Biography

Bruce Goode is a Professor of Biology at Brandeis University. He earned his B.S. and his Ph.D. in Cell Biology from the University of California Santa Barbara in the lab of Stuart Feinstein. He was a postdoctoral fellow with David Drubin at the University of California Berkeley. In 2000, Dr. Goode started his lab at Brandeis, where his research program focuses on mechanisms of actin cytoskeleton remodeling in cell motility and intracellular traffic. Projects in the Goode lab are multi-disciplinary in nature, combining in vitro single molecule TIRF imaging, single particle electron microscopy, biochemistry, genetics, and live cell imaging. Dr. Goode was the recipient of the Pew Scholars Award and scholar awards from the March of Dimes and American Cancer Society. He also received a Research Career Development Award from the NIH. He is on the F1000 advisory board and the editorial boards of The Journal of Cell Biology and Molecular Biology of the Cell. He was the Editor-in-Chief of Cytoskeleton from 2009-2016. Noteworthy accomplishments in his lab include: (1) the discovery that formins nucleate actin assembly; (2) demonstration that formin activities in vivo are controlled in diverse ways by direct binding partners (e.g., APC, CLIP-170, Bud6, Bud14, Smy1, and Hof1); (3) elucidation of unconventional forms of Arp2/3 complex regulation by Coronin, GMF, and Abp1; (4) discovery of multi-component mechanisms driving actin disassembly (involving AIP1, Coronin, Cofilin, Twinfilin, and Srv2/CAP); and (5) identifying novel mechanisms for coordinating microtubule and actin dynamics (e.g., by APC, CLIP-170, and Profilin).

Abstract

"Multi-component mechanisms driving actin disassembly"

This talk will address the question of how cells, in a matter of seconds, can rapidly remodel their actin cytoskeleton to change cell shape & polarity, and to re-route intracellular traffic. This has been a long-standing question, because actin filaments are inherently stable polymers that turn over very slowly. I will describe recent observations made by our lab and others that fundamentally change our view of how cellular actin networks are rapidly dismantled, highlighting the roles of seven conserved proteins (Cofilin, Coronin, AIP1, Srv2/CAP, Twinfilin, GMF, and Abp1). Key to this progress has been the combined use of genetics, biochemistry, and multi-wavelength single molecule imaging to define the mechanisms of these proteins in real time. This work has shown that these proteins collaborate in an assortment of multi-component mechanisms to influence actin filament dynamics in novel ways, not observed for individual components. Thus, actin network turnover is regulated by a protein ensemble.