A coupled model for the formation of Atherosclerosis due to inflammation processes

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ABSTRACT

Atherosclerosis is a disease in blood vessels that often results in plaque formation and lumen narrowing. It is an inflammatory response of the tissue caused by disruptions in the vessel wall nourishment. Blood vessels are nourished by nutrients originating from the blood of the lumen. In medium-sized and larger vessels, nutrients are additionally provided from outside through a network of capillaries called vasa vasorum. It has recently been hypothesized [1] that the root of atherosclerotic diseases is the malfunction of the vasa vasorum. This, so called outside-in-theory, is supported by a recently developed numerical model [2] accounting for the inflammation initiation in the adventitial layer of the blood vessel.

Building on the previous findings, this presentation proposes an extended material model for atherosclerosis formation that is based on the outside-in-theory. Beside the description of growth kinematics and nutrient diffusion, the roles of monocytes, macrophages, foam cells, smooth muscle cells and collagen are accounted for in a nonlinear continuum mechanics setting. Cells are activated due to a lack of vessel wall nourishment and proliferate, migrate, differentiate and synthesize collagen, leading to the formation of a plaque. Numerical studies show that the onset of atherosclerosis can qualitatively be reproduced. Thus, the in silico model backs the new theory.

REFERENCES

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