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Analysis on bifurcation solutions of an atherosclerosis model☆

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HIGHLIGHTS

- The inflammatory process model of atherosclerosis is concerned.
- The dissipation and persistence of the system are obtained.
- The bifurcation from the simple eigenvalue can be extended to infinity.
- The bifurcation from the double eigenvalue is intensively investigated.

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ABSTRACT

This paper is concerned with the inflammatory process model resulting in the development of atherosclerosis subject to no-flux boundary conditions. The dissipation and persistence of the system are obtained. The steady-state bifurcations are also studied in two cases. The bifurcation from the simple eigenvalue can be extended to infinity by increasing d_2 to infinity, and the bifurcation from the double eigenvalue is intensively investigated. The techniques include the spectrum analysis of operators, the bifurcation theory, space decompositions and the implicit function theorem.

1. Introduction

Atherosclerosis is one of inducements that leads to the death of population in the western developed countries. With the improvement of people's living standards and the changing of eating habits, the disease has also become one of the main causes of death in China. For a long time past, atherosclerosis has always been the research emphasis in scope of medicine and biochemistry.

The pathogenesis mechanism of atherosclerosis could be expressed in [1,2] as follows: Numerous pathophysiologic observations in humans and animals led to the formulation of the response-to-injury

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hypothesis of atherosclerosis, which initially proposed that endothelial denudation was the first step in atherosclerosis [3]. Atherosclerosis is a chronic inflammatory disease. Because high plasma concentrations of cholesterol are one of the principal risk factors for atherosclerosis, the process of atherogenesis has been considered by many to consist largely of the accumulation of lipids within the artery wall [4]. This mechanical inhibition of the inflammation may become a part of the disease process. The interaction between the flow and the cap may lead to a thrombus, or to the degradation and rupture of the plaque liberating dangerous solid parts in the flow [5].

Therefore, according to the pathogenesis mechanism of atherosclerosis, in this paper, we study the following model [6]

$$\begin{cases} u_t = d_1 u_{xx} + f_1(v) - m_1 u, & x \in (0,l), t > 0, \\ v_t = d_2 v_{xx} + f_2(v)u - m_2 v, & x \in (0,l), t > 0, \\ u_x = v_x = 0, & x = 0, l, \quad t > 0, \\ u(x,0) = u_0(x) \geq\not\equiv 0, \qquad v(x,0) = v_0(x) \geq\not\equiv 0, & x \in (0,l) \end{cases} \tag{1.1}$$

for $l \in R^+$. Here $u(x,t)$ is the density of immune cells and $v(x,t)$ is the density of the cytokines secreted by the immune cells. The function $f_1(v) = \frac{\alpha_1+\beta_1 v}{1+v/\tau_1}$ models the recruitment of the immune cells from the blood flow. The term $f_2(v)u$ models the cytokines production rate, with $f_2(v) = \frac{\alpha_2 v}{1+v/\tau_2}$. The terms $-m_1 u$ and $-m_1 v$ represent the degradation of the immune cells u and the cytokines v, respectively, $d_1 u_{xx}$ and $d_2 v_{xx}$ describe their diffusion or cell displacement in the intima. All the parameters of the model, $\alpha_1, \beta_1, \tau_1, \alpha_2, \tau_2, m_1, m_2, d_1$ and d_2 are assumed to be non-negative. ***Throughout this paper, for*** f_1 ***to be an increasing function of*** v, ***we impose the condition***

$$\tau_1 > \frac{\alpha_1}{\beta_1}. \tag{1.2}$$

The atherosclerosis model has been studied in some literatures. For model (1.1), Khatib et al. [6] discovered the concentration of the oxidized low density lipoproteins (ox-LDL) in the intima is the critical parameter. For two types of low and high level of oxidized low density lipoproteins concentration with two species, the existence of traveling wave solutions was investigated. Subsequently, Khatib et al. [7] suggested a 2D mathematical model of the initiation and development of atherosclerosis. They proved the existence of traveling waves described by this system and the theoretical results are confirmed by the results of numerical simulations. Recently, Liu and Li [8] considered the local bifurcation around a constant steady-state solution from simple eigenvalues by the classical Crandall & Rabinowitz bifurcation theorem.

The present paper is organized as follows. In Section 2, we investigate the non-negative constant equilibrium solution, dissipation and persistence of system (1.1). Under appropriate conditions, the non-existence of non-constant positive steady-state solutions and the global bifurcation of system (1.1) are established in Section 3. In addition, the bifurcation of the constant steady-state solution U_l from double eigenvalues are studied in Section 4. Some discussions are given in Section 5.

2. The constant equilibrium analysis, dissipation and persistence

In this section, we discuss mainly the non-negative constant equilibria solution, dissipation and persistence of system(1.1). Furthermore, the non-existence of non-constant positive steady-state solutions is established.

$$\begin{cases} d_1 u'' + f_1(v) - m_1 u = 0, & x \in (0,l), \\ d_2 v'' + f_2(v)u - m_2 v = 0, & x \in (0,l), \\ u_x = v_x = 0, & x = 0, l. \end{cases} \tag{2.1}$$

The number of equilibrium points depends on the parameters, the following lemma is from [6].