Joint Department of

BIOMEDICAL ENGINEERING

UNC CHAPEL HILL | NC STATE UNIVERSITY

Coulter Seminar Series Presents

"Dissecting cell-specific contributions to pulmonary fibrosis using new biomaterials and microphysiologic models"

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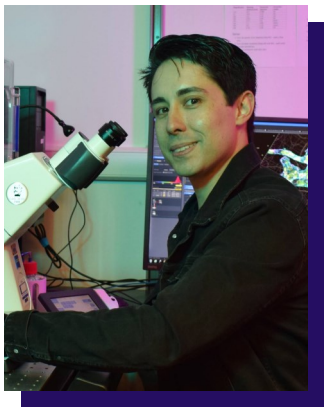

Prof. Brendon Baker directs an NIH- and NSF-funded lab that studies how structure and mechanics of the cellular microenvironment guide fundamental biologic processes including cell adhesion, migration, proliferation, and extracellular matrix synthesis. His lab focuses on the microenvironment of a broad class of tissues including stromal, interstitial, or connective tissues. Though highly varied across organs and tissues, these tissues consistently provide the scaffolding and plumbing that vitally support tissue parenchyma, and as such, are also key settings of disease. To construct these tissue spaces, his lab uses microfabrication techniques and biomaterials to create engineered microenvironments that are 3D, fibrous, and contain microvasculature. Combined with molecular tools, live imaging techniques, and multi-scale mechanical characterization, these engineered settings allow his lab to model, study, and control the interactions between cells and their connective tissue surroundings.

ABSTRACT

The hallmark of fibroproliferative disorders is an overabundance of myofibroblasts, cellular mediators of wound healing that contract and excessively secrete extracellular matrix (ECM) to cause eventual organ failure. Although the cellular precursors and signals that engender myofibroblasts have been studied, effective treatments to halt fibrotic progression do not yet exist. The endothelium and angiogenesis play a critical role in the wound healing response to injury and in vivo lineage tracing studies have confirmed endothelial cells (ECs) contribute to multiple organ-specific fibroses. During angiogenesis, endothelial tip cells adopt mesenchymal traits including proteolytic activity and invasive, migratory behavior in order to lead invading angiogenic sprouts and extend the microvasculature into the surrounding interstitium. In this talk, I'll describe our recent studies using synthetic biomaterials and microphysiologic models to understand how fibrotic changes inthe tissue microenvironment modulates myofibroblast activation and dysregulates angiogenesis to co-opt fibroblasts and endothelial cells into disease-driving cells.

Friday, October 29th
12:00 Noon

Presented From: 321 MacNider Hall (UNC)
Videoconferenced to: 4142 Engineering Building III (NC State)