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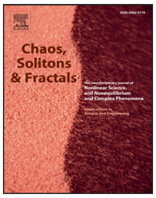

Complexity in a predator-prey-parasite model with nonlinear incidence rate and incubation delay

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ABSTRACT

This paper is concerned with a predator-prey-parasite model with nonlinear infection rate and incubation delay. To explore the system dynamics, we study the distribution of roots of the characteristic equation of the Jacobian matrix of the system which has delay-dependent coefficients. The dynamics displayed by the system can exhibit some of the key features observed in the natural systems, such as appearance and disappearance of cycles in succession. It is shown that the switching phenomenon between stable coexistence and oscillatory coexistence of interior equilibrium as well as of predator-free equilibrium is an interplay of three system parameters, viz. the crowding coefficient, the prey consumption rate and the length of incubation delay. We also discuss the stability of the delayed-system when the non-delayed system is assumed to be unstable.

1. Introduction

In recent times, theoretical biologists are paying more interest to study ecoepidemiological models which integrate an epidemic model with a predator-prey model [1]. This is due to the fact that parasites have the ability to alter the quantitative dynamics of their host population and can even send host population to extinction in the worst case. Predators, on the other hand, can greatly influence the population dynamics of hosts and parasites by consuming host population [2].

One of the most important task in epidemic models to express the disease transmission term mathematically. In most epidemic models, it is assumed that spread of disease occurs following the law of mass action. If $S(t)$ and $I(t)$ are the densities of susceptible and infectious populations at time t then, following mass action law, the rate of new infections (or incidence rate) at any time t is defined by $\beta(t) = \lambda g(I)S$ with $g(I) = I$. The proportionality constant λ is called disease transmission coefficient. This mass action law (or bilinear law) has some unrealistic features, viz., the function $g(I)$ becomes unbounded when I is large [3]. Liu et al. [4] argued for nonlinear incidence rate and proposed a saturated nonlinear function for $g(I)$, viz. $g(I) = \frac{\lambda I^p}{1+bI^q}$, where p, q are positive constants and b is a nonnegative constant. Here λI^p measures the infection force of the disease and $\frac{1}{1+bI^q}$ measures the inhibition effect from the behavioral change of the infectious individuals when their number increases, or from the crowding effect of the infectious individuals. Thus, the incidence rate takes the form $\beta(t) = \frac{\lambda I^p S}{1+bI^q}$. Assuming $p = 1$ and $q = 1$, Capasso and Serio [5] studied the cholera epidemic with $\beta(t) = \frac{\lambda SI}{1+bI}$. It is worth mentioning that crowding effect is negligible when I is small and the two infection rates, in this case, become equivalent. Later on many other authors [4–7] considered this incidence rate to study the dynamics of different epidemic models.

Different models were proposed and investigated for predator-prey interaction in presence of infection and delay. Sun et al. [8] introduced predator's reproduction delay in the ecoepidemiological model of Chattopadhyay and Arino [9]

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and studied the model

$$\begin{aligned} \dot{S} &= rS\left(1-\frac{S+I}{K}\right)-\lambda IS, \\ \dot{I} &= \lambda IS - mIP - \mu I, \\ \dot{P} &= \alpha mI(t-\tau)P(t-\tau)-cP, \end{aligned} \tag{1}$$

where the state variables *S, I* and *P* represent, respectively, the susceptible prey, infectious prey and predator densities at time *t*. The model assumes that the prey population grows logistically with intrinsic growth rate *r* and carrying capacity *K*. Infection spreads from infectious prey to susceptible prey by contact following mass action law with λ as the disease transmission coefficient. Infected populations unable to reproduce but contribute to carrying capacity. It is a general observation that infected preys are more susceptible to predation as they are weakened due to infection and cannot escape predation easily [10]. In their field experiments, Lafferty and Morris [11] estimated that parasitized fish are 31 times more vulnerable to predation than non-parasitized fish. It was assumed, therefore, predator consumes infectious prey only following Type I response function with attack rate *m*. Death rates of infectious prey and predator populations are represented by μ and *c*, respectively. The parameter α $(0 < \alpha < 1)$ represents the conversion efficiency of predator and $\tau \geq 0$ is the predator's reproduction delay. All parameters are assumed to be positive. Bairagi [12] replaced the predation term *mIP* by more realistic Holling Type II form $\frac{mIP}{a+I}$, *a* being the half-saturation constant, and studied the local stability of the ecoepidemic model

$$\begin{aligned} \dot{S} &= rS\left(1-\frac{S+I}{K}\right)-\lambda IS, \\ \dot{I} &= \lambda IS - \frac{mIP}{a+I} - \mu I, \\ \dot{P} &= \frac{m\alpha I(t-\tau)P(t-\tau)}{a+I(t-\tau)}-cP. \end{aligned} \tag{2}$$

In an another study, Bairagi et al. [13] considered the delay in the disease transmission term and investigated the model

$$\begin{aligned} \dot{S} &= rS\left(1-\frac{S+I}{K}\right)-\lambda IS, \\ \dot{I} &= \lambda I(t-\tau)S(t-\tau) - \frac{mIP}{a+I} - \mu I, \\ \dot{P} &= \frac{m\alpha IP}{a+I}-cP. \end{aligned} \tag{3}$$

The common observation of these delay-induced models is that stability switch and the Hopf bifurcation occurs when delay crosses some threshold value. All these models assume that disease spreads from infectious prey to susceptible prey following mass action or bilinear law. However, there are valid reasons for considering incidence rate as nonlinear instead of bilinear [14]. Mass action law does not saturate and becomes unbounded when *I* becomes large. It also does not consider the inhibition effect from the behavioral change of the susceptible individuals or from the crowding effect of the infectious individuals [5]. We, therefore, replace the bilinear disease transmission term λSI in (3) by the nonlinear term $\frac{\lambda SI}{1+bI}$. Note that it includes the crowding effect of the infectious individuals and prevents the unboundedness of the contact rate for positive *b*.

Here we extend the ecoepidemiological model (3) with nonlinear incidence and incubation delay. To incorporate the intracellular phase of pathogen's life-cycle, we assume that the newly infected prey becomes productively infectious after the effective contact between susceptible and infectious preys by a constant delay τ. The recruitment of actively infectious preys at time *t* is then given by the number of preys that were newly infected at time $t-\tau$ and are still alive at time *t*. If we assume a constant death rate β for infected prey but not yet infectious, the probability of surviving the time period from $t-\tau$ to *t* is $e^{-\beta\tau}$. The system (3) in this case reads as

$$\begin{aligned} \dot{S} &= rS\left(1-\frac{S+I}{K}\right)-\frac{\lambda SI}{1+bI}, \\ \dot{I} &= e^{-\beta\tau}\frac{\lambda S(t-\tau)I(t-\tau)}{1+bI(t-\tau)} - \frac{mIP}{a+I} - \mu I, \\ \dot{P} &= \frac{m\alpha IP}{a+I}-cP. \end{aligned} \tag{4}$$

We show that the predator–prey–parasite system (4) with saturated nonlinear incidence rate and incubation delay can exhibit stable and cyclic behavior in succession.

The paper is arranged as follows. We present some preliminary results and existence of equilibrium points in the next section. Stability analysis of the system is performed in Section 3. Permanence of the system is shown in Section 4. Section 5 is devoted for extensive numerical computations. Finally, a summary is presented in Section 6.

2. Preliminary results

We first show that the delay-induced system (4) is well-posed with some initial conditions. Let $C = C([-\tau, 0], \mathbb{R}^3_+)$ be the Banach space of continuous functions, mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$ with norm

$$\| \phi \| = sup_{-\tau \leq \theta \leq 0}\{|\phi_1(\theta)|, |\phi_2(\theta)|, |\phi_3(\theta)|\},$$

where $\phi = (\phi_1, \phi_2, \phi_3)$. The initial conditions of the model (4) are given by

$$\begin{aligned} S(\theta) &= \phi_1(\theta) > 0,\ I(\theta) = \phi_2(\theta) > 0, \\ P(\theta) &= \phi_3(\theta) > 0, \theta \in [-\tau, 0], \end{aligned} \tag{5}$$

where $(\phi_1(0), \phi_2(0), \phi_3(0)) \in C$. Following fundamental theory of functional differential equations [15], the system (4) has a unique solution with initial conditions (5).

We now show that solutions of the system (4) are positive for all $t > 0$. From the first equation of (4), we have

$$S(t) = S_0 e^{\int_0^t \{r[1-\frac{S(\nu_1)+I(\nu_1)}{K}]-\frac{\lambda I(\nu_1)}{1+bI(\nu_1)}\}d\nu_1}. \tag{6}$$

Again, the third equation of (4) gives

$$P(t) = P_0 e^{\int_0^t [\frac{m\alpha I(\nu_2)}{a+I(\nu_2)}-c]d\nu_2}. \tag{7}$$

It implies that $S(t) > 0$ and $P(t) > 0$ for all $t > 0$ whenever $S(0) > 0$ and $P(0) > 0$. We also claim that $I(t) > 0$ for all $t > 0$. If not, then there exists $t_1 > 0$ and $\epsilon > 0$ such that $I(t_1) > 0$ for $t < t_1$, $I(t) = 0$ for $t = t_1$ and $I(t) < 0$ when $t \in [t_1, t_1 + \epsilon)$. From the second equation of (4), we have