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Steady State Solution and Its Stability Analysis of a Reaction-Diffusion Mathematical Model for LDL, Lipoprotein(a), and CRP in Atherosclerosis

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Abstract This study presents a reaction-diffusion mathematical model to investigate the spatiotemporal dynamics of low-density lipoprotein (LDL), lipoprotein(a), and C-reactive protein (CRP) within the arterial wall, key mediators in the pathogenesis of atherosclerosis and related cardiovascular diseases. The model employs a system of partial differential equations to capture both the diffusion of these biomolecules and their biochemical interactions, including LDL oxidation, CRP-mediated inflammatory responses, and plaque initiation. Numerical simulations, implemented using the finite difference method, demonstrate that CRP substantially accelerates LDL oxidation and promotes plaque deposition, particularly in regions of locally elevated lipoprotein concentrations. Furthermore, the results indicate that elevated CRP levels synergistically enhance the pro-atherogenic effects of LDL and lipoprotein(a), highlighting the critical role of inflammation in plaque progression. These findings underscore the potential of anti-inflammatory therapeutic strategies, alongside lipid-lowering interventions, in mitigating atherosclerotic risk. The proposed model provides a robust computational framework for elucidating the interactive effects of lipid metabolism and vascular inflammation in the progression of cardiovascular disease.

Keywords Finite difference, reaction-diffusion, LDL, C-reactive protein (CRP), lipoprotein(a)

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1. Introduction

Atherosclerosis is a significant cardiovascular disease responsible for high mortality worldwide [1–4]. As illustrated in Figure 1, the process begins when low-density lipoprotein (LDL) penetrates the intimal layer of the arterial wall, initiating an inflammatory response that eventually leads to the formation of fibrous plaques. Fat buildup is a more complicated process described as an inflammatory response type 1, creating oxidative stress in the artery wall, which lays down plaques that obstruct blood flow. Low-density lipoprotein (LDL), lipoprotein(a), and C-reactive protein (CRP) drive lipid peroxidation, inflammation and spurring the development

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leading accelerators of the atherosclerotic process. We will explore and discuss these mechanisms as well as disease progression, and potential therapeutic interventions according to mathematical model frameworks. An important trend for the simulation of atherosclerosis is the use of reaction–diffusion models that consider both molecular and biochemical levels [5–7].

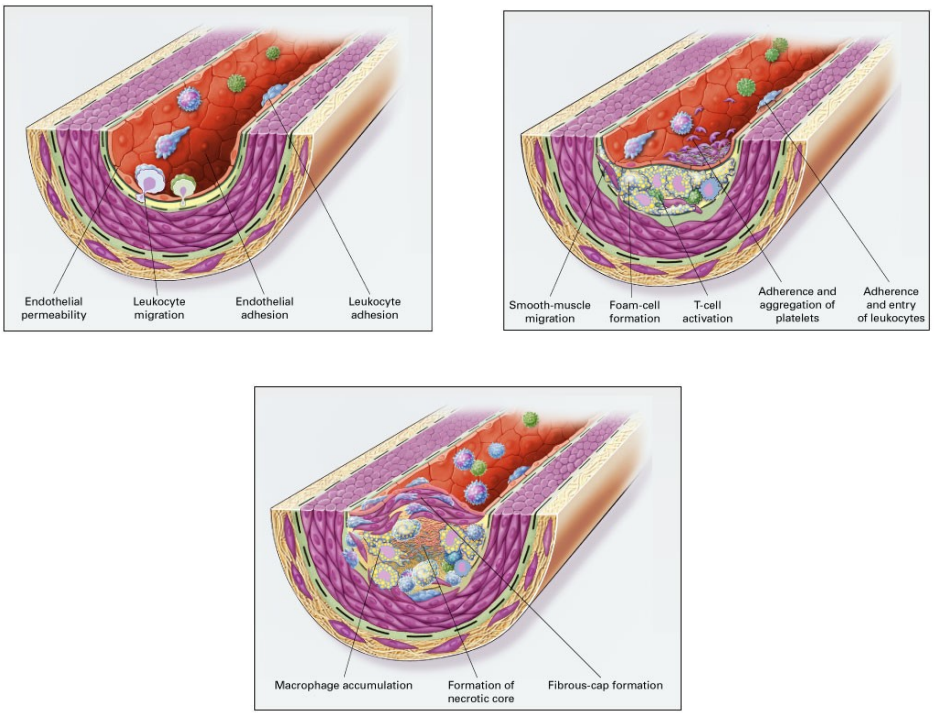

Figure 1. Early events of atherosclerosis. LDL penetrates in the intima, thus triggering an inflammatory process. The atheromatous plaque is eventually covered by a fibrous cap made of smooth muscle cells [8].

The atherosclerosis inflammatory processes have been modeled mathematically [9, 10]. The governing equations used in low-density lipoprotein (LDL) oxidation were either ordinary differential equation (ODE) [11,12] or partial differential equation (PDE) [13–17].

Research revealed the importance of high-density lipoprotein and C-reactive protein in forming atherosclerotic plaques. Low-density lipoprotein, often referred to as "bad cholesterol" is a lipoprotein that transports cholesterol to peripheral tissues where it can accumulate in the arteries and oxidize.

Oxidized low-density lipoprotein (Ox-LDL) is especially bad because it leads to the development of atherosclerotic plaques: Ox-LDL triggers an inflammatory response, recruiting immune cells like macrophages to take up the LDL and morph into foam cells (iconic in their plaque-harboring stature). C-reactive protein, an acute phase reactant that is released in circulation during infection and inflammation and which promotes low density lipoprotein oxidation, as above, also plays a key role in platelet activation (via transcription-induced arachidonic acid release), and the immune response to damaged endothelial tissue: C-reactive lipoprotein released by macrophages enhances monocyte chemotactic sites in plaque-mediated