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Stability and bifurcation of a predator–prey model with disease in the prey and temporal–spatial nonlocal effect

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ABSTRACT

In this paper, we consider the dynamics of a predator–prey model with disease in the prey and ratio-dependent Michaelis–Menten functional response. The model is a reaction–diffusion system with a nonlocal term representing the temporal–spatial weighted average for the prey density. The limiting case of the system reduces to the Lotka–Volterra diffusive system with logistic growth of the prey. We study the linear stability of the two non-trivial steady states either with or without nonlocal term. The bifurcations to three types of periodic solutions occurring from the coexistence steady state are investigated for two particular kernels, which reveal the important significance of temporal–spatial nonlocal effects.

1. Introduction

In this paper, we are interested in the following reaction–diffusion system having ratio-dependent Michaelis–Menten functional response with disease in the prey:

$$\begin{cases} u_t - \triangle u = u[1+\alpha(u+v) - (1+\alpha)G**(u+v)] - \beta uv, \\ v_t - \triangle v = \beta uv - av - \frac{lwv}{mw+v}, \\ w_t - \triangle w = -dw + \frac{blwv}{mw+v}, \end{cases} \tag{1.1}$$

where $G**(u+v)$ is defined by

$$(G**(u+v))(\mathbf{x},t) := \int_{\mathbb{R}^n}\int_{-\infty}^{t} G(\mathbf{x}-\mathbf{y}, t-s)(u(\mathbf{y},s)+v(\mathbf{y},s))\ \mathrm{d}s\ \mathrm{d}\mathbf{y},\ (\mathbf{x},t)\in\mathbb{R}^n\times(0,\infty).$$

We assume that the kernel G satisfies

(G) $G\in L^1(\mathbb{R}^n\times[0,\infty))$, $tG\in L^1(\mathbb{R}^n\times[0,\infty))$, $\int_{\mathbb{R}^n}\int_0^\infty G(\mathbf{x},t)\ \mathrm{d}t\ \mathrm{d}\mathbf{x}=1$ $(n\geq 1)$. Furthermore, $G=G(r,t)\geq 0$, where $r=|\mathbf{x}|$. That is, the nonlocal effect depends only on the distance, and not the direction, of $\mathbf{y}$ from $\mathbf{x}$.

There are two species in the model: the prey $u+v$ and the predator w. Throughout this paper, we make the following assumptions.

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(A1) In the presence of a disease, we assume the total prey population is composed of two population classes: one being the susceptible prey (u), and the other being the infected prey (v).

(A2) Only susceptible prey u is capable of reproducing, i.e., the infected prey v is removed by death (with death rate $a \geq 0$) or by predation before having the possibility of reproducing. However, the infected prey v still contributes to population growth of the susceptible prey u, according to a logistic growth law.

(A3) The disease is transmitted only within the prey population and the disease is not genetically inherited. The infected populations do not recover or become immune. The infection follows the mass action mechanism, where $\beta \in (0, 1)$ is called the transmission coefficient. Therefore, the *SI* epidemic model yields:

$$\begin{cases} u_t - \triangle u = u(1-u-v) - \beta uv, \\ v_t - \triangle v = \beta uv - av. \end{cases}$$

(A4) The predator catches only the infected prey with a ratio-dependent Michaelis–Menten functional response function (please see [1] for more details of this response function)

$$\eta(v, w) = \frac{lwv}{mw + v} \qquad (m > 0).$$

Here, the predator has a death rate $d > 0$, and the rate of translating the prey population into the predator population is $b \in (0, 1)$.

For more explanation of "the predator catching only the infected prey" in (A4), one can refer to [2].

The following lemma describes the existence of non-negative equilibria of model (1.1).

Lemma 1.1. *The following results about the non-negative equilibria of model (1.1) hold:*

(1) *the model (1.1) has the trivial equilibrium* $E_0 = (0, 0, 0)$ *and the disease-free equilibrium* $E_{10} = (1, 0, 0)$ *in the absence of predator;*

(2) *if* $\beta > a$, *then there exists a semi-trivial solution in the absence of predator*

$$E_{20} = \left(\frac{a}{\beta}, \frac{\beta - a}{\beta(1+\beta)}, 0\right);$$

(3) *if* $bm(\beta - a) > bl - d > 0$, *then there exists a unique positive equilibrium* $E^* = (u^*, v^*, w^*)$, *where*

$$u^* = \frac{a}{\beta} + \frac{bl - d}{bm\beta}, \quad v^* = \frac{1 - u^*}{1 + \beta}, \quad w^* = \frac{bl - d}{dm} v^*.$$

Note that under the condition $bm(\beta - a) > bl - d > 0$, there exist four non-negative equilibria for the system (1.1). Furthermore, it leads to $0 < u^* < 1$.

Gourley and Britton [3] considered a predator–prey reaction–diffusion system:

$$\begin{cases} u_t - D\triangle u = u[1 + \alpha u - (1+\alpha)G * *u] - uv, \\ v_t - \triangle v = av(u - b), \end{cases} \tag{1.2}$$

where u and v represent the densities of prey and predator respectively, in which they carried out a linear stability analysis for each of the three steady states of system (1.2). Moreover, they investigated the bifurcations from the coexistence steady state with some particular forms of G, with α as bifurcation parameter.

The main object of the present paper is to generalize (1.2) to the predator–prey system (1.1) and investigate the dynamical properties of (1.1). As far as we know, there are no any similar results published for this kind eco-epidemic model with nonlocal reaction in the whole space and the infinite delay in the existing literatures, and thus the results here are novel. Note that the term $\alpha(u + v)$ in (1.1) with $\alpha > 0$, represents an advantage to the prey species in local aggregation. Okubo [4] has described in detail some of the reasons for animal aggregation in nature, and the behaviors of the animals within the groups they have formed (also see Britton [5]). On the other hand, the term $-(1+\alpha)G * *(u + v)$ with $\alpha > -1$, represents intra-specific competition of the prey for resources. This term involves a temporal convolution and therefore introduces delay effect into the system, because of the need to consider the regeneration time of resources. The convolution in space then arises because of the fact that the populations are always moving by diffusion (random movement), and have not been at the same position in space at the previous times. Thus intra-specific competition for resources depends not simply on population density at one point for space and time, but on a weighted average involving values at all previous times and at all positions in space. Based on biological background, the quantity α should be positive. However, the mathematical analysis does not require such an assumption so we will in fact make no constraint on the sign of α in this paper. In Section 5, we shall give a discuss on the above mentioned nonlocal effects both on delay and space convolution imposed by α.