

The blood is Newtonian in LDL deposition through artery walls

The formation of an atherosclerotic plaque causes various cardiovascular diseases, such as a heart attack. In this process, the Low-Density Lipoprotein (LDL) has a main role, since it infiltrates in the artery wall and promotes the process, causing a restriction of the lumen. Understanding in detail how the LDL deposits in the arterial wall is very important for the scientists, in order to see in detail how the atherosclerotic plaque grows, and to find innovative techniques to stem this phenomenon. Concentration values across the wall can be known by solving conservation equations on a geometrical reconstruction of the artery. Assuming that the arterial wall is heterogeneous, it is possible to understand in which layer there is more LDL deposition, and how this phenomenon is affected by other physical factors such as hypertension or hyperthermia. A sketch of an arterial wall is presented in Figure 1.

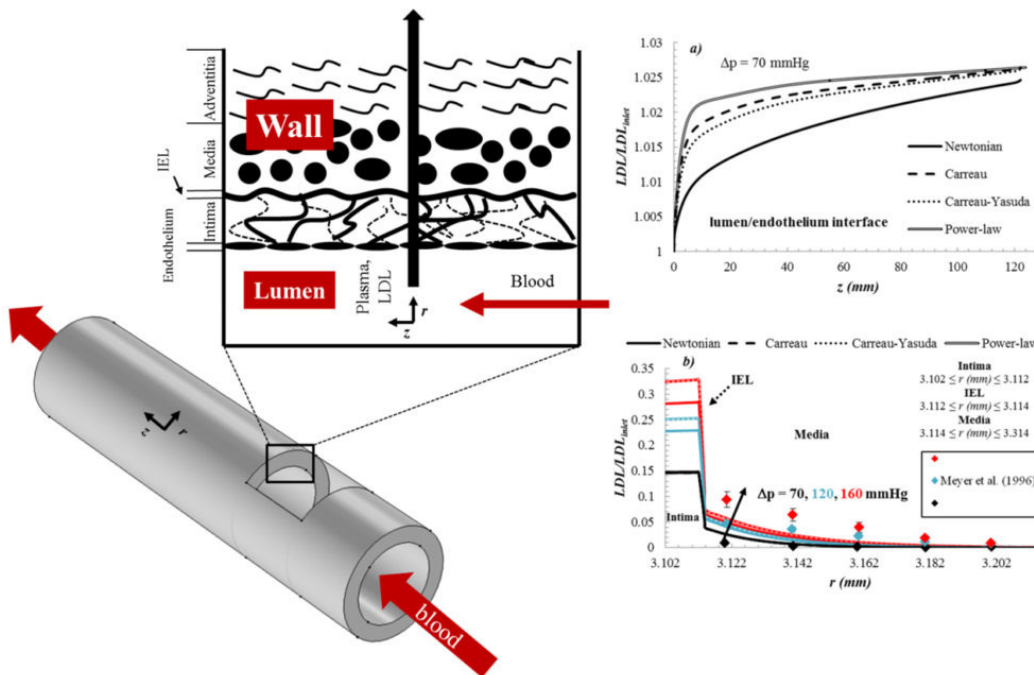


Fig. 1. A sketch of the arterial wall with a) LDL concentration along the lumen/endothelium interface for different viscosity models and b) across the arterial wall for various viscosity models and hypertension (Δp).

In order to solve conservation equations, physical properties of the blood are required. One of these magnitudes is the viscosity, which characterizes the resistance offered by a fluid to its movement. An example of high-viscosity fluid is the oil. Depending on the relationship between viscosity and velocity variations, a fluid can be classified as Newtonian (linear relationship) or non-Newtonian (non-linear relationship). If we want to characterize the behavior (velocity, pressure or other fluid-dynamics magnitudes) of a fluid, then assuming the fluid to be Newtonian simplifies the solution of the problem.

An example of non-Newtonian fluid is the blood. Its non-Newtonian nature is caused by the presence of blood cells suspended in blood plasma. This means that characterizing in detail the fluid-dynamic

behavior of blood is rather complex. However, there are some particular fluid-dynamic conditions for which the blood can be modeled as a Newtonian fluid. These conditions occur in large vessels like medium or large arteries.

In this study, it is demonstrated that in medium and large arteries the blood can be assumed to be Newtonian when LDL deposition needs to be characterized. This greatly simplifies the complexity of the problem. In Figure 1a, the LDL concentration on the lumen/endothelium interface along the flow direction is presented. The infiltration of LDL through the wall, ruled by a membrane called endothelium, causes an increase of LDL concentration. The LDL concentration along the radial direction is presented in Figure 1b. Among the various layers, it is important to observe that predicting the LDL deposition in the intima layer is very important, since this is the layer in which the atherosclerotic plaque takes place. Effects of hypertension (increasing pressure differences D_p) are also presented, together with comparisons with experimental data from literature. It is shown that hypertension increases LDL. From the above results, it is possible to conclude that for medium and large arteries a Newtonian fluid assumption can be employed in modeling LDL deposition.

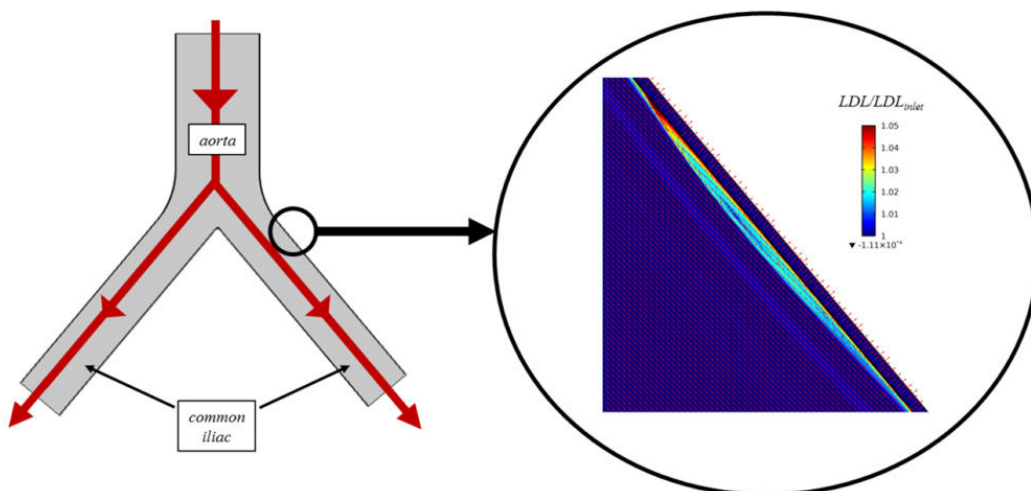


Fig. 2. LDL concentration fields in the common iliac wall: the red arrows represent the blood flow.

Further, studies carried out on the aorta-iliac bifurcation showed that a non-Newtonian model might be better when the geometry becomes more complex, as it occurs in a bifurcation. In Figure 2 the aorta-iliac bifurcation model is represented, together with the concentration field for a Newtonian model. It is shown that there is a local increase in the bifurcation zone. This local increase might not be predicted when non-Newtonian models are employed.

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Publication

[Analysis of non-Newtonian effects on Low-Density Lipoprotein accumulation in an artery.](#)

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