

PIONEERS IN BIOMEDICAL RESEARCH SEMINAR

Presented by the Fralin Biomedical Research Institute at VTC and co-sponsored by the institute's Cancer Research Group



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Control of TGF-beta Signaling and Epithelial-Mesenchymal Transition

Epithelial-mesenchymal transition (EMT) is a normal differentiation process during development that enables epithelial cells to partially or completely repress epithelial junctions and reprogram gene expression, resulting in more motile and often more invasive cell behavior. EMT, which is generally thought of as a reversible process, allows the cells to reach new destinations and generate new cell populations. EMT is also repurposed pathologically during fibrosis and cancer progression, and allows carcinoma cells to disseminate, while additionally enabling the cells to acquire stem cell characteristics and cancer drug resistance. Studying the activities and signaling pathways of a secreted growth factor and cytokine, named transforming growth factor-beta (TGF-beta), it was shown a long time ago that TGF-beta can induce EMT. Consistent with this notion and its known activities, increased TGF-beta signaling is now seen as a driver of fibrosis and cancer dissemination that leads to metastasis. Dr. Derynck's research highlights the cooperation of two signaling pathways, Smad signaling and Akt-mTOR signaling, in the progression and completion of EMT, and illustrates that prolonged exposure of cancer cells stabilizes EMT, which stands in contrast to the general notion that EMT is always reversible. When compared to reversible EMT, stabilized EMT has increased stem cell properties and cancer stem cell properties that depend on mTOR signaling. The recognition of the roles of EMT in fibrosis and cancer, and the key role of TGF-beta as driver of the EMT process allow for therapeutic opportunities.

FRIDAY, APRIL 30 at 11:00 a.m.

This seminar will be webcast live at fbri.vtc.vt.edu/events/live-webcast and presented via Zoom at <https://virginiatech.zoom.us/j/84567483942>.



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