Atherosclerosis is a major burden for all societies and there is a great need for a deeper understanding of involved key inflammatory, immunological and biomechanical processes. A decisive step for the prevention and medical treatment of atherosclerosis is to predict what conditions determine whether early atherosclerotic plaques continue to grow, stagnate or become regressive. The driving biological and mechanobiological mechanisms that determine the stability of plaques are yet not fully understood. We develop a spatially resolved and quantitative mathematical model of key contributors of early atherosclerosis. The stability of atherosclerotic model plaques is assessed to identify and classify progression-prone and progression-resistant atherosclerotic regions based on measurable or computable in vivo inputs, such as blood cholesterol concentrations and wall shear stresses. The model combines Darcy’s law for the transmural flow through vessel walls, the Kedem-Katchalsky equations for endothelial fluxes of lipoproteins, a quantitative model of early plaque formation from a recent publication and a novel submodel for macrophage recruitment. The parameterization and analysis of the model suggest that the advective flux of lipoproteins through the endothelium is decisive, while the influence of the advective transport within the artery wall is negligible. Further, regions in arteries with an approximate wall shear stress...
exposure below 20 % of the average exposure and their surroundings are potential regions where progression- prone atherosclerotic plaques develop.

Stichworte:
atherosclerosis, parameter estimation, stability analysis, macrophage recruitment, transmural flow, lipoprotein flux

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