

Kaunas to give biomedical engineering seminar Dec. 2

Texas A&M University

Dr. Roland Kaunas, assistant professor in the Department of Biomedical Engineering Texas A&M University, will give a talk Wednesday (Dec. 2) at 3:30 p.m. in Room 203 of the Zachry Engineering Center.

Kaunas's talk, "Coping with Stress — at the Cellular Level," is part of the biomedical engineering department's seminar series.

Abstract

Endothelial cell dysfunction contributes to early atherosclerotic plaque formation by recruiting inflammatory cells and disrupting endothelial barrier function leading to accumulation of LDL in the arterial wall. Plaques typically initiate at branches and curvatures in the arterial tree, where the mechanical environment facilitates chronic activation of proatherosclerotic signaling in the endothelium. Interestingly, endothelial cells tend to align along the vessel axis throughout the arterial tree — except at these proatherosclerotic sites. Our research indicates that such alignment is an adaptive mechanism that allows endothelial cells to cope with their mechanical environment.

Fluid shear stress and cyclic circumferential strain each induce cell alignment in a manner dependent on actin stress fiber reorganization. Theoretical models and experimental results will be presented to describe a mechanochemical mechanism regulating stretch-induced spatio-temporal changes in stress fiber tension and orientation. These studies indicate that stress fibers reorganize in order to maintain their tension at an equilibrium level dependent on myosin activity. Stress fiber alignment acts to decrease stretch-induced elevations in fiber turnover and tension that disrupt endothelial cell-cell junctions and stimulate proinflammatory signaling (JNK, p38) and gene expression.

Similarly, fluid shear stress-induced endothelial cell and stress fiber alignment also results in modulating cell-cell junction integrity and proinflammatory signaling. In summary, we propose that endothelial cells in healthy, straight segments of the arterial tree can adapt to their mechanical environment, but endothelial cells are unable to cope with the mechanical environment at branches and curvatures.

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