1, 6708 PB Wageningen, The Netherlands
[c] Mahadevananda Mahavidyalaya, Department of Mathematics, Monirampore, P.O.-Barrackpore, Kolkata 700120, India

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ABSTRACT

We study the effects of a non-specified infectious disease of the predator on the dynamics a predator–prey system, by evaluating the dynamics of a three-dimensional model. The predator population in this (PSI) model is split into a susceptible and an unrecoverable infected population, while all newborn are susceptible. The incidence rate at which susceptible become infectious is described by a Holling type II functional response giving saturation when the number of susceptibles increases. From a modeling context this three-dimensional model is in the limit case similar to the well-known 3D Rosenzweig–MacArthur (RM) model, with the infected population replacing the top-predator. The RM model is known for the Shil'nikov bifurcation, which is associated to the chaotic behaviour. The effects of the disease are considered to be changes in the parameters that represent relative predation efficiency and mortality rates. A combination of analysis, numerical integration and numerical continuation techniques are used to perform a bifurcation analysis of the model. The positive stationary solution of the disease free, two-dimensional predator–prey system is either a stable equilibrium or a stable limit cycle where the transition occurs at the Hopf bifurcation. For a biologically applicable parameter set, it is found that when the infected individuals feed less fast or less effective than the susceptibles there is bi-stability where the two-dimensional disease free state co-exists with a stable equilibrium for the three-dimensional PSI system. The introduction of a disease can also cause chaos when the infected predator individuals are ecologically not functioning (not feeding and no offspring). However, under small parameter changes first the Shil'nikov bifurcation, and hence the chaotic behaviour, disappears followed by the Hopf bifurcation that marks the existence of limit cycles of the three-dimensional PSI system. As such, an infectious disease has a strongly stabilizing effect on the predator–prey system, similar to the existence of weak links in food webs.

1. Introduction

The last two decades ecologists and epidemiologists have become increasingly interested in the structuring effects of parasites and pathogens within food webs and multiple-species communities. The merger of the two disciplines, ecology that studies population dynamics and epidemiology that studies the effects of a disease on a population, is known as *eco-epidemiology*. Anderson and May (1986) were the first to combine the two fields and considered ecological and epidemiological issues simultaneously. This has led to the insight that infectious diseases can have regulating effects not only on their host population, but also on other species their host interacts with (Anderson and May, 1986; Dobson and Hudson, 1986; Grenfell and Dobson, 1995; Sait et al., 2000; Hudson et al., 2001, 2006; Holt et al., 2003; Lafferty et al., 2006, 2008; Naji et al., 2010). Predator–prey models where the prey species is infected by some disease have been studied by Anderson and May (1986), Hadeler and Freedman (1989), Venturino (1994), Chattopadhyay and Arino (1999), Xiao and Chen (2001), Hethcote et al. (2004) and Greenhalgh and Haque (2006) and where the predator species is infected instead, by Venturino (2002), Haque and Venturino (2006, 2007), Hilker and Schmitz (2008), Stiefs et al. (2009) and Oliveira and Hilker (2010). The study of the effects on the dynamics of a predator–prey system with an infected predator has a great importance, when the question of predator control is concerned.

In the present study we consider the RM predator–prey model (Rosenzweig and MacArthur, 1963) as a reference model, where the prey population is limited by the carrying capacity. We focus on the effects of an infectious disease of the predator on the possible occurrence of complex dynamics like chaos. In Das et al. (2009) the effects of a disease in the prey population was considered, here only the predator species is infected by the disease and therefore in the model the infected predator

* Corresponding author. Tel.: +31 20 5987129.
E-mail address: kooi@bio.vu.nl (B.W. Kooi).

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subpopulation is considered explicitly as a third dynamical equation. Newborn predator individuals are born uninfected: hence there is no vertical infection. Individuals that have become infected remain infected until death. The disease can work via different modes of actions on demographic parameters, and here we primarily study the effects on the consumption efficiency of the infected predators, and secondary the mortality rates of the susceptible and infected predators.

From ecological modeling it is well known, that the up-scaling from two to three variables possibly allows for the occurrence of chaotic behaviour in the model. In fact, the three-dimensional variant of the RM model, where a top predator population that feeds on the predator population is included, was the first ecological model in which chaos was shown to occur (Hogeweg and Hesper, 1978; Hastings and Powell, 1991; Klebanoff and Hastings, 1994a,b; McCann and Yodzis, 1995; Kuznetsov and Rinaldi, 1996; De Feo and Rinaldi, 1998; Kuznetsov et al., 2001; Deng and Hines, 2002). Since infected individuals can only be "born" through infection of a susceptible predator individuals, the infected subpopulation could be perceived as a top predator that feeds on the predator population. Indeed, in a limit case where infection does not change the ecological functioning, the PSI model collapses to the 3D-RM model, displaying all the complex dynamical characteristics. These dynamics have been studied extensively (Hastings and Powell, 1991; De Feo and Rinaldi, 1998; Kuznetsov et al., 2001; Deng and Hines, 2002) and recently in van Voorn et al. (2010), in which an overview of the known global bifurcations and their consequences is given.

Also in other, epidemiological systems chaos has been shown to occur. Schaffer and Kot (1985a) have been especially persuasive in their view that chaos may be a much more important phenomenon than ecologists had earlier believed. A number of simple epidemiological systems with seasonality in contact rates unequivocally demonstrate chaos (Schaffer and Kot, 1985a). In Schaffer and Kot (1985b) and Olsen et al. (1988), it is shown that measles in New York, Baltimore and Denmark may be a specific example of this behaviour. Grenfell and Dobson (1995) investigated the effects of locally chaotic dynamics on global persistence in standard epidemiological models. And recently in Chatterjee et al. (2006) and Upadhyay et al. (2007) it is observed that chaotic dynamics occur in eco-epidemiological models.

In this paper we consider the complex dynamical behaviour in the PSI model, and under which conditions non-equilibrium dynamics disappears and the infected system possesses a stable equilibrium. We analysed the resulting PSI model using bifurcation theory (Guckenheimer and Holmes, 1985; Wiggins, 1988, 1990; Kuznetsov, 2004), where the asymptotic behaviour of the system (equilibria, periodic cycles, chaos) is evaluated under parameter variation for qualitative changes. A qualitative change in the asymptotic behaviour is then referred to as a bifurcation point. For examples of ecological applications in general see Bazykin (1998) and Kooi (2003), and for specifically the RM model in Kuznetsov and Rinaldi (1996), Boer et al. (2001), Kuznetsov et al. (2001), Doedel et al. (2008, 2009) and van Voorn et al. (2010) and references therein. The mortality rates of the susceptible and infected subpopulations are taken as bifurcation parameters while the consumption efficiency of the infected predator population is varied.

Expressions for the basic reproduction numbers, R_0, are related to the occurrence of a transcritical bifurcation of an equilibrium or limit cycle. These *TC* bifurcations form the boundaries of positive existence regions in the parameter space. The two-dimensional disease-free PS-system has a positive equilibrium below a critical value of the mortality rate of the predator at a transcritical bifurcation. This equilibrium becomes unstable below a second critical value, a Hopf bifurcation, where the system shows oscillatory behaviour. The results show that chaos can occur in a predator–prey system with disease in the predator population: a cascade of period doublings is found to be a route that leads to chaos. The organizing centre for chaos in the food chain model is found to be the homoclinic saddle point equilibrium Shil'nikov bifurcation. The chaos disappears when infected predator individuals consume the prey and contribute by reproduction of susceptible individuals to the growth of the predator population. When the consumption efficiency of the infected population is above a certain value the PSI system is unconditionally stable, also where the disease-free PS system possesses oscillatory behaviour. This reveals a strong stabilizing effect of an infectious disease of the predator.

2. Model formulation

The classical predator–prey 2D-RM model (Rosenzweig and MacArthur, 1963) reads

$$\frac{dX}{dT} = RX\left(1 - \frac{X}{K}\right) - \frac{AXY}{B+X}, \tag{1a}$$

$$\frac{dY}{dT} = \frac{CAXY}{B+X} - DY, \tag{1b}$$

where X and Y are the prey and predator population sizes. The prey population grows logistically with carrying capacity K and intrinsic growth rate R. The predator consumes the prey according to the Holling type-II functional response (Holling, 1959). Here, B represents the half saturation constant, A is the maximum ingestion rate, C is the conversion factor and D is the death rate of predator population.

The 3D-RM model version includes a top predator population, and reads

$$\frac{dX}{dT} = RX\left(1 - \frac{X}{K}\right) - \frac{A_1XY}{B_1+X}, \tag{2a}$$

$$\frac{dY}{dT} = \frac{C_1A_1XY}{B_1+X} - D_1Y - \frac{A_2YZ}{B_2+X}, \tag{2b}$$

$$\frac{dZ}{dT} = \frac{C_2A_2YZ}{B_2+X} - D_2Z, \tag{2c}$$

where Z is the top-predator (see also Das et al. (2009)).

Continuing with the predator–prey model (1) the predator population consists of two subpopulations. When the size of the susceptible subpopulation is denoted by P and the infected subpopulation by Q, then $Y = P + Q$. We assume that the disease spreads within the predator population only and that the total predator population can be split into a susceptible and an infected part. We further assume that all newborn individuals are susceptible and uninfected. Individuals that have become infected remain infected until death.

In the classical epidemiological models the Law of Mass Action incidence rate per susceptible-infected couple is assumed to describe the transmission. Here we use the Holling type II function to describe the infection mechanism.

With the above assumptions, the model (1) takes the following form:

$$\frac{dX}{dT} = RX\left(1 - \frac{X}{K}\right) - \frac{AX(P+bQ)}{B_1+X}, \tag{3a}$$

$$\frac{dP}{dT} = \frac{AX(C_1P + C_2bQ)}{B_1+X} - \frac{\lambda PQ}{B_2+P} - D_1P, \tag{3b}$$

$$\frac{dQ}{dT} = \frac{\lambda PQ}{B_2+P} - D_2Q. \tag{3c}$$