Coupled discrete and continuum methods for modelling of atherosclerotic disease in the coronary arteries

Nenad Filipovic*, Zarko Milosevic*, Igor Saveljic*, Themis Exarchos† and Oberdan Parodi††

* Faculty of Engineering, BioIRC Kragujevac
University of Kragujevac
Sestre Janjica 6, 34000 Kragujevac, Serbia
e-mail: fica@kg.ac.rs, web page: http://www.bioirc.ac.rs

† Foundation of Research and Technology Hellas
-Biomedical Research Institute, GR45110, Ioannina, Greece
e-mail: themis.exarchos@gmail.com

†† National Research Council Pisa, Italy
e-mail: oberdan.parodi@virgilio.it

ABSTRACT

In the large arteries when the blood vessel endothelial dysfunction started there is process of lipipd, cholesterol, calcium and cell elements accumulation in the vessel wall which we denote as atherosclerosis disease.

In this study we coupled discrete and continuum methods Dissipative Particle Dynamics (DPD) and Finite Element (FE) to simulate process of formation and progression of the plaque disease in the coronary artery for real patient data.

In the discrete DPD approach blood flow is modeled as a movement of the collection of DPD particles and a motion of each individual DPD particle is described by the Newton law equation. Additionally to three standard forces the conservative (repulsive), dissipative and random interaction forces, we introduced additional binding force for formation of the foam cells.

Continuum approach is governed by the Navier-Stokes equations, together with the continuity equation. Mass transfer within the blood lumen and through the arterial wall is coupled with the blood flow and is modeled by the convection-diffusion equation. LDL transport in lumen of the vessel is described by Kedem-Katchalsky equations. The formation of the atherosclerosis is solved using three additional reaction-diffusion partial differential equations [1]. Coupled DPD and FE approach shows lipids concentration in the intimal area within correlation of the low and for oscillatory shear stress zone.

We run simulation on the group of 5 patients which predicts new plaque formation after 12 to 24 months follow up which corresponds to size and plaque composition [2]. Concentration of macrophages obtained by computer simulation indicates that there is a newly formed matter in the intima, especially in the region before LAD bifurcation.

Matching of plaque location, size and composition progression in time between clinical and coupled DPD and FE computer model shows a potential benefit for future prediction of this vascular decease.

REFERENCES
