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Global attractivity, spreading speeds and traveling waves of delayed nonlocal reaction–diffusion systems

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Abstract

The purpose of this work is to study the spatial dynamics of some delayed nonlocal reaction–diffusion systems in whole space. We first establish a series of comparison theorems to investigate the global attractivity of the equilibria for a delayed nonlocal reaction–diffusion system with and without quasi-monotonicity. Then we show that the spreading speed of a general system without quasi-monotone conditions is coincident with the minimal wave speed. Applying a fluctuation method, we further provide some sufficient conditions to ensure the upward convergence of the spreading speed and traveling wave solutions. Finally, we point out the effects of the delay and nonlocality on the spreading speed of the non-quasi-monotone systems.

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Keywords: Global attractivity; Spreading speed; Traveling wave solution; Minimal wave speed; Time delay and nonlocality

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1. Introduction

To model the spatial spread of epidemics via the environmental pollution produced by the infective human population, Capasso and Maddalena [5] proposed the following reaction–diffusion system:

$$\begin{cases} u_t(x,t) = d_1 u_{xx} - a_{11}u(x,t) + a_{12}v(x,t), \\ v_t(x,t) = d_2 v_{xx} - a_{22}v(x,t) + g\big(u(x,t)\big), \end{cases} \tag{1.1}$$

where $u(x,t)$ and $v(x,t)$, respectively, represent the spatial densities of bacteria and infective population at a point x in the habitat $\Omega \subseteq \mathbb{R}$ and time $t \geq 0$; $d_1 > 0$ and $d_2 \geq 0$ are diffusion coefficients; a_{11} is the natural death rate of bacteria and $a_{12}v$ is the contribution of infectious population to the density of bacteria; a_{22} is the natural diminishing rate of infected individuals. The nonlinearity $g(u)$ gives the "force of infection" on human due to the concentration of bacteria. In the past decades, dynamics of the model (1.1) with specific reaction terms have been widely discussed and analyzed, see [3,4,6,11,12,37,43] and references cited therein.

Later, in view of the latent period of a virus, Thieme and Zhao [25] modified the system (1.1) by studying the following reaction–diffusion model with distributed time delay:

$$\begin{cases} u_t(x,t) = d_1 u_{xx} - a_{11}u(x,t) + a_{12}v(x,t), \\ v_t(x,t) = d_2 v_{xx} - a_{22}v(x,t) + \displaystyle\int_0^\infty g\big(u(x,t-s)\big)P(ds), \end{cases} \tag{1.2}$$

where P is a probability measure on $\mathbb{R}_+$ which describes the distribution of the latent period. In particular, if the latent period has a fixed length τ, system (1.2) takes the form:

$$\begin{cases} u_t(x,t) = d_1 u_{xx} - a_{11}u(x,t) + a_{12}v(x,t), \\ v_t(x,t) = d_2 v_{xx} - a_{22}v(x,t) + g\big(u(x,t-\tau)\big). \end{cases} \tag{1.3}$$

However, for some infection agents, such as indirect transmission diseases (typhoid fever, schistosomiasis, malaria, etc.), u depends on not only the infective humans v at the spatial point x but also at the spatial neighbor points of x (even points in the whole region). Following this aspect, Capasso [2] further proposed and studied the following nonlocal model (see also [3,38]):

$$\begin{cases} u_t(x,t) = d_1 u_{xx} - a_{11}u(x,t) + \displaystyle\int_\Omega J(x-y)v(y,t)dy, \\ v_t(x,t) = d_2 v_{xx} - a_{22}v(x,t) + g\big(u(x,t)\big), \end{cases} \tag{1.4}$$

where the kernel function $J(x)$ describes the transfer kernel of the infective agents produced by the infective humans.

Recently, many researchers pointed out that the interaction effect of spatial movement, nonlocal interaction and time delay such as latent period on the spread of the disease should be taken into account for more realistic models. We refer to the survey papers Gourley et al. [10]