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A multiphase spatial model for HDL-assisted stabilisation of early atherosclerotic plaques

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Atherosclerosis is among the leading causes of death worldwide due to its implication in heart attacks and strokes. The disease is characterised by the localised thickening of artery walls due to the buildup of fatty cholesterol-filled streaks. A key factor in determining whether an atherosclerotic plaque becomes problematic is the interplay between low density lipoprotein (LDL) and high low density lipoprotein (HDL), which are responsible for transporting cholesterol around the body. LDL is pro-atherogenic and its buildup in the artery wall will trigger an immune response whereby the recruited immune cells become engorged on lipid and remain in the artery wall. HDL in contrast has the atheroprotective properties of enabling lipid export and the egress of immune cells from the plaque. Plaque dynamics consist of many nonlinear interactions between various cellular and biochemical species including the interactions between immune cells and lipoproteins.

In this talk, we present a multiphase PDE model for an early stage atherosclerotic plaque. The model accounts for interactions between macrophages, apoptotic cells, and lipids. We model the plaque on a 1D domain with a moving boundary. Our model is based on a multiphase framework, and incorporates the effects of cell crowding by having the domain expand or contract according to the total amount of material in the plaque. We discuss how this model gives insight into how early plaque growth and regression depends on the levels of LDL and HDL in the bloodstream, and the roles of foam cell egress and HDL-enabled reverse cholesterol transport in slowing plaque growth and preventing the accumulation of potentially problematic levels of apoptotic material.

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