



**REGIONAL CENTRE FOR BIOTECHNOLOGY**

**Seminar series**

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**Epithelial-specific TGF- $\beta$  signaling of the lung: Evidence for an epithelial role in Pulmonary Fibrosis**

**Dr. Sai Krishnaveni Manda, PhD**

**University of Southern California, Los Angeles**

**Friday, 17th, August, 2011**

**11:00 AM**

**Seminar Room**

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### Abstract

Idiopathic pulmonary fibrosis (IPF) is a progressive disorder characterized by extracellular matrix (ECM) deposition and remodeling by  $\alpha$ -smooth muscle actin (SMA)-expressing myofibroblasts. Epithelial injury is thought to be central to disease pathogenesis with release of mediators triggering fibroblast activation/proliferation. In addition, injured epithelial cells may directly contribute to fibroblast accumulation by undergoing epithelial-mesenchymal transition (EMT). TGF- $\beta$  and its signaling pathway has evolved as key mediator of both EMT and fibrosis. In this seminar, we will delineate how TGF- $\beta$  contributes to EMT, and how depletion of TGF- $\beta$  signaling confers resistance to bleomycin-induced pulmonary fibrosis in an *in vivo* mouse model thus emphasizing a potential therapeutic option. We would also discuss how epithelial cells respond to various extracellular matrices and the signaling pathways involved therein.