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Efficient numerical methods for spatially extended population and epidemic models with time delay

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ABSTRACT

Reaction–diffusion models with time delay have been widely applied in population biology as well as epidemiology. This type of models can possibly exhibit complex dynamical behaviors such as traveling wave, self-organized spatial pattern, or chaos. Numerical methods play an essential role in the study of these dynamical behaviors. This paper concerns the finite element approximation for reaction–diffusion models with time delay. Two fully discrete schemes and corresponding a priori error estimates are derived. Generally, the research on evolution of population and epidemic needs to survey long-time dynamical behaviors of these models, so that it is important to improve the speed of numerical simulation. To this end, interpolation technique is used in our schemes to avoid numerical integration of reaction term. An outstanding advantage of using interpolation of reaction term is that it improves the operation speed greatly, meanwhile does not reduce convergence order. Applications are given to some model problems arising from population biology and epidemiology. From these simulations some interesting phenomena can be found and we try to explain them in biological significance.

1. Introduction

Populations are living in a spatial world, a large number of fundamental elements of population biology and epidemiology, ranging from individual behavior to species abundance, diversity, and population dynamics, exhibit spatial variation. For instance, population density will depend upon space in a spatially heterogeneous environment. Populations may be structured by spatial location, one way of including spatial dependence in a population model is to allow motion of individuals in the population, describing the population size as a function of time and spatial location under some specific assumptions on the nature of the motion of individuals. Since population size is now a function of more than one variable (a time variable and a number of spatial variables, the number depending on the dimension of the space in which individuals move), this approach will lead to partial differential equation (PDE) models. These equations are often of reaction–diffusion type, with a reaction term corresponding to the (local) population dynamics and a diffusion term describing the motion of individuals in space [6]. As a powerful tool for describing spatial and temporal dynamics, reaction–diffusion models have been widely applied in population biology as well as epidemiology.

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In population biology, the interaction of several populations, such as predator–prey, competition, symbiosis, is important and interesting. Understanding the dynamics of corresponding reaction–diffusion models will be very helpful for investigating multiple species interactions and maintaining ecological balance. Take a predator–prey model for example, assume that the local growth of the prey is logistic and that the predator shows Holling type II functional response, we arrive at Eq. [26]

$$\begin{cases} \dfrac{\partial u}{\partial t} = d_1 \Delta u + u(1-u) - \dfrac{uv}{u+\alpha}, \\ \dfrac{\partial v}{\partial t} = d_2 \Delta v + \dfrac{\beta uv}{u+\alpha} - \gamma v, \end{cases} \tag{1.1}$$

where $u(x, t)$ and $v(x, t)$ stand for the densities of prey and predator, respectively. The positive constants d_1 and d_2 denote diffusion coefficients, $\Delta = \sum_{i=1}^{d} \partial^2/\partial x_i^2$ is the Laplacian operator, the parameters α, β, γ are real and strictly positive. There is a large body of related work with respect to the above predator–prey system (see, e.g., [12,13,28,38,44]).

To capture the impact of spatial heterogeneity of environment and movement of individuals on the persistence and extinction of a disease, epidemic models governed by reaction–diffusion equations are proposed. For instance, a space-dependent SIS epidemic model with standard incidence rate is [2]

$$\begin{cases} \dfrac{\partial S}{\partial t} = d_S(x)\Delta S - \dfrac{\beta(x)SI}{S+I} + \gamma(x)I, \\ \dfrac{\partial I}{\partial t} = d_I(x)\Delta I + \dfrac{\beta(x)SI}{S+I} - \gamma(x)I, \end{cases} \tag{1.2}$$

where $S(x, t)$ and $I(x, t)$ denote the density of susceptible and infected individuals at location x and time t, $d_S(x)$ and $d_I(x)$ are positive diffusion coefficients for the susceptible and infected populations, and $\beta(x)$ and $\gamma(x)$ are positive Hölder continuous functions that represent the rates of disease transmission and recovery at x, respectively. Reaction–diffusion epidemic models have been studied by a number of authors (see, e.g., [4,8,9,34,36,42]).

During the evolution of population and the spread of infectious disease, the population density may be affected by the present as well as the past state. If this effect is taken into consideration then the density functions are governed by a coupled system of reaction–diffusion equations with time delay. Factors that introduce time delay may include age structure of the population (influencing the birth and death rates), maturation periods, incubation periods, food storage or ingestion delays, resource regeneration times, and so on [11]. For example, a space-dependent Holling-Tanner predator–prey model with discrete time delay which we have studied in [10] takes the form

$$\begin{cases} \dfrac{\partial u}{\partial t} = d_1 \Delta u + u(1-u) - \dfrac{uv}{u+\alpha}, \\ \dfrac{\partial v}{\partial t} = d_2 \Delta v + v\left(\gamma - \dfrac{\beta v(x, t-\tau)}{u(x, t-\tau)}\right), \end{cases} \tag{1.3}$$

where τ is the time delay. Literature on spatially extended population and epidemic models with discrete time delay may also be listed in a catalogue (see, e.g., [5,17,18,35,41]).

The solutions of population and epidemic models governed by reaction–diffusion equations have rich dynamic behaviors involving traveling wave, self-organized spatial pattern or chaos. Furthermore, time delay causes these models to exhibit much more complex dynamical behaviors than the counterpart without time delay. It is difficult or impossible to solve analytically these problems. For most of the population and epidemic models it is necessary to employ numerical methods to intensively study the dynamic properties, especially traveling wave, pattern and soliton.

There are several numerical methods for PDEs such as spectral method, finite difference (FD) method, finite volume (FV) method, finite element (FE) method, boundary element method, etc. Anaya et al. [3] constructed a combined FV-FE scheme to a predator–prey system in a polluted environment. Dehghan [16] developed a Legendre spectral method for solving a one-dimensional predator–prey system on a large spatial domain. However, among of these methods mentioned above, FD method and FE method are two major tools. FD method replaces the derivative in PDEs with difference formula, which is simple, intuitionistic, easy to master and widely used. The paper [20] presents two FD schemes for studying the dynamics of the model (1.1). Pao [32] investigated numerical methods based on FD for a system governed by nonlinear parabolic equations with time delay. FD schemes for reaction–diffusion equations can be found in a voluminous literatures (see, e.g., [46]).

FE method has some different characteristics in contrast to FD method. Firstly, FE method is based on the variational principle of mathematical physics, rather than PDE itself. Secondly, mesh generation for computational domain can be triangulations, tetrahedrons or quadrilaterals, hexahedrons, rather than just rectangles, cuboids. Thirdly, the solution of PDE is approximated by a simple function on the grid, for example piecewise polynomial function, rather than giving values only on grid nodes. Scheme combining FD with FE is one of the most popular methods for parabolic equations especially for population and epidemic models. The main idea of this method is that space discretization is performed with FE method while a classical FD discretization is carried out for time discretization. A large number of references applied this method to population and epidemic models. Keller et al. [25] resorted to a backward Euler-FE approach to numerically simulate an SEI model for the spread of rabies. Milner [27] introduced a numerical scheme combining Runge–Kutta method and FE method