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Multiphase modelling of early fibrous cap formation in atherosclerosis

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Atherosclerotic plaque growth is characterised by a process of chronic, non-resolving inflammation that leads to the accumulation of cellular debris and extracellular fat in the inner artery wall. In advanced plaques, smooth muscle cells (SMCs) are recruited from deeper in the artery wall to synthesise a fibrous tissue cap that sequesters the thrombogenic plaque content from the bloodstream. The fibrous cap therefore provides crucial protection from plaque rupture and the formation of blood clots that occlude vessels and cause heart attacks and strokes. Despite the important role played by the plaque fibrous cap in preventing the clinical consequences of atherosclerosis, the mechanisms that underlie cap formation remain poorly understood. In particular, it is unclear why certain plaques become strong and stable while others become fragile and dangerously vulnerable to rupture.

In this talk, we discuss the use of a multiphase approach with non-standard boundary conditions to investigate early fibrous cap formation in the intimal layer of the artery wall. We model the highly nonlinear process of SMC migration from the media in response to a diffusible chemical signal produced at the endothelium. Simulations indicate that the emergence of a stable fibrous cap requires a critical balance between the relative rates of cell supply from the media, chemotactic migration within the intima and cell loss by apoptosis (or phenotype change). Moreover, we identify a number of disease-associated parameters that may be linked to variations in cap stability. This model represents the first detailed *in silico* study of fibrous cap formation in atherosclerosis, and establishes a framework that can be extended to investigate other aspects of plaque development.

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