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Effect of delay in a Lotka–Volterra type predator–prey model with a transmissible disease in the predator species

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ABSTRACT

We consider a system of delay differential equations modeling the predator–prey ecoepidemic dynamics with a transmissible disease in the predator population. The time lag in the delay terms represents the predator gestation period. We analyze essential mathematical features of the proposed model such as local and global stability and in addition study the bifurcations arising in some selected situations. Threshold values for a few parameters determining the feasibility and stability conditions of some equilibria are discovered and similarly a threshold is identified for the disease to die out. The parameter thresholds under which the system admits a Hopf bifurcation are investigated both in the presence of zero and non-zero time lag. Numerical simulations support our theoretical analysis.

1. Introduction

Population biology has its roots in theoretical ecology. By its very nature it is a science that focuses on understanding, explaining, and predicting changes in the size of populations. The dynamics of the biological populations are captured by mathematical systems that are mainly expressed, when modeling continuous situations, by differential equations. Mathematical models render precise theoretical arguments assessing the factors affecting the populations' rate of change. In addition, analysis of such model enables forecasting the profound economic implications of renewable resources management. They may also suggest successful strategies for biological control. In the last few decades a significant number of predator–prey models have been proposed and extensively studied. In the natural world, however species do not exists alone, and moreover are subject to diseases, which are contracted by interactions with the environment. In addition to purely epidemiological models, for which some standard classical references are [1,6], in the past twenty years also systems combining demographic as well as epidemic aspects have been proposed. Systems of this type are now known as ecoepidemic models. Several such studies are reviewed in a number of recent publications [10,14,17,24]. The importance of parasites influence on the dynamics of plant as well as animal populations is nowadays Dobson and Crawley recognized Dobson and Crawley [7]. It is recognized that viruses, bacteria and parasites make their hosts more vulnerable to predation; see the references in Beltrami and Carroll [2]. Originated from Venturino [25,26], some more recent studies in this field are Haque and Venturino [19], Haque et al. [20], Hethcote et al. [21], and Haque and Greenhalgh [16]. Most of the proposed models are based on the assumption that the infection affects the prey. Only in Venturino [27], Haque and Venturino [18], and Haque [15], has the case of a disease spreading among the predators been considered.

Many natural and man made processes in biology and medicine are better modeled using time delays, for some sample references we refer to MacDonald [23], Gopalsamy [11], Kuang [22], and Beretta and Kuang [3]. Since time delays occur in almost every situation, is not realistic to ignore them. In particular, Kuang [22] observes that animals take time to digest food, and this delays their further activities. Thus dynamical models without delays are a worse approximation of reality than those incorporating time lags. In view of this fact, in the current work, we consider a Lotka–Volterra type predator–prey ecoepidemic model. We assume the transmissible disease to affect the predators, and in this species we also incorporate a delay in gestation of the newborns. This represents the first attempt of considering the effects of delay for ecoepidemic models with disease in the predators.

We determine threshold conditions for which the epidemic in the predator population will be eradicated. The main results of

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our investigation are the stability and bifurcations related to the two most important equilibria of the ecoepidemic system, namely the endemic equilibrium, and the disease-free one, which represents just the coexistence equilibrium of the underlying demographic model. We also discuss permanence of the ecosystem.

The paper is organized as follows. In Section 2 we state the model in consideration and the hypotheses on which it is formulated. Section 3 contains some preliminary results, mainly boundedness of the solutions for the system both with and without delays. Then in Section 4 the model with no delay is analyzed, identifying its equilibria, giving conditions for their feasibility and stability and discussing the permanence of the ecosystem. The same issues are studied in Section 5 for the delayed model, focusing mainly on the two most relevant equilibria, the disease-free one and the coexistence. Simulations are reported in Section 6, and a final discussion concludes the paper, Section 7.

2. Basic assumptions and model formulation

To formulate our model, we make the following assumptions.

(A1) The disease spreads among the predator species only by contact. The disease can be transmitted vertically i.e., passing a disease from parent to offspring, though, but at a later stage in Section 5 we will drop this assumption. This implies that the total predator population $n(t)$ consists of susceptible $y(t)$ and infected $z(t)$ predators, i.e. $n(t) = y(t) + z(t)$.
(A2) In the absence of predators, the prey population $x(t)$ grows logistically with the intrinsic growth rate $r > 0$ and carrying capacity r/B, in which B measures intraspecific competition of the prey.
(A3) The infected predator population $z(t)$ cannot recover. Their total death rate $\delta > 0$ encompasses natural and disease-related mortality.
(A4) The disease incidence follows the simple law of mass action.
(A5) The sound and infected predators hunt the prey with different searching efficiencies, denoted respectively by m and pm, with $0 < p < 1$. This is due to the fact that sound predators are more efficient to catch the prey than the infected ones, weakened by the infection.
(A6) The sound predator population's birth rate b, due to other available food sources, and death rate d give a net reproduction function $\mu = b - d \in \mathbf{R}$. The dynamical behavior of the system for the two possibilities will be discussed separately.
(A7) Reproduction of the predator population after predations is not instantaneous, but delayed by a constant time lag $\tau > 0$ [28,31], due to gestation. The conversion factor of a consumed prey into a sound predator is e $(0 < e < 1)$.

With the above assumptions, our model takes the form in which all parameters but μ are assumed to be positive:

$$\frac{dx}{dt} = rx\left(1 - \frac{x}{r/B}\right) - mxy - pmxz = rx - Bx^2 - mxy - pmxz, \quad (2.1)$$

$$\frac{dy}{dt} = emx(t-\tau)y(t-\tau) - ayz + \mu y(t-\tau), \quad (2.2)$$

$$\frac{dz}{dt} = azy + empx(t-\tau)z(t-\tau) - \delta z, \quad (2.3)$$

with the initial conditions $\phi = (\phi_1, \phi_2, \phi_3)$ defined in the Banach space

$$C_+ = \{\phi \in C([-\tau, 0], \mathbf{R}^3_+) : \phi_1(\theta) = x(\theta), \phi_2(\theta) = y(\theta), \phi_3(\theta) = z(\theta)\},$$

where $x(\theta) > 0$, $y(\theta) > 0$, $z(\theta) > 0$, $\theta \in C[-\tau, 0]$ are given functions.

The model (2.1)–(2.3) presented above, in absence of time lags bears some resemblance with the earlier one [27], containing also only quadratic nonlinearities for the interaction terms, but in which the epidemics is of SIS type. However, here predators do not have other food sources nor do they feel the population pressure of the other individuals of the same population. A number of situations in which possible predators are affected by various diseases are outlined in [12]. Here we mention just the following pairs of parasites affecting hosts: rabies and foxes, *Vulpes vulpes*; Sarcoptes spp. affecting both foxes and coyotes, *Canis latrans*; *Yersinia pestis* and the Prairie dog, Cynomys spp.; *Stomoxys calcitrans* and *Panthera leo*; *Aeromonas hydrophila* and *Alligator mississippiensis*; in the marine environment we mention Phocine distemper virus affecting both the common seal, *Phoca vitulina* and the striped dolphin, *Stenella coeruleoalba*.

2.1. Existence and positive invariance

For $t > 0$ letting, $X \equiv (x, y, z)^T, F : C_+ \to \mathbf{R}^3_+$, $F = (F_1, F_2, F_3)^T$, the system (2.1)–(2.3) can be rewritten as $\dot{X} = F(X)$. Here $F_i \in C^\infty(\mathbf{R}_+)$, for $i = 1, 2, 3$. $F_1 = rx - Bx^2 - mxy - pmxz$, $F_2 = emx(t-\tau)y(t-\tau) - ayz + \mu y$, $F_3 = ayz + empx(t-\tau)z(t-\tau) - \delta z$. For $\theta \in [-\tau, 0)$, let $X(\theta) = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C_+$ and $\phi_i > 0$, $i = 1, 2, 3$, are given functions. Since the vector function F is a smooth function of the variables (x, y, z) in the positive octant $\Omega^0 = \{(x, y, z) : x > 0, y > 0, z > 0\}$, local existence and uniqueness of the system's solution hold.

Whenever $X(\theta) \in C_+$ such that $X_i = 0$, letting $X_t \equiv X(\theta + t)$, then it is easy to check that

$$F_1(x)|_{x=0, X_t \in C_+} \geqslant 0, \quad F_2(x)|_{y=0, X_t \in C_+} \geqslant 0, \quad F_3(x)|_{z=0, X_t \in C_+} \geqslant 0.$$

Any solution of the system (2.1)–(2.3) with $X(\theta) \in C_+$, say $X(t) = X(t, X(\theta))$, implies that $X(t) \in \mathbf{R}^3$ for all $t > 0$, [30].

3. Some preliminary results

3.1. Boundedness of the system with zero time lag

Boundedness is a necessary condition for the system (2.1)–(2.3) to be biologically realistic. The following propositions ensure the boundedness of the system (2.1)–(2.3) in the limiting case $\tau = 0$.

Proposition 3.1. *The prey population is always bounded from above.*

Proof. From (2.1) the following inequalities follow

$$\frac{dx}{dt} \leqslant rx - Bx^2 = rx\left(1 - \frac{x}{r/B}\right), \quad \limsup_{t \to +\infty} x(t) \leqslant \frac{r}{B}. \qquad \square$$

Proposition 3.2. *For $\mu < 0$, all solutions of (2.1)–(2.3) starting in Ω^0 are uniformly bounded with an ultimate bound.*

Proof. Define a function $\chi = ex + y + z$. Taking its time derivative along the solutions of (2.1)–(2.3), as $x(t) \leqslant r/B$, for each $\min\{-\mu, \delta\} > \phi > 0$, the following inequality holds

$$\frac{d\chi}{dt} + \phi\chi \leqslant ex(r + \phi - Bx) + (\phi + \mu)y + (\phi - \delta) \leqslant ex(r + \phi - Bx)$$
$$= eBx\left\{\frac{(r+\phi)}{B} - x\right\} \leqslant e\frac{(r+\phi)^2}{4B} \equiv \rho.$$

Integrating the differential inequality; see Birkhoff and Rota [4], we find

$$\chi(t) \leqslant e^{-\phi t}\chi(t_0) + \frac{\rho}{\phi}(1 - e^{-\phi t})$$
$$\leqslant \max\left(\chi(0), \frac{\rho}{\phi}\right), \quad \limsup_{t \to +\infty} \chi(t) \leqslant \frac{\rho}{\phi}, \quad (3.1)$$