Applied Mathematical Modelling 37 (2013) 4825–4841

Contents lists available at SciVerse ScienceDirect

Applied Mathematical Modelling

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

Predator–Prey model with Holling response function of type II and SIS infectious disease

Jean Jules Tewa [a,d,*], Valaire Yatat Djeumen [b,d], Samuel Bowong [c,d]

[a] Department of Mathematics and Physics, National Advanced School of Engineering (Polytechnic), University of Yaounde I, P.O. Box 8390, Yaounde, Cameroon
[b] University of Yaounde I, Faculty of Science, Department of Mathematics, P.O. Box 812, Yaounde, Cameroon
[c] Laboratory of Applied Mathematics, University of Douala, Department of Mathematics and Computer Science, P.O. Box 24157, Douala, Cameroon
[d] UMI 209 IRD/UPMC UMMISCO, University of Yaounde I, Cameroon GRIMCAPE Team Project, LIRIMA, Yaounde, Cameroon

ARTICLE INFO

Article history:
Received 13 March 2012
Received in revised form 5 September 2012
Accepted 1 October 2012
Available online 12 October 2012

Keywords:
Predator
Prey
Infectious disease
Response function
Bifurcation
Global stability

ABSTRACT

We analyze the influence of a SIS infectious disease affecting Preys or both Predators and Preys in a Predator–Prey model. The response function used here is Holling function type II. Many thresholds are computed and used to investigate the global stability results. The disease can disappear from the community, persist in one or two populations of the community. At least one population can disappear from the community because of disease. In some cases, the model exhibits periodic solutions with persistence of the disease or without disease. Numerical simulations are used with nonstandard numerical schemes to illustrate our results.

1. Introduction

It is well known that Epidemiology and Ecology are two major and distinct fields of research. There are many epidemiological models [1–15] and many ecological models [16–31], but certainly few models of the two fields. The main questions regarding population dynamics concern the effects of infectious diseases in regulating natural populations, decreasing their population sizes, reducing their natural fluctuations, or causing destabilizations of equilibria into oscillations of the population states. With the Holling function response of type II, it is well known that the mortality of Preys due to predation increases as well as the number of Preys decreases and become constant at the end.

There has been many Predator–Prey models with infectious diseases: Anderson and May [16] model in which the pathogen tends to destabilize the Prey-Predator interactions; Hadeler and Freedman model [17] in which the authors considered that Predators could only survive on the Prey if some of the Preys were more easily caught due to being diseased; Venturino model [18] with mass action incidence in which an SI or SIS disease spreads among either the Preys or the Predators or the model given in [19] where he consider similar SI and SIS models with disease spread among the Preys when there is logistic growth of Preys and Predators; Hudson et al., model [21] in which they considered the macroparasitic infections in red grouse and looked at situations in which parasitic infections of Preys made them more vulnerable to predation. It is assumed in [20] that the Predators are infected when swallowing the infected Preys, and that the Preys

* Corresponding author at: Department of Mathematics and Physics, National Advanced School of Engineering (Polytechnic), University of Yaounde I, P.O. Box 8390, Yaounde, Cameroon. Tel.: +237 77 71 13 69; fax: +237 22 23 50 70.
E-mail addresses: tewajules@gmail.com (J.J. Tewa), yatatvalaire@yahoo.fr (V.Y. Djeumen), sbowong@gmail.com (S. Bowong).

0307-904X/$ - see front matter © 2012 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.apm.2012.10.003

are infected by contacting the excrement of the infected Predators. Generally, there are more macroparasitic infections which can affect only Preys, only Predators, Preys and Predators. There are many works of Haque and coworkers [25,26,29] concerning transmissible diseases spreading among the Prey or Predator population in Predator–Prey models or symbiotic communities but although these models have some similarities with our models, there are many differences concerning for example the horizontal incidence (mass incidence or standard incidence) or the presence of disease in the community (Prey, Predator, Prey and Predator). In [30], the authors investigate the effect of delay in a Lotka–Volterra type Predator–Prey model with a transmissible disease in the Predator species.

In the models considered in this paper, the Holling function response of type II is used for interactions between Predators and Preys. The authors of [25] used the same kind of model with standard incidence but the disease was only in the Predator population. The novelties in this paper are: Horizontal incidence follows standard incidence which is more appropriated for large and non constant population; the form of response function when there is a disease with the coefficient θ affecting mortality and recruitment; the dynamics around origin is complicated because of standard incidence and population reaches the origin either by following the axis or in spiral pattern; some numerical analysis are with nonstandard schemes. We also include the possibility that infectious disease can persist in the Predator population and can be acquired by the Predators during the predation process. Moreover, we use a nonstandard numerical scheme for some simulations. It has been proved in [1] that the simulations can be very different using a nonstandard numerical scheme.

2. The model formulation

The Holling function response of type II is defined by $h(H)=\frac{B\omega_0 H}{1+B\omega_1 H}$, where H denotes the Prey population, ω_0 and ω_1 denote respectively the time taking by a Predator to search and capture Preys, B is the predation rate per unit of time. Then, the Predator–Prey model with Holling function of type II if P denotes the Predator population is

$$\begin{cases} \dot{H}(t)=r\left(1-\frac{H}{K}\right)H-g(H,P), \\ \dot{P}(t)=eg(H,P)-\gamma P, \end{cases} \tag{1}$$

where r denotes the intrinsic growth rate of the Preys, K is the carrying capacity of the environment, γ is the mortality rate of Predators, e is the coefficient in conversing Prey into Predator and $g(H,P)=\frac{B\omega_0 HP}{1+B\omega_1 H}$.

When there is no Predator, the dynamics of Prey population is governed by the logistic equation $\dot{H}(t)=r\left(1-\frac{H}{K}\right)H$. The function $g(H,P)$ can also be written as $g(H,P)=\frac{\alpha HP}{1+aH}$, where $\alpha=B\omega_0$ denotes the Prey searching rate, $a=B\omega_1$ denotes the satiety rate of Predators. Setting $\beta=e\alpha$, the System (1) becomes

$$\begin{cases} \dot{H}(t)=r\left(1-\frac{H}{K}\right)H-\frac{\alpha HP}{1+aH}, \\ \dot{P}(t)=\frac{\beta HP}{1+aH}-\gamma P. \end{cases} \tag{2}$$

The SIS compartmental model in epidemiology with standard incidence is given by

$$\begin{cases} \dot{S}=b-\mu S-\lambda\frac{IS}{N}+\sigma I, \\ \dot{I}=\lambda\frac{IS}{N}-\sigma I-\mu I, \end{cases} \tag{3}$$

where S denotes the susceptible population, I the infectious population, σ is the recover rate of infectious individuals to become susceptible such that $N=S+I$ is the total population, μ is the mortality death rate. We assume that all recruitments are in susceptible compartment at a constant rate b; λ is the adequate contact rate between susceptibles and infectious. If $\sigma=0$, the model (3) becomes a simple SI model. The incidence is assumed to be standard incidence.

Our task here is to combine the preceding models (2) and (3), in order to analyze the influence of SIS infectious disease in a Predator–Prey community. The following hypothesis hold in our models.

1. (H1) In the absence of infection and predation, the Prey population grows logistically.
2. (H2) In the presence of infection, the Prey population are divided into two disjoint classes, namely, susceptible population, and infected population.
3. (H3) The mode of disease transmission follows the standard incidence. The disease is spread among the Prey population without Predators for the first model, with Predators for the second model and in the Prey and Predator populations for the third and last model.
4. (H4) The disease is not genetically inherited. The infected population do not recover or become immune.
5. (H5) It is assumed that Predator cannot distinguish the infected and healthy Prey.
6. (H6) We assume that only susceptible Prey is capable of reproducing and contributing to its carrying capacity.