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Research paper

A diffusive virus infection dynamic model with nonlinear functional response, absorption effect and chemotaxis☆

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ABSTRACT

From a biological perspective, a diffusive virus infection dynamic model with nonlinear functional response, absorption effect and chemotaxis is proposed. In the model, the diffusion of virus consists of two parts, the random diffusion and the chemotactic movement. The chemotaxis flux of virus depends not only on their own density, but also on the density of infected cells, and the density gradient of infected cells. The well posedness of the proposed model is deeply investigated. For the proposed model, the linear stabilities of the infection-free steady state E_0 and the infection steady state E^* are extensively performed. We show that the threshold dynamics can be expressed by the basic reproduction number R_0 of the model without chemotaxis. That is, the infection-free steady state E_0 is globally asymptotically stable if $R_0 < 1$, and the virus is uniformly persistent if $R_0 > 1$. In addition, we use the cross iteration method and the Schauder's fixed point theorem to prove the existence of travelling wave solutions connecting the infection-free steady state E_0 and the infection steady state E^* by constructing a pair of upper-lower solutions. At last, numerical simulations are presented to confirm theoretical findings.

1. Introduction

Recently, the virus infection dynamic models with diffusion term have become more and more popular. In order to study the evolutionary competitiveness of lytic virus, authors in article [1] proposed the spatial dynamics of viral model with diffusion term, and found some interesting phenomena that lytic viruses can be evolutionary competitive and the efficacy of the flooding depends on the diffusion rate of the antibodies. In articles [2] and [4], authors investigated the existence of the travelling wave solutions for an HBV infection model with bilinear mass-action function response and time delay. For the HBV infection model with saturation response of the infection rate and time delay, the global stability properties of the steady states were studied by the authors in article [3]. In article [5], authors discussed the global stability properties of the steady states by constructing the suitable Lyapunov functions and investigated the existence of the travelling wave solutions by applying the cross iteration method and the Schauder's fixed point theorem for the HBV infection model with Beddington-DeAngelis response and time delay. From articles [1,8–11,13,26] and [40], it can be found that some exten-

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sive developments have been performed on the mathematical models with diffusion term to place the relevant models in naturally more sensible.

Although the virus infection dynamic models with diffusion term have been extensively studied by the authors in above articles, they only considered the free diffusion of virus. Recently, authors in article [6] reveal a new phenomenon, that is, vaccinia virus spreads much faster than the free diffusion itself. A new theory which is called the repulsion of super-infecting virions by infected cells is proposed to explain for the innovative phenomenon in article [6]. Mathematical models have played a magnificent role in the development of the virus infection dynamics. To investigate the repulsion effect of infected cells on super-infecting virions, authors in article [8] proposed the following mathematical model

$$\begin{cases} \dfrac{\partial U(x,t)}{\partial t} = D\Delta U + \xi(x) - \beta(x)U(x,t)\omega(x,t) - dU(x,t), \\ \dfrac{\partial V(x,t)}{\partial t} = D\Delta V + \beta(x)U(x,t)\omega(x,t) - aV(x,t), \\ \dfrac{\partial \omega(x,t)}{\partial t} = \nabla(D_\omega(V)\nabla\omega) + k(x)V(x,t) - \mu\omega(x,t). \end{cases} \tag{1.1}$$

In the model (1.1), $U(x, t)$, $V(x, t)$ and $\omega(x, t)$ represent the densities of uninfected cells, infected cells and virus, at location x and time t, respectively. $\xi(x)$ is the production rate of uninfected cells and d represents the death rate of uninfected cells. $\beta(x)$ is the infection rate. Infected cells die at rate $aV(x, t)$. $k(x)V(x, t)$ represents the production of free viruses and μ is the death rate of free viruses. $\xi(x)$, $\beta(x)$ and $k(x)$ are assumed to be positive, continuous and bounded functions. It is assumed that all the aforementioned parameters are non-negative in order to be biologically sensible.

For the model (1.1), target cells and infected cells were assumed to have the same diffusion rate D. The diffusion of free viruses relies not only on its concentration gradient but also on the concentration of infected cell, that is

$$\vec{J}_\omega = D_\omega(V)(-\nabla\omega),$$

where $D_\omega(V)$ was an increasing function of the concentration of infected cells $V(x, t)$, that is,

$$D_\omega(V) = D_0 + q(V).$$

Here, D_0 is free diffusion coefficient of viruses and the function $q \in C^2(R^+, R^+)$ is an increasing function of V.

It should be noted here that, in the article [8], authors studied the nonexistence of the travelling wave solutions for the case $\Omega = R$. The linear stabilities of steady states were discussed. Moreover, article [8] indicated that the repulsion effect could promote the spread of the viruses by numerical simulations.

Mathematical modelling of chemotaxis (the movement of cells or organisms in response to chemical gradients) has developed into a large and diverse subject, whose aspects include medicine, the modelling of specific systems and the mathematical behaviour of the underlying equations. In medicine, chemoattraction represents a directed movement of organisms up a concentration gradients of chemotactic agents. Conversely, chemorepulsion is defined as a directed movement of organisms down a concentration gradient of chemotactic agents (see, for example, [17], [18]). From mathematical standpoint, chemoattraction and chemorepulsion describe the directed movements of organisms towards or away from the chemotactic agents, respectively (see, for example [15]).

In terms of article [6], it can be seen that the spread rate of viruses should depend on the concentration of infected cells, and high concentration of infected cells can repel the spread of viruses. The repellant activity of infected cells on virus causes the active environment of virus up from the uninfected cells. As mentioned in article [6], we can conclude that infected cells acts as a chemorepellant for virus. The diffusion of virus consists of two parts, the random diffusion and the chemotactic movement. The random diffusion flux of virus is proportional to their density gradient

$$\vec{J}_D = D_0(-\nabla\omega).$$

Therefore, the chemotaxis flux J_ω of virus depends on the their own density, the density of infected cells, and the density gradient of infected cells. The chemotaxis flux of virus can be described as

$$\vec{J}_\omega = -\omega\chi_2(V,\omega)\nabla V.$$

The derivation of this chemotactic term can be referred from the articles of [15] and [16]. The function $\chi_2(V, \omega)$ represents the chemotactic response, which denotes chemorepulsion if it is positive (see, for example, [14], [15], [19], [20], [21]). In the light of the above remarks, a diffusive virus infection dynamic model with nonlinear functional response, absorption effect and chemotaxis is proposed

$$\begin{cases} \dfrac{\partial U(x,t)}{\partial t} = D\Delta U + \xi - f(U(x,t),\omega(x,t))\omega(x,t) - dU(x,t), \\ \dfrac{\partial V(x,t)}{\partial t} = D\Delta V + f(U(x,t),\omega(x,t))\omega(x,t) - ag(V(x,t)), \\ \dfrac{\partial \omega(x,t)}{\partial t} = D_0\Delta\omega + \nabla(\omega\chi_2(V,\omega)\nabla V) + kg(V(x,t)) - \mu\omega(x,t) - f(U(x,t),\omega(x,t))\omega(x,t), \end{cases} \tag{1.2}$$

for $t > 0, x \in \Omega \subset R^3$, with the homogeneous Neumann boundary conditions

$$\frac{\partial U(x,t)}{\partial n} = \frac{\partial V(x,t)}{\partial n} = \frac{\partial \omega(x,t)}{\partial n} = 0, \ \ t > 0, \ \ x \in \partial\Omega, \tag{1.3}$$