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Threshold Dynamics of an Epidemic Model with Latency and Vaccination in a Heterogeneous Habitat*

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Abstract In this paper, we derive and analyze a nonlocal and time-delayed reaction-diffusion epidemic model with vaccination strategy in a heterogeneous habitat. First, we study the well-posedness of the solutions and prove the existence of a global attractor for the model by applying some existing abstract results in dynamical systems theory. Then we show the global threshold dynamics which predicts whether the disease will die out or persist in terms of the basic reproduction number $\mathfrak{R}_0$ defined by the spectral radius of the next generation operator. Finally, we present the influences of heterogeneous spatial infections, diffusion coefficients and vaccination rate on the spread of the disease by numerical simulations.

Keywords Diffusive epidemic model, Threshold dynamics, Heterogeneous habitat, Vaccination strategy.

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1. Introduction

Since Gumel and Moghadas [6] proposed an epidemic model with nonlinear incidence and vaccination strategy, researchers have done a lot of work on this model and its derived versions (see, e.g., [24]). In these models, the role of vaccination is only to reduce the chance of vaccinated individuals being infected and not fully immunize, which is different from most existing models that follow the assumption that vaccinated individuals will not be infected at all (see, e.g., [30]).

In real world, the nature of disease varies with temperature, humidity and other factors in different environments. The spatial heterogeneity plays an important role in the theory of epidemiology. So far, many mathematical models with spatial dependence have been studied (see, e.g., [13, 29]). On the other hand, many diseases have a latent period before the hosts becoming infectious. For instance, dengue fever is a viral disease, which is transmitted to humans by the Aedes aegypti mosquito feeding during the day. When an infectious mosquito bites a susceptible

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human, the virus is injected into his or her bloodstream and begins an latent period which takes from three to seven days [4]. Suppose that the latency τ is brought into this population, namely, the susceptible or vaccinated individuals will infect other uninfected individuals after being infected τ time, resulting in dividing the population into five epidemiological classes living in the spatial habitat Ω with smooth boundary $\partial\Omega$: susceptible, vaccinated, exposed, infectious and recovered classes, denoted by $S = S(x,t)$, $V = V(x,t)$, $E = E(x,t)$, $I = I(x,t)$ and $R = R(x,t)$, respectively.

Note that if an individual is infected by the disease in one location, and can move freely during the latent period, this individual may appear at any location in the domain when this individual becomes infectious. This means that the mobility of the individuals in the latent period will lead to non-local infection. Some non-local reaction-diffusion models in a spatially continuous habitat have been widely studied (see, e.g., [12,23,25,28]). To incorporate non-local infection into the model properly, we introduce an infection age variable θ, and let $u(x,t,\theta)$ represents the density of infected population with infection age θ at time t and location $x \in \Omega$. Using the standard method on describing age structured population with spatial diffusion [17], we have

$$\frac{\partial u(x,t,\theta)}{\partial t} + \frac{\partial u(x,t,\theta)}{\partial \theta} = D(x,\theta)\Delta u(x,t,\theta) - \mu(x)u(x,t,\theta) - \gamma(x,\theta)u(x,t,\theta), \tag{1.1}$$

where $D(x,\theta)$ and $\gamma(x,\theta)$ are the diffusion rate and the recovery rate at location x and age θ, respectively, and $\mu(x)$ denotes the natural death rate which is independent of the infection age. It easily follows that

$$E(x,t) = \int_0^\tau u(x,t,\theta)d\theta \quad \text{and} \quad I(x,t) = \int_\tau^\infty u(x,t,\theta)d\theta.$$

From the biological considerations, infected individuals connot recover during the latent period. To make the model mathematically tractable yet without losing the main features, we assume that

$$D(x,\theta) = \begin{cases} D_E(x) & \text{for} \quad x \in \Omega, \theta \in [0,\tau), \\ D_I(x) & \text{for} \quad x \in \Omega, \theta \in [\tau,\infty), \end{cases}$$

$$\gamma(x,\theta) = \begin{cases} 0 & \text{for} \quad x \in \Omega, \theta \in [0,\tau), \\ \gamma(x) & \text{for} \quad x \in \Omega, \theta \in [\tau,\infty). \end{cases}$$

Integrating both sides of (1.1) with respect to θ from 0 to τ, and from τ to ∞, respectively, we obtain that

$$\frac{\partial E(x,t)}{\partial t} = D_E(x)\Delta E(x,t) - \mu(x)E(x,t) + u(x,t,0) - u(x,t,\tau) \tag{1.2}$$

and

$$\frac{\partial I(x,t)}{\partial t} = D_I(x)\Delta I(x,t) - (\mu(x)+\gamma(x))I(x,t) + u(x,t,\tau) - u(x,t,\infty). \tag{1.3}$$

Biologically, it can be assumed that $u(x,t,\infty) = 0$ (see e.g., [7]). Let $\beta_1(x)$ and $\beta_2(x)$ be the transmission coefficients of susceptible and vaccinated individuals at