Abstract: Oxidative and nitrosative stress has been implicated in pathogenesis of many diseases including cardiovascular diseases, atherosclerosis, diabetes, and ischemia/reperfusion injuries. An excess formation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) leads to oxidative and nitrosative stress, respectively. Over past four decades, many researchers using many experimental methods have evaluated NO, ROS and RNS physiological and pathophysiological roles. We do not have quantitative knowledge of the role of microvascular architecture and how interactions among these NO, ROS and RNS species with many other enzyme systems are controlled and affected in vascular tissue leading to dysfunctions and damages. Based on signal transduction pathways, biochemical kinetics and biotransport, we have developed a series of computational models spanning over several levels of biological organization to address important biological questions. The significance of these models is in their predictive ability and hypotheses development and testing for vasoregulation and endothelial cell dysfunction. In particular, I will present results on biochemical pathways interactions of NO, ROS and RNS in endothelial cell model, and the role of leukocyte adhesion in vascular tissue.

Date: Thursday March 8th, 2012
Time: 12:00pm – 1:00 pm
Location: BME 2220 – Bioengineering Center
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