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Ecoepidemic predator–prey model with feeding satiation, prey herd behavior and abandoned infected prey

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ABSTRACT

In this paper we analyse a predator–prey model where the prey population shows group defense and the prey individuals are affected by a transmissible disease. The resulting model is of the Rosenzweig–MacArthur predator–prey type with an SI (susceptible-infected) disease in the prey. Modeling prey group defense leads to a square root dependence in the Holling type II functional for the predator–prey interaction term. The system dynamics is investigated using simulations, classical existence and asymptotic stability analysis and numerical bifurcation analysis. A number of bifurcations, such as transcritical and Hopf bifurcations which occur commonly in predator–prey systems will be found. Because of the square root interaction term there is non-uniqueness of the solution and a singularity where the prey population goes extinct in a finite time. This results in a collapse initiated by extinction of the healthy or susceptible prey and thereafter the other population(s). When also a positive attractor exists this leads to bistability similar to what is found in predator–prey models with a strong Allee effect. For the two-dimensional disease-free (i.e. the purely demographic) system the region in the parameter space where bistability occurs is marked by a global bifurcation. At this bifurcation a heteroclinic connection exists between saddle prey-only equilibrium points where a stable limit cycle together with its basin of attraction, are destructed. In a companion paper (Gimmelli et al., 2015) the same model was formulated and analysed in which the disease was not in the prey but in the predator. There we also observed this phenomenon. Here we extend its analysis using a phase portrait analysis. For the three-dimensional ecoepidemic predator–prey system where the prey is affected by the disease, also tangent bifurcations including a cusp bifurcation and a torus bifurcation of limit cycles occur. This leads to new complex dynamics. Continuation by varying one parameter of the emerging quasi-periodic dynamics from a torus bifurcation can lead to its destruction by a collision with a saddle-cycle. Under other conditions the quasi-periodic dynamics changes gradually in a trajectory that lands on a boundary point where the prey go extinct in finite time after which a total collapse of the three-dimensional system occurs.

1. Introduction

Recently the role of social behavior in the context of interacting populations has been introduced in predator–prey models. In the classical Rosenzweig–MacArthur model [19,20] both prey and predators have an homogeneous spatial distribution. The prey grows logistically in the absence of the predator and the natural predator mortality rate. The predator–prey interaction is described by a Holling type II functional response (the predation rate per predator which is a monotonic increasing prey-dependent hyperbolic relationship) where a handling time of the prey introduces feeding saturation. In [7] the predators are assumed to have a heterogeneous spatial distribution (for instance when they form a colony or school). Then the functional response depends on both predator and prey densities in a manner that reflects feeding interference between predators. This leads to a ratio-dependent or Beddington–DeAngelis type of functional response (see also [6]). In [10,21] on the other hand, the prey spatial distribution is heterogeneous giving group defense and the Holling type IV or Monod–Haldane functional response is used. This expression is also only prey-dependent but the function is now not monotonically increasing. The predation rate per predator decreases for larger prey densities. Bate and Hilker [4] note that Holling type IV functional responses usually result in an upper threshold of prey density, beyond which the predator cannot survive. Further, in recent work

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[11] the predators functional response is derived starting from first principles.

Here we study a different formulation with heterogeneous prey spatial distribution on the ground. The prey gather together in herds where only prey individuals that live close to the herds boundary on the ground are subject to hunting by predators. In [1,5,23,25] this feature has been taken into account in ecoepidemiological systems. These, besides ecological situations dealing with demographically interacting populations, consider also a transmissible disease in the system, see [16,24] for an introductory account.

In a parallel paper [8] an ecoepidemiological model in which the epidemics spread among the predators was proposed. Here we investigate a model where the prey are affected by a disease that propagates by contact. With respect to earlier formulations, these models exhibit the feature of feeding satiation, modeled via a Holling type II response function such as in the Rosenzweig–MacArthur model [20]. However, here the prey-dependent hyperbolic relationship is expressed as a function of the "square root" of the prey size instead of the prey size itself. It differs from the herd behavior model presented in [1], because it takes into account the feeding satiation phenomenon also explored in [8]. In the recent paper [4] a similar problem was studied but the predator group-defending prey functional response was the Holling type IV instead of the "square root" functional response.

The paper is organized as follows. In Section 2 we present the ecoepidemic model and the outline the methodology of the study. The two-dimensional models, the epidemic one, with infected prey population, and the purely demographic, i.e. disease-free, predator–prey model, are analysed respectively in Sections 3 and 4. Here we extend the analysis of [8] by a phase portrait analysis to study the total collapse of the system caused by a heteroclinic connection between the two prey-only saddle equilibria.

In Section 5 we move to the analysis of the full model where the prey is affected by the infectious disease, assuming that diseased individuals are left behind by the herd. We start with a classical existence and stability analysis of all equilibria in Sections 5.1 and 5.2. In Section 5.3, the numerical bifurcation analysis is carried out, completed for the special instance of codimension-two bifurcations. In addition to the bifurcations of the classical predator–prey models, i.e. transcritical, tangent (saddle node) and Hopf bifurcations, here also the torus (Neimark–Sacker) bifurcation occurs. A new phenomenon is represented by the abrupt destruction of the quasi-periodic dynamics on a torus similar to what was found in [3,6].

In Section 6 the results of all particular cases will be compared with the results of the ecoepidemic model with the infected predator population, instead of the prey, analysed in [8] and a final discussion concludes the paper. Assuming that the carrying capacity is sufficiently high to support coexistence of prey and predator, due to the weakening of the prey population by infection, the predator feeding on the prey population can persist for higher predators natural mortality rates.

2. Modeling and analysis approach

2.1. The model

We consider the model presented in [23], which we briefly illustrate again here for the convenience of the reader, to better emphasize the changes in that main model. The basic ecological model is an adapted Rosenzweig–MacArthur model first discussed in [19] where both prey and predators have an homogeneous spatial distribution. Mathematically, the consumption rate of the prey by the predator is expressed via a hyperbolic relationship.

In our case the spatial distribution of the prey population, forming a herd and occupying a certain portion of the ground, is heterogeneous. The prey individuals most subject to hunting are those close to the herd boundaries. The area occupied by the herd is proportional to the prey population and therefore to the size of the herd itself. The prey density on the herd perimeter is therefore proportional to the square root of the size of the herd and thus in the hyperbolic relationship of the standard Holling type II term, the prey size is here replaced by a square root of the prey size. The prey population grows logistically in the absence of the predator. In the absence of the prey, the predators die exponentially fast.

In order to model the spread of the disease, the prey population is divided into two classes consisting of healthy and diseased individuals. The latter are assumed to be too weak to reproduce and to compete for resources. Therefore the basic two-population demographic predator–prey model is extended into a three-dimensional predator—susceptible prey—infected prey model. As in the classical two compartmental SI-model the law of mass action is used to formulate the infection rate of the susceptible by infected prey, assuming possible contacts among all the individuals of the herd. The infected prey are assumed to be too weak both to reproduce and to compete for resources, i.e. they do not appear in the logistic reproduction function for the healthy prey. The infected prey are further assumed to drift away from the herd when become infected; this for instance occurs for elephants. But in the process, they are still able to infect other individuals in the herd. Once alone, they can easily be hunted by the predators. In view of the ease of these captures, we assume that the predators never get tired of hunting sick isolated prey individuals, this implying that in this case the hunting term is bilinear, i.e. a mass action term, as in the classical Lotka–Volterra model. On the other hand, as stated above, we assume that they can become satiated by hunting the healthy prey in the herd, observing that this hunt requires more effort than that one on the infected prey. Thus, mathematically, this is better modeled by a Holling type II response function. The predators' different attitudes in the prey capture therefore determine the different choices for the functional responses among healthy and infected prey.

The model where the state variables and parameters are overlined in order to be able to introduce re-scaled versions later, reads

$$\frac{d\overline{R}}{d\tau} = r\overline{R}\left(1-\frac{\overline{R}}{\overline{K}}\right) - \overline{\lambda}\,\overline{R}\,\overline{I} - \frac{\overline{a}\sqrt{\overline{R}}\,\overline{F}}{1+\overline{T}\,\overline{a}\sqrt{\overline{R}}}, \tag{1a}$$

$$\frac{d\overline{I}}{d\tau} = \overline{I}\left(\overline{\lambda}\,\overline{R} - \overline{b}\,\overline{F} - \overline{\mu}\right), \tag{1b}$$

$$\frac{d\overline{F}}{d\tau} = \overline{F}\left(\frac{\overline{e}\,\overline{a}\sqrt{\overline{R}}}{1+\overline{T}\,\overline{a}\sqrt{\overline{R}}} + \overline{e}\,\overline{b}\,\overline{I} - \overline{m}\right). \tag{1c}$$

The system consists of the equation for healthy prey $\overline{R}(\tau)$, reproducing logistically and being subject to the negative effects of hunting as well as to the infection process. The infected prey $\overline{I}(\tau)$ do not reproduce so that they are absent in the logistic growth term in the first equation, nor do they contribute to the population pressure on the susceptible prey, because we assume them to be too weak for that. The spread of the infection is modeled via a bilinear term with rate parameter $\overline{\lambda}$. The disease is unrecoverable, i.e. once entered into this class, an infected individual only exits it by dying at rate $\overline{\mu}$, incorporating natural plus disease-related effects or possibly by predation modeled with the Holling type I functional response with rate parameter $\overline{b}$. Note that here we disregard the possible healthy prey population pressure on the infected prey, i.e. we do not introduce a term of the type $\overline{c}\,\overline{R}\,\overline{I}$ into the second equation, assuming that the mortality is already represented by the linear term. Note also that the infected prey are assumed to be left behind by the herd, so that they are hunted on a one-to-one basis by the predators. Hence, they are also an "easy"