



Numerical Simulation of Low-Density Lipoprotein Concentration Boundary Layer in a Straight Artery and the Effects of Wall Shear Stress

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ABSTRACT: Low-density lipoprotein, which is recognized as bad cholesterol, typically has been regarded as the main cause of atherosclerosis. An abnormal accumulation of low-density lipoprotein in the artery wall and, as a result, in the formation of oxide, can lead to atherogenesis. Therefore in present study, the concentration boundary layer of low-density lipoprotein in a straight artery is investigated numerically. The governing equations consist of continuity, momentum conservation, and the particles transport in the blood based on appropriate boundary conditions have been solved using one of the most powerful computational fluid dynamics techniques known as the Projection method. Results are obtained and presented as profiles and contours of concentration, blood velocity and wall shear stress, which are in good agreement with numerical and analytical results of previous studies. Effects of factors such as filtration velocity and wall shear stress on the low-density lipoprotein surface concentration and concentration boundary layer thickness are investigated. The results show that increasing the wall suction (high blood pressure) and reducing the Wall Shear Stress results in an increase in surface concentration. Increasing Reynolds number and Schmidt number decreases the concentration boundary layer thickness, and surface concentration increase about 7% higher than that of the bulk flow.

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1- Introduction

Atherosclerosis is a common type of Cardiovascular Disease (CVD) generally found in large and medium arteries [1]. It is well accepted that the situation of atherosclerosis may be because of the unusual accumulation of macromolecules, like Low-Density Lipoproteins (LDLs), in the arterial wall [2, 3]. The macromolecular congestion causes narrowing and blockage of the arteries [4]. This fact indicates that there must be a relationship between macromolecular transport in the arterial wall and the formation of atherosclerosis. So, the knowledge gap is that in case atherogenesis has started with this unusual high accumulation, or that this disorder is just a secondary effect of atherogenesis [5]. Therefore, there is a lot of interest in the mathematical modeling of macromolecular transport (e.g. LDLs), in the wall.

In general, there are three methods for studying the transport of arterial mass, experimental, analytical and numerical approaches. Experimental methods are powerful, but they also have important errors. This includes the prevalence of common diseases between humans and animals, the proper change in control of relevant test conditions, and the problem of measurements by appropriate spatial resolution [6].

To the best of our knowledge, the low-density lipoprotein concentration distribution within a straight artery boundary layer was not considered in the previous works. Hence, the

goal of our work is to study the LDL concentration boundary layer characteristics such as boundary layer thickness and distribution and the LDL Surface Concentration (LSC).

2- Methodology

LDL concentration boundary layer and species transport in a straight artery are studied numerically. For simplicity, the following assumptions have been applied:

- The blood is a homogeneous, Newtonian, incompressible fluid with constant properties.
- The vessel has a constant circular cross section.
- The arterial wall assumed to be permeable with a constant filtration rate.
- The rate of diffusive mass flux into the arterial wall is equal to the convective one at the surfaces.

Fig. 1 shows a schematic of a straight artery. Considering the above assumption, the governing equations consisting of continuity, momentum conservation, and LDL mass transport can be written as follows:

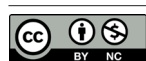
$$\nabla \cdot \vec{V}' = 0 \quad (1)$$

$$\rho \frac{\partial \vec{V}'}{\partial t'} + \rho \vec{V}' \cdot \nabla \vec{V}' = -\nabla P' + \mu \nabla^2 \vec{V}' \quad (2)$$

$$\frac{\partial c'}{\partial t'} + \vec{V}' \cdot \nabla c' = D_e \nabla^2 c' \quad (3)$$

where \vec{V}' is the velocity vector, P' the pressure, ρ the density,

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μ the dynamic viscosity of blood, c' the LDL concentration and D_e the mass diffusivity of LDL into the blood.

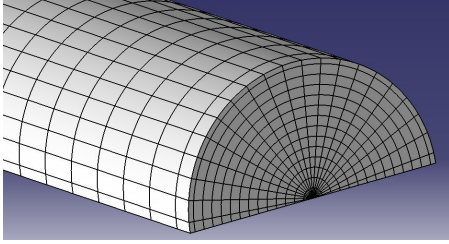


Fig. 1. Schematic of a straight artery with staggered mesh

We consider the inlet velocity profile to the lumen is fully developed. A fully developed parabolic velocity distribution is governed by the velocity profile at the entrance of the lumen. Therefore, the following boundary conditions can be applied at the inlet, at the outlet, at the centerline of the vessel and at the walls.

$$u = U_0 \left[1 - \left(\frac{r}{R} \right)^2 \right], \quad v = 0, \quad \frac{c}{c_0} = 1 \quad (4)$$

$$\frac{\partial u}{\partial x} = 0, \quad \frac{\partial v}{\partial x} = 0, \quad \frac{\partial c}{\partial x} = 0 \quad (5)$$

$$\frac{\partial u}{\partial r} = 0, \quad v = 0, \quad \frac{\partial c}{\partial r} = 0 \quad (6)$$

$$u = 0, \quad v = V_w, \quad -D_e \frac{\partial c}{\partial r} + V_w c = 0 \quad (7)$$

In this study the projection method which is introduced by Chorin [7] for the first time is employed to solve the transient Navier–Stokes equations using forward in time and central in space finite difference discretization. Although the unsteady solutions are physically accurate here, the concentration is focused on the steady state solutions where the transient terms vanish. In the projection algorithm, the Navier-Stokes and continuity equations are written as:

$$\frac{V^{n+1} - V^n}{\Delta t} + A(V^n) + \nabla p^{n+1} = \frac{1}{Re} \nabla^2 V^n \quad (8)$$

$$\nabla V^{n+1} = 0 \quad (9)$$

Then, defining the temporary velocity, V^* , Eq. (8) can be split into Eqs. (10) and (11)

$$\frac{V^* - V^n}{\Delta t} + A(V^n) - \frac{1}{Re} \nabla^2 V^n = 0 \quad (10)$$

$$\frac{V^{n+1} - V^*}{\Delta t} + \nabla p^{n+1} = 0 \quad (11)$$

Taking the divergence of Eq. (11) and using Eq. (9) leads to the Poisson equation for the pressure:

$$\nabla^2 p^{n+1} = -\frac{1}{\Delta t} \nabla V^* \quad (12)$$

The Neumann conditions for the pressure at the boundary is obtained by projecting Eq. (11) normal to the boundaries

$$\left(\frac{\partial p}{\partial n} \right)_\Gamma^{n+1} = -\frac{1}{\Delta t} (V_\Gamma^{n+1} - V_\Gamma^*) \cdot n \quad (13)$$

where the subscript Γ indicates the boundary. Due to explicit discretization, the following stability conditions must be satisfied

$$\frac{\Delta t}{Re \text{Min}\{(dx)^2, (dr)^2\}} \leq \frac{1}{6}$$

$$\text{Max}(u^2 + v^2) Re \Delta t \leq 2 \quad (14)$$

3- Results and Discussions

The effects of Reynolds number on the LSC and concentration boundary layer are shown in Figs. 2 and 3, respectively. As can be seen, the Reynolds number influences the accumulation of the LDL particles at the surface and its boundary layer thickness. By increasing the Reynolds number, both concentration and its boundary layer thickness decrease due to the dominant interaction between the blood and the arterial walls.

4- Conclusions

The main results drawn from the study can be summarized as follows:

- The LSC is about 4 to 10% higher than bulk values. By increasing filtration velocity, the LSC increases linearly to 8%.
- By increasing the wall shear stress, the LSC decreases to a constant value.
- The LSC and its boundary layer thickness decrease with the Reynolds number.
- The concentration boundary layer occupies about 4% of the radius of the artery.

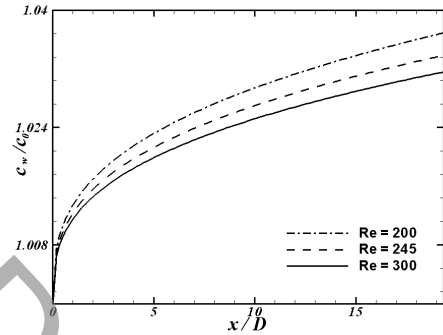


Fig. 2. Effect of Reynolds number on LSC

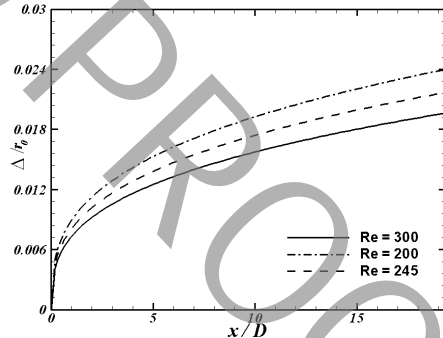


Fig. 3. Effect of Reynolds number on the concentration boundary layer thickness

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