Department of
Biomedical Engineering
UNIVERSITY OF WISCONSIN–MADISON

Growth Factor Signaling and Metabolic Homeostasis in Single Cells

Fall 2017 Seminar Series
Monday, September 18, 2017
12 - 1 PM
Tong Auditorium (1003 Engineering Centers)

About the Speaker

John Albeck, PhD, is an assistant professor of Molecular and Cellular Biology at the University of California, Davis. He received a B.A. in Biological Sciences from Cornell University in 2000, and completed his doctoral work in Computational and Systems Biology at MIT in 2007, working with Peter Sorger to develop computational single-cell models of programmed cell death. From 2007 to 2013, He was a postdoctoral scholar and Instructor in Cell Biology in the lab of Joan Brugge at Harvard Medical School. With training in both quantitative methods and experimental biology, Dr. Albeck has focused his work on identifying systems-level properties that govern the behavior of cells in both normal tissue and in cancers.

Since 2013, his research group at UC Davis has brought together biologists and engineers to study how multiple pathways are integrated to control cellular metabolism, proliferation, and death. Their approach is centered on the continuing development of technology to image biochemical signaling activities in individual cells, and the analysis of time-resolved data from millions of cells to infer regulatory principles that go awry in the development and progression of cancer.

John Albeck

Assistant Professor of Molecular & Chemical Biology, University of California-Davis

As single-cell technologies expand, it is becoming clear that many cellular signaling events are very dynamic, necessitating a time-lapse approach to capture rapid kinetics within the cell. I will present our work on using live-cell imaging with genetically encoded reporters to approach two long-standing questions in the regulation of cell growth in humans. This work centers on two kinases – ERK and AMPK – that play key roles in homeostasis of healthy tissues and which are currently being targeted by cancer therapies.

The first question is how the dynamics of ERK activity encode information from extracellular stimuli, allowing different growth factor receptors to activate distinct gene expression programs using the same intermediate signaling pathways. Mutations in this pathway, and candidate therapies targeting these mutations, exert unique effects on ERK kinetics, making it essential to decode the cellular “language” of ERK activity dynamics.

The second question is how cellular energetics are maintained and balanced despite continuing fluctuations in both the supply of nutrients and the demand created by anabolic processes required for cell growth. We find that the interlinked AMPK, Akt, and ERK pathways undergo surprisingly dynamic fluctuations in response to metabolic stresses, suggesting new models for how cellular homeostasis is maintained on the scale of minutes by coordination of catabolic and anabolic processes.