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## Repulsion effect on superinfecting virions by infected cells for virus infection dynamic model with absorption effect and chemotaxis\*

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A mathematical model for virus infection dynamics with absorption effect and chemotaxis is proposed to study the repulsion effect on superinfecting virions by infected cells. The basic reproduction number  $R_0$  is established. Furthermore, we show that the threshold dynamics can be expressed by the basic reproduction number  $R_0$  in a bounded domain. It is shown that the infection-free steady state  $E_0$  is asymptotically stable if  $R_0 < 1$ , and the virus is uniformly persistent if  $R_0 > 1$  in the case of spatially heterogeneous infections. The stability properties and Turing instability of the proposed model have been extensively discussed for the case of spatially homogeneous infections. In addition, the existence of the travelling wave solutions is discussed in unbounded domain. At last, numerical simulations are carried out to illustrate the main results.

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## 1. Introduction

It is well known that spatial structure is playing an important role in better understanding of the virus dynamics (for example, [1,2]). In the articles [3,4], authors considered an HBV infection model, in which susceptible cells and infected cells are assumed to be hepatocyte and cannot move, but viruses move freely under normal conditions. For the proposed model, the global stability properties of corresponding steady states and the existence of the travelling wave solutions are investigated. Further, the existence of travelling wave solutions has been discussed for the models which are satisfying the quasi-monotonicity or exponential quasimonotonicity conditions (see for example, [5,6]). In the articles [7-9], the authors

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investigated the existence of travelling wave solutions of a series of models satisfying the partial quasimonotonicity (PQM) and partial exponential quasi-monotonicity by constructing the coupled upper-lower solutions. In the articles [10–15], some further developments have been performed on the mathematical models with diffusion term.

In the recent years, although the virus dynamic models with diffusion term have been extensively studied (see, for example [3,4,8,9]), most of them have assumed that the diffusion coefficients are constants, that is, the free diffusion of virus is only considered. Recently, a very interesting phenomenon was discovered in paper [16], that is vaccinia virus spreads much faster than the free diffusion itself. Although this phenomenon was first discovered in the vaccinia virus, authors in [16] show that the similar phenomenon can be found in some other kinds of virus. The authors of paper [16] proposed a new theory which is called the repulsion of superinfecting virions by infected cells to explain this interesting phenomenon. Mathematical models describing the virus dynamics have played an important role in better understanding virus pathogenesis. To investigate the repulsion effect of infected cells on superinfecting virions, the authors of paper [13] proposed the following mathematical model

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

where 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.  $\xi(x)$  represents the production rate of uninfected cells. d is the death rate of uninfected cells. Uninfected cells become infected cells at rate  $\beta(x)U(x,t)\omega(x,t)$ . Infected cells are produced at rate  $\beta(x)U(x,t)\omega(x,t)$  and die at rate aV(x,t). k(x)V(x,t) is the production of free viruses.  $\mu$ is the death rate of free viruses.  $\xi(x)$ ,  $\beta(x)$  and k(x) are assumed to be positive, continuous and bounded functions. d, a and  $\mu$  are positive constants.

For the model (1.1), target cells and infected cells were assumed to follow the Fickian diffusion with the same constant diffusion rate D. The fluxes of target cells and infected cells are related to their concentration gradient and go from regions of high concentration to regions of low concentration, that is

$$\vec{J}_U = -D\nabla U, \qquad \vec{J}_V = -D\nabla V.$$

Authors of paper [16] have revealed that the spread of viruses can be promoted by the high concentration of infected cells. The flux of free viruses relies not only on its concentration gradient but also on the concentration of infected cells, that is

$$\overrightarrow{J}_{\omega} = D_{\omega}(V)(-\nabla\omega).$$

Thus, in paper [13],  $D_{\omega}(V)$  was assumed to be an increasing function of the concentration of infected cells V(x,t), that is,

$$D_{\omega}(V) = D_0 + q(V),$$

where  $D_0$  is a constant representing free diffusion rate of free viruses. The function  $q \in C^2(\mathbb{R}^+, \mathbb{R}^+)$  is an increasing function of V which represents the movement of free viruses due to repulsion of superinfecting virions by infected cells. Obviously, q(0) = 0 meaning that if there is no infected cell, then there is no repulsion effect.

It should be mentioned here that, in paper [13], authors studied the nonexistence of the travelling wave solutions for the case  $\Omega = R$ . The linear stabilities of the infection-free steady state  $E_0$  and the infection steady state  $E^*$  were investigated. Furthermore, from the numerical simulations, paper [13] indicates that Download English Version:

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