A coupled multiphysics approach for modeling in-stent restenosis

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Restenosis refers to the uncontrolled growth of tissue in vessel walls as part of an inflammatory re-sponse that follows cardiovascular interventional procedures including balloon angioplasty and stent implantation. Although the risk of restenosis has reduced with the advent of drug-eluting stents, it is not completely eliminated. An in silico replication of neointimal hyperplasia, the mechanism be-hind restenosis, shall therefore provide the necessary means to derive in-sights about the biochemical and cellular interactions within the vessel wall, and eventually address the risks of restenosis in a patient-specific manner. In this regard, the interactions between four important biochemical species in the vessel wall are modeled within the scope of this work. They are platelet-derived growth factor (PDGF), transforming growth factor (TGF)-β, extracellular matrix (ECM) and smooth muscle cells (SMC). The complex interactions between these species are then coupled to a continuum mechanical model of the vessel wall embedded with a finite growth theory, where the local SMC density drives the growth process and the local ECM (hence collagen) concentration controls the compliance of the vessel wall. Multiphysics-based frameworks for modeling damage-driven growth and remodeling have been presented in several earlier works [1, 2], but the presented model specifically addresses the inflammatory response due to endothelium denudation.

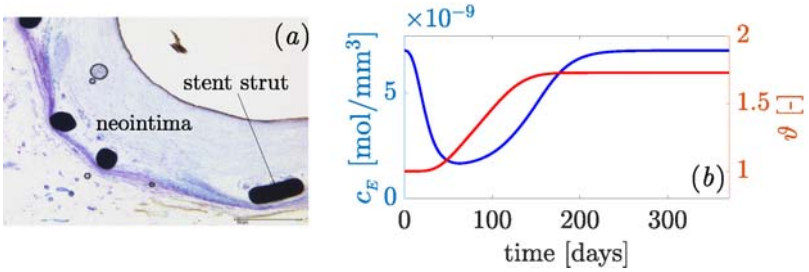

Fig. 1: (a) restenosis in rat aorta [3] (b) evolutions of ECM and growth stretch

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