Abstract Submitted
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A Mechanical and Biochemical Model of Intimal Atherosclerotic Lesions[1] PAK-WING FOK, University of Delaware, REBECCA VANDIVER, St. Olaf College — We investigate a 1D axisymmetric model of intimal hyperplasia using hyperelasticity theory. Our model incorporates growth of the intima due to cell proliferation which in turn is driven by the release of a cytokine such as Platelet-Derived Growth Factor (PDGF). The main novelty of our model is that the growth rate is tied to local stresses and the local concentration of PDGF. The resulting system is a triple free boundary problem with different regions of the vessel wall having different homeostatic stress, depending on the local PDGF concentration. This system is coupled to force-balance equations that yield distributions for the stress and deformation. We find that rapid intimal thickening coupled to a quiescent media puts the intima in a state of compression and results in slow time scales of evolution. Our results are compared with intima-media thickness measurements of carotid arteries from previous clinical studies.

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