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Mathematical analysis of the global dynamics of an eco-epidemiological model with time delay

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Abstract

In this paper, an eco-epidemiological predator–prey model with time delay representing the gestation period of the predator is investigated. In the model, it is assumed that the predator population suffers a transmissible disease by contact. By analyzing corresponding characteristic equations, the local stability of each of feasible equilibria and the existence of Hopf bifurcations at the disease-free equilibrium, the prey–infected predator equilibrium and the endemic-coexistence equilibrium are established. By means of Lyapunov functionals and LaSalle's invariance principle, sufficient conditions are obtained for the global asymptotic stability of the predator-extinction equilibrium, the disease-free equilibrium, the prey–infected predator equilibrium and the endemic-coexistence equilibrium of the model.

1. Introduction

The effect of disease in ecological system is an important topic from mathematical as well as ecological point of view. Since the pioneering work of Anderson and May [1], great attention has been paid to the modelling and analysis of eco-epidemiological systems recently (see, for example, [2–25]). An increasing number of works are devoted to the study of the relationships between demographic processes among different populations and diseases. Most of these works dealt with predator–prey models with disease in the prey (see, for example, [4,7,11,14, 18–21,24]). Recently, several authors proposed different eco-epidemiological predator–prey models by assuming that the predator population suffers a transmissible disease (see, for example, [6,9,17,21,22,26–28]). Biologically relevant examples were given in Table 2 in [29] for

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the case when a transmissible disease spreads among the predator population. In particular, one could cite rabies and Sarcoptes spp. in foxes (*Vulpis vulpis*) and coyotes (*Canis latrans*), where rabbits are considered as the prey. Other examples are the Phocine Distemper Virus affecting both the common seal (*Phoca vitulina*) and the striped dolphin (*Stenella coeruleoalba*), Otostrongylus circumlitis and Uncinaria lucasi affecting respectively ringed seals (*Phoca hispida*) and northern fur seals (*Callorhinus ursinus*), in these cases the prey are given by fishes. In [17], after reviewing the classical Lotka–Volterra type predator–prey model and SIS epidemic model, Venturino formulated two eco-epidemiological models with disease in the predators and mass action and standard incidence rates. In the two models, it was assumed that the disease spreads among predators only and that the infected individuals do not reproduce, only sound ones do. Analysis of the long-term behavior of solutions of the two models was carried out to show that whether and how the presence of the disease in the predator species affects the behavior of the ecological system, but also whether the introduction of a sound prey can affect the dynamics of the disease in the predator population. Following the work of Venturino [17], in [28], Zhang and Sun considered a predator–prey model with disease in the predator and general functional response. Sufficient conditions were obtained for the permanence of the eco-epidemiological system.

It is well known that time delays of one type or another have been incorporated into mathematical models of population dynamics by many researchers. We refer to the monographs of Gopalsamy [30], Kuang [31] and MacDonald [32] for general delayed biological systems and to Beretta and Kuang [33] and Wangersky and Cunningham [34] and references cited therein for studies on delayed predator–prey systems. In general, delay differential equations exhibit much more complicated dynamics than ordinary differential equations since a time delay could cause a stable equilibrium to become unstable and cause the population to fluctuate. Time delay due to gestation is a common example, because generally the consumption of prey by predator throughout its past history governs the present birth rate of the predator. In view of this fact in [35], Haque et al. considered the following Lotka–Volterra type eco-epidemiological predator–prey model with time delay:

$$\begin{aligned} \dot{x}(t) &= x(t)(r-a_{11}x(t))-a_{12}x(t)S(t)-a_{13}x(t)I(t), \\ \dot{S}(t) &= ka_{12}x(t-\tau)S(t-\tau)-r_1S(t)-\beta S(t)I(t), \\ \dot{I}(t) &= \beta S(t)I(t) + ka_{13}x(t-\tau)I(t-\tau)-r_2I(t), \end{aligned} \tag{1.1}$$

where $x(t), S(t)$ and $I(t)$ represent the densities of the prey (rabbits), susceptible predators (sound foxes) and the infected predators (infected foxes) at time t, respectively. The parameters a_{11}, a_{12}, a_{13}, k, r, r_1, r_2 and β are positive constants. In system (1.1), the following assumptions were made:

(A1) In the absence of predation, the prey population $x(t)$ grows logistically with the intrinsic growth rate $r>0$ and carrying capacity r/a_{11}.

(A2) The total predator population N is composed of two population classes: one is the class of susceptible (sound) predator, denoted by S, and the other is the class of infected predator, denoted by I.

(A3) The disease spreads among the predator species only by contact and the disease can be transmitted vertically, and the disease does not cross the species barrier. The infected predator population do not recover or become immune. The disease incidence is assumed to be the simple mass action incidence βSI, where $\beta>0$ is called the disease transmission coefficient.