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Dynamics and profiles of a diffusive host–pathogen system with distinct dispersal rates [☆]

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Abstract

In this paper, we investigate a diffusive host–pathogen model with *heterogeneous parameters* and *distinct dispersal rates* for the susceptible and infected hosts. We first prove that the solution of the model exists globally and the model system possesses a global attractor. We then identify the basic reproduction number \mathcal{R}_0 for the model and prove its threshold role: if $\mathcal{R}_0 \leq 1$, the disease free equilibrium is globally asymptotically stable; if $\mathcal{R}_0 > 1$, the solution of the model is uniformly persistent and there exists a positive (pathogen persistent) steady state. Finally, we study the asymptotic profiles of the positive steady state as the dispersal rate of the susceptible or infected hosts approaches zero. Our result suggests that the infected hosts concentrate at certain points which can be characterized as the pathogen's most favoured sites when the mobility of the infected host is limited.

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1. Introduction

Since the pioneering work of Anderson and May [3], host–pathogen models have attracted considerable attention from mathematical biologists and bio-mathematicians, as exploration of such model systems can help better understand the mechanisms of spread of infectious diseases. The original host–pathogen model proposed and studied by Anderson and May is the following ODE system

$$\frac{du_1}{dt} = r(u_1 + u_2) - \beta u_1 u_3,
\frac{du_2}{dt} = \beta u_1 u_3 - \alpha u_2,
\frac{du_3}{dt} = -\delta u_3 + \gamma u_2 - \beta (u_1 + u_2) u_3,$$
(1.1)

where $u_1(t)$ and $u_2(t)$ represent the densities of susceptible and infected hosts at time t respectively, $u_3(t)$ is the density of pathogen particles, r is the reproductive rate of the host, β is the transmission rate, α is the mortality rate of the infected hosts induced by the invaded pathogen, γ and δ are the reproduction and decay rates of the pathogen particles respectively.

The model (1.1) has two obvious drawbacks: (i) intra-species competition is ignored (hence there is no self-restriction mechanism in the model) so that even in the absence of pathogen, the host population would grow unbounded exponentially; (ii) spatial effects (e.g., spatial heterogeneity and mobility) are also neglected. Dwyer [9] made an attempt to overcome the above two drawbacks by considering spatial model with one dimensional Laplacian operator $\partial/\partial x^2$ accounting for the random movement of hosts and a logistic growth for the hosts, given by the following system

$$\begin{cases} \frac{\partial u_1}{\partial t} = ru_1 \left(1 - \frac{u_1 + u_2}{K} \right) - \beta u_1 u_3 + d \frac{\partial^2 u_1}{\partial x^2}, \\ \frac{\partial u_2}{\partial t} = \beta u_1 u_3 - \alpha u_2 - r \frac{u_1 + u_2}{K} + d \frac{\partial^2 u_2}{\partial x^2}, \\ \frac{\partial u_3}{\partial t} = -\delta u_3 + \gamma u_2, \end{cases}$$
(1.2)

where $x \in \mathbb{R}$ is the spatial variable, and *K* is the carrying capacity. Here the consumption of the pathogen by the hosts is ignored, so there are only two terms in the third equation, and the pathogen is assumed to be immobile in the environment. In [9], Dwyer assumed that all parameters are constants and studied how these parameters affect the spatial spread of the pathogen by considering travelling wave solutions of (1.2).

Recently, based on the facts that the habitat of a host species is generally bounded and heterogenous, Wang et al. [25] considered a similar model but in an isolated bounded domain of general dimension and allowed space dependent parameters, represented by the following system

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