

ΣΕΜΙΝΑΡΙΟ

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ΘEMA:	Applications of Cellular and Tissue Engineering in the Cardiovascular System
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ΠΕΡΙΛΗΨΗ:

The ultimate goal of my research is to improve human health by utilizing the cardiovascular system on the molecular, cellular, and tissue level. Understanding how the mechanical environment affects tissue development, maintenance and remodeling *in vivo*, might be used to engineer better quality tissue grown *in vitro*.

Cardiac Muscle Tissue Engineering

1.5 million people in the United States sustain a myocardial infarction each year, an event often associated with diminished cardiac function. We propose that implantation of cardiac muscle tissue equivalents, with the proper structure and function, could potentially improve regeneration of cardiac muscle. In addition to the *in vivo* application, these tissues could provide a useful *in vitro* model system to test newly developed pharmaceuticals, and to study basic aspects of cardiac muscle physiology and pathophysiology. Cardiac muscle can be engineered *in vitro*, using freshly isolated neonatal rat ventricular cells cultured on three dimensional fibrous polymer meshes in tissue culture bioreactors. Engineered tissues had structural, biochemical and molecular cardiac muscle-specific features, and could sustain propagation of electrical impulses (functional characteristics).

Effects of Fluid Shear Stress on Vascular Cells

After cardiovascular interventions such as balloon angioplasty, vascular smooth muscle cells (SMC) may be directly exposed to blood flow and their behavior may be modulated by the local hemodynamic environment. To systematically study the contribution of fluid shear stress to SMC function, cultured human aortic smooth muscle cells were subjected to physiological levels of shear stress (5-25 dyn/cm²) using parallel plate flow chambers. Fluid shear stress decreased the growth rate of SMC and this was not a result of cell injury, which is consistent with *in vivo* observations in which it was found that areas of low shear stress were associated with greater intimal thickening. Exposure of SMC to shear stress resulted in a rapid nitric oxide release, an event which may play a regulatory role in the blood vessel wall in the absence of endothelium following vascular injury, and may also be important in normal vessel homeostasis. In addition, shear stress differentially mediated gene and protein expression in SMC.