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A predator–prey–disease model with immune response in infected prey ☆

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ABSTRACT

In this paper, a predator–prey–disease model with immune response in the infected prey is formulated. The basic reproduction number of the within-host model is defined and it is found that there are three equilibria: extinction equilibrium, infection-free equilibrium and infection-persistent equilibrium. The stabilities of these equilibria are completely determined by the reproduction number of the within-host model. Furthermore, we define a basic reproduction number of the between-host model and two predator invasion numbers: predator invasion number in the absence of disease and predator invasion number in the presence of disease. We have predator and infection-free equilibrium, infection-free equilibrium, predator-free equilibrium and a co-existence equilibrium. We determine the local stabilities of these equilibria with conditions on the reproduction and invasion reproduction numbers. Finally, we show that the predator-free equilibrium is globally stable.

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1. Introduction

Mathematical biologists have been working on merging two major areas of interest Ecology [20,22,25] and Epidemiology [17] for a long time. Diseases that affect the prey in particular may affect the entire predator–prey system [10,19,21]. The pathogen may not infect the predator but it creates a differential pressure on the predator–prey dynamics, causing destabilization of equilibria or reducing natural oscillations. A key objective of these models is to investigate the correlation between the disease and the predator–prey system.

Predator–prey–pathogen models have been a topic of significant interest since the early 1980s. Anderson and May [2] in 1982 paved the way of merging ecological predator–prey models, which were initiated by Lotka and Volterra, and the epidemiological models, introduced by Kermack and McKendrick. Hethcote et al. [18] showed how the parasite population could affect the demographic behavior of the host population. The fusion of ecology and epidemiology is a comparatively new branch of study, now with a 30 year history, known as eco-epidemiology. Eco-epidemiological models could also address scenarios, such as the ones presented by Getz and Pickering in 1983 [12], where a parasitic disease has been found to regulate the host population density. Furthermore Bairagi et al. [3] showed that a disease might be the sole reason for co-existence of the species. They established this result by combining an SI (Susceptible–Infected) disease model with a regular predator–prey N-P(Prey–Predator) model to obtain an eco-epidemiological ODE model.

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In the paper [23] the authors were successful in showing the effect of a predator on the prevalence of the disease in the prey. The simulations in the article show that existence of predator abundance creates oscillations in the SIR model of the prey population. Another scenario proposed by Bairagi et al. [3] suggests that predator may avoid infected prey and predate only on healthy susceptible ones which may lead to prey extinction. Their simulations show the existence of a bifurcation in the model.

Since then the field of eco-epidemiology has been attracting significant attention and many studies have been completed using eco-epidemiological modeling [1,4–9,14,24]. All the studies mentioned above clearly demonstrate the volume of work going at present on the predator–prey dynamics where the prey population is infected. The infection in the prey population has many consequences for predator–prey dynamics and these models need more attention to elucidate the complex dynamics of predator–prey and disease.

Researchers interested in epidemiology have known for a long time that an intrinsic connection exists between the within-host status of the pathogen, its interaction with the immune system, and between-host ability of the pathogen to transmit and invade the host population. Even though there is a large body of literature treating within-host models and epidemiological models separately, it wasn't until the seminal work of Gilhrist and Sasaki [13] that the two fields were connected with an ODE within-host model nested in an epidemiological time-since-infection structured model. Since then the interest toward models linking within-host dynamics with epidemiological dynamics has been rising. Linked models have been also termed immuno-epidemiological models. Bridging the gap between the two fields [16] is now of primary interest not only for models of macro-parasitic infections, but also for micro-parasitic infections, such as HIV.

One area that has not been addressed this far is the impact of within-host dynamical interaction of a pathogen with the immune system and the between-host distribution of the disease in the presence of a predator or a competition between species. There is still a lot to be learned on how ecological interaction of predation or competition affects the within-host dynamics of the pathogen with the immune system, how within-host interactions affect the distribution of disease on population level, and ultimately the predator–prey interaction.

In the present article we discuss a predator–prey PDE model where the prey population is infected by a pathogen. An immunological SI model has been designed to represent the dynamics of the disease inside a host in the prey population. The major goal of this paper is to identify the probable characteristic of the disease and its major role in determining the dynamics of the predator–prey system. We investigate the long term behavior of the predator–prey dynamics in the light of the disease in the prey population. In the next section we introduce a predator–prey model with infected prey structured by immune status. We term these linked models immuno-eco-epidemiological models. In Section 3 we present analysis of the within-host model. In Section 4 we define the equilibria of the immuno-eco-epidemiological model and we investigate their local stabilities. In Section 5 we obtain the global stability of the predator-free equilibrium. Section 6 contains a summary of our results.

2. Model

We consider the following model to represent the predator–prey interaction with the prey in the population being infected. We use an ODE model to describe the within-host parasite dynamics. The virus particles present in a single infected prey over the infection period play an important role that decides the rate of infection among the prey population in a general epidemiological model. The following age-structured PDE model explains the prey–predator interaction in the epidemiological environment. Here $S(t)$ denotes the number of susceptible prey population at time t, $P(t)$ is the number of predators in the population. We use the variable $i(\tau,t)$ to represent the density of infected prey population at time t where τ is the age of infection in the prey population. The model is given as follows.

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - S(t)\displaystyle\int_0^\infty \beta(\tau) i(\tau,t)\,d\tau - \dfrac{a_1 S(t)P(t)}{1+cS(t)} - m_0 S(t), \\ \dfrac{\partial i(\tau,t)}{\partial \tau} + \dfrac{\partial i(\tau,t)}{\partial t} = -m_0 i(\tau,t) - mV(\tau) i(\tau,t) - \alpha(\tau) P(t) i(\tau,t), \\ i(0,t) = S(t)\displaystyle\int_0^\infty \beta(\tau) i(\tau,t)\,d\tau, \\ \dfrac{dP(t)}{dt} = \dfrac{a_2 S(t)P(t)}{1+cS(t)} - dP(t) + kP(t)\displaystyle\int_0^\infty \alpha(\tau) i(\tau,t)\,d\tau, \end{cases} \tag{2.1}$$

where Λ is the birth rate of the susceptible prey population and m_0 is the death rate of the prey population. $\beta(\tau)$ represents the infection rate in prey population with age of infection τ and is related to the number of virus particles $V(\tau)$ by the relation $\beta(\tau) = cV(\tau)$. We use a Holling function of Type II to represent the predation rate for the susceptible population, where a_1 is called the attack rate and the average time spent on processing a food item is called the handling time, given by c/a_1. α denotes the predation rate in the infected class, a_2/a_1 is the conversion efficiency of susceptible prey mass into