Motors in transport and cytoskeleton remodeling

Julie P. I. Welburn^{a,*} and E. Michael Ostap^{b,*}

^aWellcome Trust Centre for Cell Biology, School of Biological Sciences, University of Edinburgh, Edinburgh EH9 3BF, Scotland, UK; ^bPennsylvania Muscle Institute and Department of Physiology, Perelman School of Medicine at the University of Pennsylvania, Philadelphia PA 19104

The "Motors in Transport and Cytoskeleton Remodeling" Minisymposium covered topics that ranged from the discovery of novel modes of communication between cytoskeletal motors and their filaments to motor regulation, recruitment, and cell function.

Motor remodeling of the microtubule cytoskeleton

An emerging theme this year is that microtubules are not merely passive tracks for motor-driven transport, but may be modified by a range of motors and may dynamically modulate motor activity. **Laurent Blanchoin** (Cytomorphlab, CEA) revealed that stepping microtubule motors in vitro can damage the microtubule lattice by removing tubulin heterodimers. The lattice can be actively repaired by the incorporation of new GTP-tubulin, which stabilizes the integrity of the microtubule lattice. **Julie Welburn** (University of Edinburgh) discussed key roles for Kinesin-13 MCAK and Kinesin-8 Kif18b motors in shortening and remodeling microtubules in mitosis to ensure proper mitotic spindle assembly and positioning. New work showed that the Kif18b motor is a weak microtubule depolymerase that cooperates with Kinesin-13 MCAK during mitosis to rapidly shorten microtubules. **Stephanie Ems-McClung** (Walczak lab, Indiana

University) showed that the RanGTP gradient sets up an effector gradient of Kinesin-14 XCTK2 association with importin alpha/beta. This association specifies the mode of XCTK2 cross-linking by inhibiting anti-parallel microtubule cross-linking, thereby promoting parallel cross-linking near the poles for pole formation. Tommaso Cupido (Kapoor lab, Rockefeller University) presented clever new computational and experimental approaches that resulted in the design of novel spastin inhibitors. These inhibitors were then used to reveal the importance of spastin in nuclear envelope formation after cell division. Serapion Pyrpassopoulos (Ostap lab, University of Pennsylvania) reported that the tension applied to the microtubule could influence the dwell time of processively stepping Kinesin-1 on microtubules, using a three-bead trap assay. Depending on how the microtubule is tethered, the geometry of forces applied on the kinesin and the microtubule vary, influencing the attachment duration of the kinesin.

Motors in cargo transport, filament nucleation, and filament dynamics

John Canty (Yildiz lab, University of California, Berkeley) described how two different dynein cargo adaptors, BicD2 and BicDR1, can influence the mechanical and kinetic properties of cytoplasmic dynein. New work showed that the cargo adaptor can allosterically modulate the motor properties of dynein to tune levels of force production and the velocity of transport. Aga Kendrick (Reck-Peterson lab, University of California, San Diego) explained how the Hook3 dynein cargo adaptor could also interact with the kinesin Kif1c through its C terminus. The reconstituted Dynein-Hook3-Kif1c exhibited either plus or minus end-directed motility. How the directionality of the complex is controlled is an interesting future question. Meng-Meng Fu (Barres lab, Stanford University) discovered novel roles for myosin and dynein motors in mRNA transport in oligodendrocytes. Notably, it was found that motor-mediated switching between actin and microtubule filaments appears to be crucial for normal myelination. Christopher Alexander (Hammer lab, National Heart, Lung and Blood Institute) presented exciting new work in Purkinje neurons on the role of myosin 18A, which has a noncatalytic motor with poorly understood function. Myosin 18A coassembles with myosin-II and beta-PIX to promote dendritic spine maturation by activating Arp2/3-dependent actin polymerization. Daniel Cortes (Maddox lab, University of North Carolina-Chapel Hill) developed computational models to explore the effect of myosin-II kinetics and force-sensitivity on cytokinetic ring contractility. Myosins in contractile ring networks were simulated to have slip, catch, and catch-slip bond activities, which resulted in a large range of contractile behaviors.

DOI:10.1091/mbc.E19-01-0011

Molecular Biology of the Cell Volume 30 Page 734

MBoC is pleased to publish this summary of the Minisymposium on "Motors in Transport and Cytoskeleton Remodeling," held at the 2018 ASCB EMBO Meeting, San Diego, December 10, 2018.

^{*}Address correspondence to: Julie P. I. Welburn (Julie.Welburn@ed.ac.uk) or E. Michael Ostap (ostap@pennmedicine.upenn.edu).

^{© 2019} Welburn and Ostap. This article is distributed by The American Society for Cell Biology under license from the author(s). Two months after publication it is available to the public under an Attribution–Noncommercial–Share Alike 3.0 Unported Creative Commons License (http://creativecommons.org/licenses/by-nc-sa/3.0).

[&]quot;ASCB®," "The American Society for Cell Biology®," and "Molecular Biology of the Cell®" are registered trademarks of The American Society for Cell Biology.