# Stability Analysis of an Eco-epidemiological Model with Time Delay and Holling Type-III Functional Response*

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**Abstract** In this paper, an eco-epidemiological model with diseases in the predator and Holling type-III functional response is analyzed. A time delay due to the gestation of the predator is considered in this model. By analyzing the corresponding characteristic equations, the local stability of each of feasible equilibria and the existence of Hopf bifurcations at the disease-free equilibrium and the endemic-coexistence equilibrium are established respectively. By using Lyapunov functionals and LaSalle's invariance principle, sufficient conditions are obtained for the global stability of the predator-extinction equilibrium, the disease-free equilibrium and the endemic-coexistence equilibrium respectively. Finally, numerical simulations are performed to illustrate the theoretical results.

**Keywords** Eco-epidemiological model, Time delay, Holling type-III functional response, Stability, Hopf bifurcation.

**MSC(2010)** 34K20, 34K60, 92D25.

## 1. Introduction

Dynamics of predator-prey model is one of important subjects in mathematical ecology, and some important results have been studied and derived by many researchers ( [2, 8, 9, 13, 14, 16, 17, 22]). It is well-known that the effect of disease on ecological system is an important topic. Since the pioneering work of Anderson and May [1], many works have been devoted to the study of the effects of a disease on a predator-prey model recently ( [3, 6, 7, 10, 12, 15, 18–21]). Most of these works dealt with predator-prey models with disease are in the prey. Recently, several authors have proposed different eco-epidemiological predator-prey models by assuming that the predator population suffers a transmissible disease ( [3,6,7,10,15,18,21]). In [10], by assuming that a transmissible disease spreads among the predator population,

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Lu et al., considered the following eco-epidemiological model

$$
\begin{aligned}
\dot{x}(t) &= rx(t) - ax^2(t) - a_1 x(t) S(t), \\
\dot{S}(t) &= a_2 x(t-\tau) S(t-\tau) - r_1 S(t) - \alpha S(t) I(t) + \sigma I(t), \\
\dot{I}(t) &= \alpha S(t) I(t) - (r_2 + \sigma) I(t),
\end{aligned}
\qquad (1.1)
$$

where $x(t)$, $S(t)$ and $I(t)$ represent the densities of the prey population, the susceptible predator and the infected predator at time $t$ respectively. The parameters $a$, $a_1$, $a_2$, $r$, $r_1$, $r_2$, $\alpha$ and $\sigma$ are positive constants. In model (1.1), the following assumptions have been made:

(A1) In the absence of predation, the prey population $x$ grows logistically with the intrinsic growth rate $r$ and carrying capacity $r/a$.

(A2) The total predator population $N(t)$ is divided into two distinct classes: susceptible (sound) predator $S(t)$ and infected predator $I(t)$. Therefore, at any time $t$, the total density of predator population is $N(t) = S(t) + I(t)$.

(A3) The disease spreads among the predator species only by contact, and the disease cannot be transmitted vertically. The disease incidence is assumed to be the bilinear incidence $\alpha SI$, where $\alpha > 0$ is called the disease transmission coefficient. The parameter $\sigma$ represents the recovery rate, i.e., the rate at which the infected predators recover from the disease and become susceptible again.

(A4) Only the susceptible predators have the ability to capture the prey with capturing rate $a_1$, and the infected predator are unable to catch the prey because of a high infection. The ratio $a_2/a_1$ is the conversion rate of nutrients into the reproduction of the susceptible predators by consuming prey. The parameters $r_1$ and $r_2$ are the natural death rate of the susceptible predator and the infected predator respectively. Here, $r_1 \leq r_2$.

(A5) The reproduction of the predator population after consuming prey ia not instantaneous, and a duration of $\tau$ time units elapses when an individual prey is killed and the moment when the corresponding addition is made to the predator population.

In [10], the stability of each of feasible equilibria and the existence of Hopf bifurcations at the disease-free equilibrium and the positive equilibrium are established respectively. By means of Lyaponov functionals and LaSalle's invariance principle [4, 5], sufficient conditions for the global stability of each of the nonnegative equilibria of model (1.1) have been figured out.

In model (1.1), it is assumed that the per capita rate of predation depends on the prey numbers only. However, Holling found that each predator increased its consumption rate when exposed to a higher prey density, and also predator density increased with growing prey density. In 1965, Holling [8] used the following function

$$
p(x) = \frac{x^2}{1 + mx^2}
$$

as one of the predator response functions. It is now referred to as the Holling type-III response function. Holling type-III functional response reveals that the risk of