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Global dynamics of an SIR epidemic model with nonlocal diffusion☆

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ABSTRACT

In this paper, we are concerned with the global asymptotic stability of each equilibrium of an SIR epidemic model with nonlocal diffusion. Under the assumption of Lipschitz continuity of parameters, the eigenvalue problem associated with the linearized system around the disease-free equilibrium has a principal eigenvalue corresponding to a strictly positive eigenfunction. By setting the eigenfunction as the integral kernel of a Lyapunov function, we prove the global asymptotic stability of the disease-free equilibrium when the basic reproduction number $\mathcal{R}_0$ is less than one. We also prove the uniform persistence of the system when $\mathcal{R}_0 > 1$ by using the persistent theory for dynamical systems. Furthermore, in a special case where the dispersal rate for susceptible individuals is equal to zero, we prove the existence, uniqueness and global asymptotic stability of the endemic equilibrium when $\mathcal{R}_0 > 1$ by constructing a suitable Lyapunov function.

1. Introduction

Since the pioneering work of Kermack and McKendrick [1], differential equations as epidemic models have attracted much attention of many researchers. The heterogeneity (position, age, sex, etc.) of each individual is known to be an important factor in spread of infectious diseases, and hence, motivates more realistic model for disease dynamics. In particular, the spatial heterogeneity would give insights into disease spread and control, and there have been quite a few publications along this line (see, for instance, [2–15] and the references therein). Recently, Allen et al. [2] proposed a susceptible–infective–susceptible (SIS) reaction–diffusion model under heterogeneous environment,

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$$\begin{cases} \dfrac{\partial}{\partial t}S(t,x) = d_S \Delta S(t,x) - \dfrac{\beta(x)S(t,x)I(t,x)}{S(t,x)+I(t,x)} + \gamma(x)I(t,x), & t>0, \quad x\in\Omega, \\ \dfrac{\partial}{\partial t}I(t,x) = d_I \Delta I(t,x) + \dfrac{\beta(x)S(t,x)I(t,x)}{S(t,x)+I(t,x)} - \gamma(x)I(t,x), & t>0, \quad x\in\Omega, \\ \dfrac{\partial}{\partial \mathbf{n}}S(t,x) = \dfrac{\partial}{\partial \mathbf{n}}I(t,x) = 0, & t>0, \quad x\in\partial\Omega, \end{cases} \tag{1.1}$$

where $S(t,x)$ and $I(t,x)$ denote the density of susceptible and infective individuals in a given spatial region Ω, which is assumed to be a bounded domain in $\mathbb{R}^n (n \geq 1)$ with smooth boundary $\partial\Omega$. Ω is isolated from outside for the host, implying the homogeneous Neumann boundary condition; $\mathbf{n}$ is the outward unit normal vector on $\partial\Omega$ and $\partial/\partial\mathbf{n}$ means the normal derivative along $\mathbf{n}$ on $\partial\Omega$; d_S and d_I are the dispersal rates for susceptible and infective individuals, respectively; the positive functions $\beta(x)$ and $\gamma(x)$ represent the rates of disease transmission and recovery at position x, respectively. Allen et al. studied the existence, uniqueness and asymptotic profile of the equilibria. Although the theoretical conclusions exhibit the delicacy by defining the low-risk and high-risk sites, which is in terms of the rates of disease transmission and recovery, due to the mathematical difficulties, they could not derive any stability results for the endemic equilibrium. Subsequently, Peng and Liu [16] discussed the global stability of the endemic equilibrium of (1.1) in two special cases: (i) the diffusion rates of susceptible individuals and infective individuals are the same; (ii) the rate of disease transmission is proportional to the rate of disease recovery for any fixed constant. Biological results in [2,16] revealed that controlling the diffusion rate of the susceptible individuals can help eradicate the infection, while controlling the diffusion rate of the infectious individuals cannot.

Furthermore, Peng [17] studied the asymptotic profiles of the endemic equilibrium of (1.1) when the diffusion rate of either the susceptible individuals or the infective ones goes to infinity or zero, which provided new aspects and understanding of the impacts of diffusion rates on spatial–temporal dynamics of infectious diseases. Note that the rates of disease transmission adopted in [2,16] obey the standard incidence mechanism. In this circumstance, the basic reproduction number is independent on the total population. Recently, Wu and Zou [18] continued to study the impacts of spatial heterogeneity of environment and movement of individuals on the persistence and extinction of a disease. They explored the asymptotic profiles of the endemic steady state for large and small diffusion rates by using the mass action mechanism. This leads to more challenges in mathematical analysis that (i) the equilibrium problem is a nonlocal elliptic problem; (ii) the mass action term exhibits an unbounded infection force. Cui and Lou [19] considered circumstances that populations may take passive movement in certain direction due to external environmental forces such as water flow [20,21], wind [22] and so on. They added an advection term to model in [2,16], and studied the effects of diffusion and advection in heterogeneous environments. For other recent studies on the models which are related to (1.1), see [5,8,10,11].

The above mentioned works considered a fast disease by ignoring the demography of the host. In a recent work [7], Kuniya and Wang studied the following diffusive SIR epidemic model with mass action infection mechanism and homogeneous Neumann boundary condition,

$$\begin{cases} \dfrac{\partial}{\partial t}S(t,x) = k_S \Delta S(t,x) + b(x) - \beta(x)S(t,x)I(t,x) - \mu(x)S(t,x), & t>0, \quad x\in\Omega, \\ \dfrac{\partial}{\partial t}I(t,x) = k_I \Delta I(t,x) + \beta(x)S(t,x)I(t,x) - (\mu(x)+\gamma(x))I(t,x), & t>0, \quad x\in\Omega, \\ \dfrac{\partial}{\partial t}R(t,x) = k_R \Delta R(t,x) + \gamma(x)I(t,x) - \mu(x)R(t,x), & t>0, \quad x\in\Omega, \\ S(0,x) = S_0(x), \quad I(0,x) = I_0(x), \quad R(0,x) = R_0(x), & x\in\Omega, \\ \dfrac{\partial}{\partial \mathbf{n}}S(t,x) = \dfrac{\partial}{\partial \mathbf{n}}I(t,x) = \dfrac{\partial}{\partial \mathbf{n}}R(t,x) = 0, & t>0, \quad x\in\partial\Omega. \end{cases} \tag{1.2}$$

$S(t,x)$, $I(t,x)$ and $R(t,x)$ denote the populations of susceptible, infective and recovered individuals in position x at time t, respectively. k_S, k_I and k_R denote the dispersal rates for susceptible, infective and