Regulation of cytoskeletal dynamics and transport

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The "Regulation of Cytoskeletal Dynamics and Transport" Minisymposium incorporated exciting new research on intracellular filaments. The talks delved into the regulation of filament dynamics, modes of cross-talk, and motor protein function ranging from the single-molecule to the cellular level. **Ben Woods** (Gladfelter lab, University of North Carolina–Chapel Hill) presented biophysical mechanisms controlling septin polymerization by combining in vitro reconstitution, cell-free extracts, and physical modeling approaches. **Rachel Kadzik** (Munro and Kovar labs, University of Chicago; Wignall lab, Northwestern University) used similar techniques in addition to in vivo observations in *Caenorhabditis elegans* to demonstrate that the alignment of actin filaments in the cytokinetic ring requires templated elongation of new actin filaments within the ring, mediated by plastin, a rapidly bundling actin-binding protein. **Maria Angeles Juanes** (Goode lab, Brandeis University) used bulk pyrene assays and TIRF microscopy to show that EB1 directly inhibits the actin-nucleation capabilities of adenomatous polyposis coli (APC), revealing an unanticipated role for EB1 in actin assembly and suggesting that EB1–APC interactions in cells play roles in governing microtubule–actin cross-talk. Using in vitro assays combined with laser microsurgery and microfluidics, **Amol Aher** (Akhmanova lab, Utrecht University, Netherlands) showed that a microtubule catastrophe suppression factor, CLASP, stimulates microtubule lattice repair, thereby restoring microtubule integrity. Continuing the discussion of microtubule-associated proteins (MAPs), **Kassandra Ori-McKenney** (University of California, Davis) used in vitro reconstitution of purified motor proteins and nonenzymatic MAPs to demonstrate that MAPs exhibit distinct influences on the motility of three classes of microtubule motors, kinesin-1, kinesin-3, and dynein, suggesting a role for the MAP code in directing motor transport in cells. Also related to transport, **Tal Keren-Kaplan** (Bonifacino lab, NIH/NICHD) showed that the adaptor protein SKIP/PLEKHM2 is autoinhibited by an intramolecular interaction between its N- and C-terminal regions and that this interaction is relieved by the binding of the small GTPase, ARL8, enabling SKIP to couple lysosomes to kinesin-1 for movement toward the cell periphery. **Meng-Meng Fu** (Barres lab, Stanford University) demonstrated that mRNA transport is important for myelination in a mouse model lacking the 3'UTR of Mbp (myelin basic protein). These mice display tremors and are hypomyelinated, and their oligodendrocytes fail to transport mRNA properly in 3D cultures. The final three talks focused on cytoskeletal mechanisms related to axon outgrowth and guidance. **Stephanie Gupton** (University of North Carolina–Chapel Hill) described how the actin polymerase VASP undergoes reversible nondegradative ubiquitination, mediated by a pair of E3 ubiquitin ligases, TRIM9 and TRIM67. VASP regulation is necessary for responses of filopodia, growth cones, and axons to the guidance cue netrin-1. **Laura Anne Lowery** (Boston University) discussed how phosphorylation of the microtubule plus-end–tracking protein, TACC3, modulates its ability to bind to microtubules and also affects how it promotes axon outgrowth. Finally, **Bettina Winckler** (University of Virginia) described new work on the poorly understood intermediate filament, nestin, in neurons. Her group discovered that nestin selectively promotes the phosphorylation of the MAP, DCX, by the neuronal kinase cdk5, affecting growth cone morphology and responsiveness to guidance cues.

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