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Author: M. Garbey M. Rahman S. Berceli

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A Multiscale Computational Framework to Understand Vascular Adaptation

M.Garbey [†,*] , M. Rahman [†] and S.Berceli [†,††]

† Dept. of Biology, University of Houston, USA

†† Dept. of Surgery, University of Florida, Malcom Randall VAMC, USA

* MITIE, The Houston Methodist Hospital, Houston USA.

Highlights

- We develop a computational framework for multiscale modeling of vein graft adaptation that couple environmental condition described with PDEs and tissue adaptation at the cellular level described with agent base model.
- The implementation is designed to facilitate dialogue between computational scientist and MDs and accelerate the iterative process on model improvement: it is modular, simple and computationally efficient.
- We have been able to reproduce interesting pattern observed on our animal model and have a tool to question the impact of new potential control on the biology such as proliferation rate or inflammation.

Graphical abstract**Professor Marc Garbey**

Dr. Scott A. Berceli, M.D.

Mahbubur Rahman

Abstract

The failure rate for vascular interventions (vein bypass grafting, arterial angioplasty/stenting) remains unacceptably high. Over the past two decades, researchers have applied a wide variety of approaches to investigate the primary failure mechanisms, neointimal hyperplasia and aberrant remodeling of the wall, in an effort to identify novel therapeutic strategies. Despite incremental progress, specific cause/effect linkages among the primary drivers of the pathology, (hemodynamic factors, inflammatory biochemical mediators, cellular effectors) and vascular occlusive phenotype remain lacking. We propose a multiscale computational framework of vascular adaptation to develop a bridge between theory and experimental observation and to provide a method for the systematic testing of relevant clinical hypotheses. Cornerstone to our model is a feedback mechanism between environmental conditions and dynamic tissue plasticity described at the cellular level with an agent based model. Our implementation (i) is modular, (ii) starts from basic mechano-biology principle at the cell level and (iii) facilitates the agile development of the model.