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A non-standard finite difference scheme for a delayed and diffusive viral infection model with general nonlinear incidence rate

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ABSTRACT

A non-standard finite difference scheme is proposed to solve a delayed and diffusive viral infection model with general nonlinear incidence rate. The results show that the discrete model preserves the positivity and boundedness of solutions in order to ensure the well-posedness of the problem. Moreover, this method preserves all equilibria of the original continuous model. By constructing Lyapunov functionals, we show that the global stability of equilibria is completely determined by the basic reproduction number $\Re_0$, which implies that the proposed discrete model can efficiently blue preserve the global stability of equilibria of the corresponding continuous model. Numerical experiments are carried out to support the theoretical results.

1. Introduction

Over the past few decades, there has been a great effort in the mathematical modeling of within-host virus dynamics models. These models have been used to describe the dynamics inside the host of various infectious diseases such as HIV, HCV, HBV, HTLV, as well as the flu or even the malaria parasite. Since samples cannot always be taken too frequently from patients, or because detection techniques of the virus may not be accurate, thus testing specific hypotheses based on clinical data is a challengeable task. This justifies the central role played by mathematical models in this area. The classical model for within-host virus dynamics is a system which includes three ordinary differential equations [1,2]. For better understanding of the dynamics of these infections, many extended mathematical models have been proposed by using different kinds of differential equations [3–11] and references therein. For example, Yang et al. [9] studied the following model

$$\begin{cases} \dfrac{\partial T(x,t)}{\partial t} = \lambda - dT(x,t) - \beta_1 T(x,t)V(x,t) - \beta_2 T(x,t)I(x,t), \\ \dfrac{\partial I(x,t)}{\partial t} = \beta_1 T(x,t)V(x,t) + \beta_2 T(x,t)I(x,t) - \delta I(x,t), \\ \dfrac{\partial V(x,t)}{\partial t} = D\triangle V(x,t) + pI(x,t) - cV(x,t). \end{cases} \tag{1.1}$$

Here $T(x,t)$, $I(x,t)$ and $V(x,t)$ denote the densities of uninfected cells, infected cells and free virus at position x at time t, respectively. λ is the recruitment rate of the uninfected cells. β_1 is the virus-to-cell infection rate. d, δ and c are death rates

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of uninfected cells, infected cells and free viruses, respectively. p is the recruitment rate for free viruses. D is the diffusion coefficient and Δ is the Laplacian operator.

However, there is no delay in system (1.1). Actually, in modeling of many biological processes, time delays are usually introduced for the purpose of accurate representations of the phenomena. For example, intracellular time delays were incorporated into mathematical models in viral dynamics [3–8,10,12–15] and references therein. On the other hand, the bilinear incidence rate is a simple description of the infection in system (1.1). As mentioned in [14,15], a general incidence rate may help us to gain the unification theory by the omission of unessential details. Hence, inspired by the aforementioned work, in this paper we propose the following model:

$$\begin{cases} \dfrac{\partial T(x,t)}{\partial t} = \lambda - dT(x,t) - \beta_1 T(t) f(V(x,t)) - \beta_2 T(x,t) g(I(x,t)), \\ \dfrac{\partial I(x,t)}{\partial t} = e^{-\mu_1\tau_1}(\beta_1 T(x,t-\tau_1) f(V(x,t-\tau_1)) + \beta_2 T(x,t-\tau_1) g(I(x,t-\tau_1))) - \delta I(x,t), \\ \dfrac{\partial V(x,t)}{\partial t} = D\Delta V(x,t) + pe^{-\mu_2\tau_2} I(x,t-\tau_2) - cV(x,t). \end{cases} \tag{1.2}$$

Here μ_1 represents the death rate for infected but not yet virus-producing cells. τ_1 represents the latent delay, i.e. the time period from being infected to become productive infected cells. Therefore, the probability of surviving from time $t-\tau_1$ to time t is $e^{-\mu_1\tau_1}$. The probability of survival of immature virions is given by $e^{-\mu_2\tau_2}$ and the average life time of an immature virus is given by $\frac{1}{\mu_2}$, where τ_2 represents the time necessary for the newly produced virions to become mature. In general, we have $d < \delta$. The other parameters have the same meanings as in system (1.1). Here, the incidences are assumed to be the nonlinear responses to the concentrations of virus particles and infected cells, taking the forms $\beta_1 T f(V)$ and $\beta_2 T g(I)$, where $f(V)$ and $g(I)$ denote the force of infection by virus particles and infected cells and satisfy the following properties [16]:

$$f(0) = g(0) = 0, \quad f'(V) > 0,\ g'(I) > 0 \quad f''(V) \le 0,\ g''(I) \le 0. \tag{A1}$$

Based on condition (A1), it follows from the Mean Value Theorem that

$$f'(V)V \le f(V) \le f'(0)V, \quad g'(I)I \le g(I) \le g'(0)I, \quad \text{for } I, V \ge 0. \tag{A2}$$

Epidemiologically, condition (A1) indicates that: (i) the disease cannot spread if there is no infection; (ii) the incidences $\beta_1 T f(V)$ and $\beta_2 T g(I)$ become faster as the densities of the virus particles and infected cells increase; (iii) the per capita infection rates by virus particles and infected cells will slow down due to certain inhibition effect since (A2) implies that $(\frac{f(V)}{V})' \le 0$ and $(\frac{g(I)}{I})' \le 0$.

Obviously, the incidence rate with condition (A1) contains the bilinear and the saturation incidences. Hence, the system (1.1) can be regarded as a special case of (1.2). In this paper, we consider system (1.2) with initial conditions as follows

$$\begin{aligned} T(x,s) &= \phi_1(x,s) \ge 0, \quad I(x,s) = \phi_2(x,s) \ge 0, \\ V(x,s) &= \phi_3(x,s) \ge 0, \quad (x,s) \in \bar{\Omega} \times [-\tau, 0], \end{aligned} \tag{1.3}$$

and homogeneous Neumann boundary conditions

$$\frac{\partial V}{\partial n} = 0, \quad t > 0,\ x \in \partial\Omega, \tag{1.4}$$

where $\tau = \max\{\tau_1, \tau_2\}$ and Ω is a bounded domain in $\mathbb{R}^n$ with smooth boundary $\partial\Omega$, and $\frac{\partial}{\partial n}$ denotes the outward normal derivative on $\partial\Omega$.

Generally, the exact solution for a system like (1.1) is difficult or even impossible to be determined. Hence, researchers seek numerical ones instead. However, how to select the proper discrete scheme so that the global dynamics of solutions of the corresponding continuous models can be efficiently preserved is still an open problem [17]. Actually, Mickens has made an attempt in this regard, by proposing a robust non-standard finite difference (NSFD) scheme [18], which has been widely employed in the study of different epidemic models [19–25]. For example, Yang et al. [9] applied the NSFD scheme to discretize system (1.1) and found that the global dynamics of the discrete model are consistent with the original system. Motivated by the work of [9,18], we apply the NSFD scheme to discretize system (1.2) and obtain

$$\begin{cases} \dfrac{T_{n+1}^m - T_n^m}{\Delta t} - = \lambda - dT_{n+1}^m - \beta_1 T_{n+1}^m f(V_n^m) - \beta_2 T_{n+1}^m g(I_n^m), \\ \dfrac{I_{n+1}^m - I_n^m}{\Delta t} = e^{-\mu_1\tau_1}\left(\beta_1 T_{n-m_1+1}^m f(V_{n-m_1}^m) + \beta_2 T_{n-m_1+1}^m g(I_{n-m_1}^m)\right) - \delta I_{n+1}^m, \\ \dfrac{V_{n+1}^m - V_n^m}{\Delta t} = D\dfrac{V_{n+1}^{m+1} - 2V_{n+1}^m + V_{n+1}^{m-1}}{(\Delta x)^2} + pe^{-\mu_2\tau_2} I_{n-m_2+1}^m - cV_{n+1}^m. \end{cases} \tag{1.5}$$

Here we set $x \in \Omega = [a,b]$. Let $\Delta t > 0$ be the time step size and $\Delta x = \frac{b-a}{N}$ be the space step size with N is a positive integer. Assume that there exist two integers $m_1, m_2 \in \mathbb{N}$ with $\tau_1 = m_1\Delta t$, $\tau_2 = m_2\Delta t$. Denote the mesh grid point as $\{(x_m, t_n), m = 0, 1, 2, \ldots, N, n \in \mathbb{N}\}$ with $x_m = a + m\Delta x$ and $t_n = n\Delta t$. At each point, we denote approximations of

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