BIOMATERIALS

Forces in Biomolecular Systems SFB 863

Signals from the matrix:

Are endothelial cells responding to changes in the matrix elasticity?

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Abstract:

Our studies show, that upon shear stress stimulation, endothelial cells (EC) actively release the growth factor FGF-2. This release of FGF-2 is initiated by protease activation, a process that is further controlled by specific cell matrix adhesion. Proteolytic digestion of the extracellular matrix change its stiffness and might have profound effects on cell matrix interactions. However, up to date it is studied insufficiently how such matrix modifications are established and whether they influence on EC phenotype. We try to address that question, drawing the hypothesis that, similar to stem cells, the phenotype of EC might be dependent on the elastic properties of the extracellular matrix (e.g. the vascular wall). We pay special interest on cellular phenotypes and signaling responses induced by changes in the matrix stiffness. Since during vascular remodeling processes, matrix proteases are often activated and seem to be critically involved in generation and maintenance of signaling cascades in adaptive vascular remodeling, we propose that due to those proteolytic activities the matrix stiffness is altered and influences EC phenotype. Indeed, fragmentation of matrix proteins changes the microarchitecture and show modulator properties for adhesion dependent signaling. Moreover, EC grown on a soft matrix tend to go into apoptosis. Cells grown on intermediate flexible matrices are intending to form new capillary-like structures, and, finally, cells on stiff matrices are highly proliferative. This in vitro culture regime might serve as a model system to study not only development and progression of vascular aneurysms but also of arteriosclerosis and high blood pressure.

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