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A delayed eco-epidemiological system with infected prey and predator subject to the weak Allee effect

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ABSTRACT

We consider a system of delay differential equations to represent predator–prey eco-epidemic dynamics with weak Allee effect in the growth of predator population. The basic aim of the paper is to observe the dynamics of such system under the influence of gestation delay of predator and Allee parameter. We analyze essential mathematical features of the proposed model such as uniform persistence, stability and Hopf-bifurcation at the interior equilibrium point of the system. Global asymptotic stability analysis of the positive equilibrium points by constructing a suitable Lyapunov function for the delayed model is carried out separately. We perform several numerical simulations to illustrate the applicability of the proposed mathematical model and our analytical findings. We observe that the system exhibits chaotic oscillation due to increase of the delay parameter τ. We also observe that there is a threshold of Allee parameter above which the predator population will be washed away from the system.

1. Introduction

The model-based studies on eco-epidemiological systems are now vast after the works of Hadeler and Freedman [1]; and Chattopadhyay and Arino [2], who coined the term eco-epidemiology. On the other hand, the Allee effect named after W. C. Allee [3], has significant contribution in population dynamics. Allee effects mainly classified into two ways: strong and weak Allee effect [4,5]. There is a threshold population level for the strong Allee effect such that the species will become extinct below this threshold population density. However, when the growth rate decreases but remains positive at low population density, is called the weak Allee effect. Like Allee effect, disease is also a basic reason for the species extinction. The interplay between Allee effects and disease has influential biological relevance in nature [6] and demands in-depth research. In both ecology and epidemiology independently; Allee effect is an established concept having well defined biological objectives. The works on eco-epidemiological systems under the influence of Allee effects are few [7–12].

At the same time, one of the most important problems for eco-epidemiological dynamics is to analyze the effect of time delays on the stability of the systems. Time delay is very common phenomena in population dynamics. There have been extensive research activities on the dynamical behaviors, attractive, persistence, periodic oscillation, bifurcation and chaos of population with retarded systems [13–31]. It is well known that most of the biological processes involve time delays and need to be focused for practical purposes. In ecology, the predator must take time delays to digest their food before further activities [32–39]. Therefore, it will be more realistic if one can consider time delays in predator–prey interaction. Some detailed arguments about the importance of time delays in practical models can be found in the classical books [35,36,40]. As far as our knowledge, there are very few works on the time delayed population dynamics in the presence of Allee effect [41–44]. To the best of our knowledge, the present work is the first attempt on the study of an eco-epidemiological system under the influence of weak Allee effects and time delay.

Recently, Sasmal and Chattopadhyay [9] studied a model of predator–prey interaction with infection in prey population and the weak Allee effects on predator population. In this paper, we have considered their model with the more realistic assumption that the predator must take time delays to digest their food before having further activities take place. In addition to the above assumption, we have considered that the predator population captures only infected prey who

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contributes positive growth to the predator population. This assumption is supported by the experimental data of *Lafferty and Morris* [45], where they established that the predation rate on infected population is 31 times higher than the susceptible one.

The rest of the paper is organized as follows: in Section 2 we discuss the development of our model; Section 3 deals with the mathematical analysis of the non-delay model. Mathematical analysis of the delayed model with switching stability behavior is discussed in the Section 4. In Section 5 we discuss the boundedness, uniform persistence and permanence of the delayed model; global stability of the model around planner equilibrium (where susceptible prey coexists with infected prey) and positive interior equilibrium are discussed in Section 6. Numerical experiments with a set of hypothetical parameters are performed to observe the dynamics of the system and are presented in Section 7. The paper ends with a discussion in Section 8.

2. The model

In this paper, we have modified the model studied by Sasmal and Chattopadhyay [9]. It is well known that after consumption of prey, the predator will take some time to digest their food for further activities. We made following two assumptions:

1. We have considered that predator captures only infected prey. This assumption is supported by the experimental data of *Lafferty and Morris* [45]; they estimated that the predation rate on infected fish, on an average, is 31 times higher than the predation rate on susceptible fish. This observation allows us to neglect the predation on susceptible prey by predator. Furthermore, we have assumed that the consumption of infected prey contributes positive growth to the predator population.
2. We have considered that the reproduction of predator population after predating the prey will not be instantaneous, but mediated by some constant gestation time lag $\tau > 0$ of predator. The food chain interaction is constructed by food and feeding relationships between the organisms, thus in this interaction, after predation, some amounts of energy in the form of prey biomass converted into predator biomass. But this process is not simple; the conversion of prey energy to predator energy is not instantaneous, and several processes are involved in this mechanism. The process includes prey biomass enters into predator's digestive system and this digestion process is complicated and time consuming. After digestion, the next process is absorption, and the energy enters into the body. After entering into the predator's body, the absorbed prey food finally transformed into the predators' energy in the form of biomass. The whole transformation process requires time; detailed arguments for time lag due to gestation can be found in the classical books by Gopalsamy [35] and Kuang [36].

Thus, a predator–prey model with susceptible prey, infected prey and predator along with the weak Allee effects and time delay due to gestation time by predator, is given by the following set of nonlinear differential equations:

$$\begin{aligned}\frac{dS}{dt} &= S[1-S-I-\beta I],\\ \frac{dI}{dt} &= I[\beta S - aP - \mu],\\ \frac{dP}{dt} &= \gamma aP(t-\tau)I(t-\tau)\frac{P}{\theta+P} - dP,\\ &= P\left[P(t-\tau)I(t-\tau)\frac{\alpha}{\theta+P} - d\right].\end{aligned} \tag{2.1}$$

It is to be noted here that the infected population does not contribute to the reproduction but compete for resources with susceptible one. We have considered disease transmitted through mass action law and predator follows Holling type *I* functional response [46]. The term $\frac{P}{\theta+P}$ is the Allee effect function (known as the weak Allee function), which is the probability of finding a mate and θ is the individuals searching efficiency [47–49]. The bigger θ is, the stronger the Allee effect, and slower the per capita growth rate of the predator, especially when predator population is small. Here we have assumed that α ($\alpha = a\gamma$) is the total effect to predator by consuming infected prey, where a is the attack rate and γ is the conversion efficiency from infected prey to predator. All the variables and parameters are positive. The variables and parameters used in Model (2.1) are presented in the Table 1.

Table 1
Variables and parameters used in the Model (2.1).

Variables/parameters	Biological meaning
S	Density of susceptible prey
I	Density of infected prey
P	Density of predator
θ	Individuals searching efficiency
β	Rate of infection
a	Attack rate of predator
μ	Death rate of infected prey
γ	Conversion efficiency on infected prey
$\alpha = a\gamma$	The total effect to predator by consuming infected prey
d	Natural death rate of predator
τ	Gestation time period of predator

The initial conditions for the system (2.1) take the form

$$S(\phi) = \psi_1(\phi), \quad I(\phi) = \psi_2(\phi), \quad P(\phi) = \psi_3(\phi), \quad -\tau \le \phi \le 0,$$

where $\psi = (\psi_1, \psi_2, \psi_3)^T \in \mathcal{C}_+$ such that $\psi_i(\phi) \ge 0$ $(i = 1, 2, 3)$, $\forall$ $\phi \in [-\tau, 0]$, and $\mathcal{C}_+$ denotes the Banach space $\mathcal{C}_+([-\tau, 0], \mathbb{R}^3_+)$ of continuous functions, mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$ and denotes the norm of an element ψ in $\mathcal{C}_+$ by

$$\|\psi\| = \sup_{-\tau\le\phi\le 0}\{|\psi_1(\phi)|, |\psi_2(\phi)|, |\psi_3(\phi)|\}.$$

For biological feasibility we further assume that, $\psi_i(0) > 0$, for $i = 1, 2, 3$.

3. Mathematical analysis of the system (2.1) with no time delay

We first study the system (2.1) with no time lag. The system (2.1) without delay for gestation of predator can be written as

$$\begin{aligned}\frac{dS}{dt} &= S[1-S-I-\beta I],\\ \frac{dI}{dt} &= I[\beta S - aP - \mu],\\ \frac{dP}{dt} &= P\left[\alpha I\frac{P}{\theta+P} - d\right].\end{aligned} \tag{3.1}$$

It is easy to check that the system (3.1) has the following boundary equilibria:

$$E_0 = (0,0,0), \quad E_1 = (1,0,0), \quad E_2 = \left(\frac{\mu}{\beta}, \frac{1-\frac{\mu}{\beta}}{1+\beta}, 0\right).$$

The system (3.1) has interior attractor $E^* = (S^*, I^*, P^*)$, where $I^* = \frac{d(a\theta+\beta S^*-\mu)}{\alpha(\beta S^*-\mu)} = \frac{1-S^*}{1+\beta}$, $P^* = \frac{1}{a}(\beta S^* - \mu)$ and S^* is the roots of the quadratic equation

$$[\alpha\beta](S^*)^2 + [d\beta(1+\beta) - \alpha\mu - \alpha\beta]S^* + [\alpha\mu + d(1+\beta)a\theta - d\mu(1+\beta)] = 0.$$

The interior equilibria exists if S^* is a positive root of the above equation and satisfies $S^* > \frac{\mu}{\beta}$.

Proposition 3.1 (Local stability of equilibria for the Model (3.1)). *The local stability of equilibria of the Model (3.1) is summarized in* Table 2.