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A mathematical study of an eco-epidemiological system on disease persistence and extinction perspective

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ABSTRACT

A prey–predator system with disease in prey is proposed. The proposed system is an extension of the model analyzed by Bhattacharyya and Mukhopadhyay (2011) which did not consider the density of fish population as a dynamic variable which significantly influence the dynamics of the system. The coexistence equilibria of the system is determined and the dynamic behavior of the system is investigated around coexistence equilibria. Incidence rate of the disease is considered as a bifurcation parameter to examine the occurrence of Hopf bifurcation in the neighborhood of the co-existing equilibria. Sufficient conditions are derived for the global stability of the system around coexistence equilibria. Uniform strong persistence of the system is discussed in order to ensure long-term survival of the species. The obtained results are useful to extract the criteria for disease extinction and persistence. Finally, some numerical simulations are given to verify the analytical results, and the system is analyzed through graphical illustrations.

1. Introduction

The study of ecological systems with the influence of epidemiological parameters is termed as eco-epidemiology, a branch of ecology which, particularly, considers the effect of transmissible diseases. It is globally accepted that the interaction between predators and their prey is a complex phenomenon in ecology. The main objective of eco-epidemiological models are centered around the role of infection on species mortality, reduction in reproduction rate, characteristics of contamination, change in population size, spread of epidemic waves, permanence of the disease and global behavior of the infected species. In recent decades, in order to study the influence of disease on an environment where two or more interacting species are present, theoretical ecologist as well as epidemiologist are extensively analyzed the dynamic behavior of eco-epidemic models [1–12].

Recently, Zhang et al. [13] investigated the Hopf bifurcation of a delayed SIS epidemic model with stage structure and non-linear incidence rate. An eco-epidemic system with delay and parasitic infection in the prey has been investigated by Yongzhen et al. [14]. They derived the conditions for asymptotic stability of steady state and estimated the length of delay preserving stability. Biological persistence means the survival of all populations in future. Butler et al. [15] shown that strong

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persistence implies uniform persistence. Mukherjee [16,17] studied the persistence of prey–predator system with disease in prey. He also derived the criteria of impermanence. Freedman [18] discussed a prey–predator system with parasitic infection and obtained condition for persistence. However, Mukherjee [19] modified the conditions for uniform strong persistence obtained by Freedman [18].

Again, the conditions for long term persistence of an infection, whether at a steady level or as a sequence of outbreaks, may be expected to involve other factors. Bartlett [20] introduced the idea of a critical community size for a given disease, below which an isolated population cannot sustain the disease long term. This critical community size will depend primarily on the relation between the timescale of the infection itself and that of the re-growth of susceptible numbers. Maity [21] studied the dynamics of prey–predator system with infected predator. Hilker and Schmitz [22] proved that the predator infection can have a stabilizing effect. An eco-epidemiological mathematical model with treatment and disease infection in both prey and predator population has been investigated by Hugo et al. [23]. A significant number of articles are available on the dynamics of eco-epidemic models [24–29].

Bhattacharyya and Mukhopadhyay [30] formulated an epidemiological model based on the assumption that, in the lake ecosystem, since the plankton species is only a portion of the food for the fish population, the overall fish density depends on the productivity of the lake and does not relate directly with plankton density. From this point of view, it is legitimate to consider the impact of fish on plankton dynamics without taking into account the effect of plankton on the population dynamics of fish. This assumption is contradictory as Carpenter et al. [31], correctly, pointed out that if the abundance of large piscivorous fish is increased in a lake, then the abundance of their prey, zooplanktivorous fish, should decrease, large zooplankton abundance should increase, and phytoplankton biomass should decrease, consequently, zooplankton grazing can in turn have large impacts on phytoplankton communities. Removal of piscivorous fish can change lake water from clear to green by allowing phytoplankton to flourish. For example, Eel River in Northern California, where fish (steelhead and roach) consume fish larvae and predatory insects. These smaller predators prey on midge larvae, which feed on algae. Removal of the larger fish increases the abundance of algae, Power [32]. Also, in Pacific kelp forests, sea otters feed on sea urchins. In areas where sea otters have been hunted to extinction, sea urchins increase in abundance and decimate kelp, Estes and Palmisano [33]. This theory is clearly stimulated the fact that, in the lake ecosystem, the overall fish density depends not only on the productivity of the lake but also relate directly with plankton density. Subsequently, we have extended the system, considered by Bhattacharyya and Mukhopadhyay [30], through considering the density of fish population as a dynamic variable which significantly influence the dynamics of the system.

It may also be noted that persistence addresses the long-term survival of some or all components of a system but Bhattacharyya and Mukhopadhyay [30] did not consider uniform strong persistence of the system though they have extensively studied dynamical behavior of the system around different equilibria. Again, ecological systems with constant parameters are often found to approach a steady state in which the species coexist in equilibrium. But, if parameters used in the model are changed, other types of dynamical behavior may occur and the critical parameter values at which such transitions happen are called bifurcation points. In this context, it is to be noted infected incidence rate plays an important role towards disease dynamics of the system. Thus, we have considered infected incidence rate as a bifurcation parameter and examined the existence of Hopf bifurcation around its coexisting equilibria.

2. Model and its qualitative properties

We consider a prey–predator type system with disease in prey population. The ecological set up of the system is based on the following major assumptions:

- Prey population is divided in two class, susceptible and infected.
- The growth of susceptible prey population is assumed to be logistic with intrinsic growth rate r and carrying capacity K.
- It is considered that disease transmission is direct and follow simple mass action law with infected incidence rate β, in absence of vectors, and the populations, susceptible and infected, are randomly mixed with each other.
- Infected prey population is naturally recovered with recovery rate γ and subsequently added to the susceptible prey population but diseases free state could not be achieved as no control measures of the diseases is considered in the system.
- The infected prey is more likely to be caught than the healthy ones, therefore, it is assumed that the predator population consumes infected prey population only.
- It is further assumed that disease load in the prey population decrease with increasing predation, and the pathogen can even be removed for sufficiently high level of predation. Therefore, it is reasonable to consider a sigmoid functional response which explains the fact that at low densities of infected prey population, the effect of predation is low, but if the infected prey population size increases, predation is then more intensive i.e. the predator is considered to be generalist, Freedman [34].
- We have considered density-dependent mortality rate σP^2 for the predator. This term describes either a self limitation of consumer or the influence of predation. Self limitation can occur if there is some other factor (besides food) which becomes limiting at high population densities. On the other hand predation on consumers can increase as σP^2 due to the fact that the higher consumer densities attract more attention from predators or if consumers become more vulnerable at higher densities (see Ruan et al. [35]).