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Discretization and dynamic consistency of a delayed and diffusive viral infection model☆

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ABSTRACT

A diffusive and delayed viral infection model with nonlinear incidence has been studied, and the global dynamical behaviors of the original model is investigated by constructing Lyapunov functionals. Furthermore, the analysis is carried out for the discrete model which is obtained by applying the nonstandard finite difference (NSFD) scheme to the original continuous model. The global stability for the corresponding equilibria is investigated by constructing discrete Lyapunov functionals as well as the positivity and boundedness of solutions of the corresponding continuous model. The results imply that the discretization scheme can efficiently preserves the qualitative properties of solutions for the original continuous model. Numerical experiments are carried out to support the theoretical results.

1. Introduction

Since samples cannot always be taken frequently from patients, or detection techniques of the virus may not be accurate, testing specific hypotheses based on clinical data is a challengeable task, which justifies the central role played by mathematical models in describing the dynamics inside the host of various infectious diseases such as HBV, HCV, HIV and so on. Thus, many literatures have been studied [1–6]. For example, Manna and Chakrabarty [6] considered an HBV infection model with capsids which takes the following form

$$\begin{cases} \dfrac{\partial H}{\partial t} = s - \mu H(t) - kH(x,t)V(x,t), \\ \dfrac{\partial I}{\partial t} = kH(x,t)V(x,t) - \delta I(t), \\ \dfrac{\partial D}{\partial t} = d_1 \Delta D(x,t) + aI(x,t) - (\beta+\delta)D(x,t), \\ \dfrac{\partial V}{\partial t} = d_2 \Delta V(x,t) + \beta D(x,t) - cV(x,t), \end{cases} \tag{1.1}$$

where $H(t)$, $I(t)$, $D(t)$ and $V(t)$ denote the densities of the uninfected hepatocytes, infected hepatocytes, intracellular HBV DNA-containing capsids, and the virions at position x and at time t, respectively. The hepatocytes are assumed to be

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produced from a source at rate s, has a natural death rate μ and gets infected by the virions at a rate k, with the infected hepatocytes clearing out at a rate δ. a represents the rate of production of HBV DNA-containing capsids. β is the rate at which the capsids are transmitted to blood. c is the death rate of virions. d_1 and d_2 are the diffusion coefficients of capsids and virions, respectively with Δ being the Laplacian operator. The global dynamics of the model (1.1) has been investigated in [6] by constructing Lyapunov functions.

Note that model (1.1) does not take the time delay into account. In fact, for many infectious diseases, it is important to consider the influences of delays on the disease dynamics. In epidemiological models, delay can be caused by a variety of factors. In order to take the effect of time delay into consideration, Manna [7] and Manna and Chakrabarty [8] considered a HBV infection model with two intracellular delays, one delay represents the time needed (τ_1) in the production of productively infected hepatocytes from the uninfected ones, another delay (τ_2) means the time spend in the production of matured intracellular HBV DNA-containing capsids which in turn contributes to the production of virions. Moreover, noticing that the bilinear incidence rate is a simple description of the infection in model (1.1). However, as mentioned in [9], a general incidence rate may help us to gain the unification theory by the omission of unessential details. Therefore, motivated by authors in [6–9], we consider the following model

$$\begin{cases} \dfrac{\partial H}{\partial t} = s - \mu H(x,t) - kH(x,t)f(V(x,t)), \\ \dfrac{\partial I}{\partial t} = kH(x,t-\tau_1)f(V(x,t-\tau_1)) - \delta I(x,t), \\ \dfrac{\partial D}{\partial t} = d_1\Delta D(x,t) + aI(x,t-\tau_2) - (\beta+\delta)D(x,t), \\ \dfrac{\partial V}{\partial t} = d_2\Delta V(x,t) + \beta D(x,t) - cV(x,t). \end{cases} \tag{1.2}$$

Here, the incidences are assumed to be the nonlinear responses to the concentrations of virus particles, taking the form $kH(x,t)f(V(x,t))$, where $f(V)$ denote the force of infection by virions and satisfy the following properties [10]:

$$f(0) = 0, \quad f'(V) > 0, \quad f''(V) \le 0. \tag{1.3}$$

Based on condition (1.3), it follows from the Mean Value Theorem that

$$f'(V)V \le f(V) \le f'(0)V. \tag{1.4}$$

Epidemiologically, condition (1.3) indicates that: (i) the disease cannot spread if there is no infection; (ii) the incidences $kHf(V)$ becomes faster as the densities of the virions increase; (iii) the per capita infection rates by virions will slow down due to certain inhibition effect since (1.4) implies that $(\frac{f(V)}{V})' \le 0$.

The initial conditions for model (1.2) are

$$\begin{aligned} &H(x,\theta) = \phi_1(x,\theta) \ge 0, \ I(x,\theta) = \phi_2(x,\theta) \ge 0, \ D(x,\theta) = \phi_3(x,\theta) \ge 0, \\ &V(x,\theta) = \phi_4(x,\theta) \ge 0, \ (x,\theta) \in \bar{\Omega} \times [-\tau, 0], \ (i = 1,2,3,4), \end{aligned} \tag{1.5}$$

and homogeneous Neumann boundary conditions

$$\frac{\partial D}{\partial n} = 0, \quad \frac{\partial V}{\partial n} = 0 \quad \text{on} \quad \partial\Omega \times (0, +\infty), \tag{1.6}$$

where $\tau = \max\{\tau_1, \tau_2\}$ and $(\phi_i(x,\theta)(i = 1,2,3,4)$ is hölder continuous in $\bar{\Omega} \times [-\tau, 0]$, Ω is a bounded domain in $\mathbb{R}^n$ with smooth boundary $\partial\Omega$, and $\frac{\partial D}{\partial n}$, $\frac{\partial V}{\partial n}$ denotes the outward normal derivative on $\partial\Omega$.

Generally, the exact solution for a model like (1.2) is very difficult or even impossible to be determined. Hence, researchers seek numerical ones instead. However, how to select a proper discrete method so that the global properties of solutions of the corresponding continuous models can be efficiently preserved is still an open problem [11]. Mickens has made an attempt in this regard, by proposing a robust non-standard finite difference (NSFD) scheme [12,13], which has been widely employed in the study of different kinds of epidemic models and one important advantage of Mickens's method is that it can more efficient in preserving the global dynamics to the corresponding continuous epidemic models [6,14–18]. For example, Manna and Chakrabarty [6] used the NSFD scheme to discretize system (1.1) and found that the global dynamics of the discrete model are consistent with the original system. Therefore, motivated by authors in [6,12,13], we can obtain