

Cell polarity

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The Minisymposium on Cell Polarity featured a broad array of systems. The session began with talks on asymmetric cortical polarity cues. **Lesilee Rose** (University of California, Davis) discussed asymmetric division in *Caenorhabditis elegans*. In the one-cell embryo, PAR polarity proteins regulate cytoplasmic polarity and cortical forces that position the spindle. The latter require the DEP domain protein LET-99, which is present in a lateral-posterior band at the cortex. Recent data suggest that PAR-1 regulates LET-99 asymmetry by creating binding sites for the PAR-5 protein, which inhibits cortical LET-99 accumulation. The second talk focused on cortical polarity in the fission yeast *Schizosaccharomyces pombe*. The conserved Cdc42 GTPase is critical for polarized cell growth, and its active form is localized at cell tips. **Maitreyi Das** (Verde laboratory, University of Miami) presented evidence that the levels of active Cdc42 oscillate at the two cell tips during the switch from monopolar to bipolar growth. Oscillations result from a combination of positive and delayed negative feedbacks, providing a flexible mechanism for modulating the state of polarization.

The next two speakers presented novel models for the polarization of microtubule (MT) arrays in epithelial cells. **Jessica Feldman**

(Priess laboratory, Fred Hutchinson Cancer Research Center) showed that there is a “handoff” of MT-organizing capacity from the centrosome to the apical membrane in *C. elegans* intestinal cells. This occurs through an intriguing process in which the centrosome is localized laterally, and fragments containing γ -tubulin and other MT nucleation factors move first onto lateral membranes and then apically. Localization of these proteins to the apical surface is dependent on the PAR-3 polarity protein, which colocalizes with the MT nucleation proteins as they move. The next talk revealed an important role for septins in MT organization during the polarization of Madin–Darby canine kidney cells. Septins are known for their role in scaffolding at the plasma membrane and are thought to provide a diffusion barrier to lateral movement. **Elias Spiliotis** (Drexel University) presented evidence that septins redistribute from the cell periphery into perinuclear arrays that extend along the emerging apical–basal axis upon cell–cell contact. MTs colocalize extensively with septin filaments, and septins are needed for normal MT organization and dynamics. MT plus ends track along the septin filaments, and preliminary data suggest that specific septin subunits interact directly with microtubules and kinesins.

The session was rounded out by talks on the polarization of the actomyosin cytoskeleton and its relevance to cell motility. **Tom Shemesh** (Bershadsky laboratory, Weizmann Institute) spoke on the formation of parallel bundles of actomyosin fibers prior to cell elongation. In fibroblasts constrained to symmetric circular shapes, the actomyosin fibers arrange in linear or rotational orientations. Theoretical models indicate that friction forces between the fibers may account for the spontaneous symmetry-breaking of the system and the formation of either linear fiber orientation or counterclockwise “swirl” motion. Similar forces may couple to shape changes in elongating cells and expedite cell polarization. Finally, **Matthew Raab** (Discher laboratory, University of Pennsylvania) discussed the directed migration of cells from a soft to a stiff matrix. Nonmuscle myosin IIB (MIIB) is required for this migration and becomes polarized toward the cell rear. Interestingly, the heavy chain of MIIA is phosphorylated in cells on soft matrix, but becomes dephosphorylated on stiff matrix. Further, a phosphomimetic mutant of MIIA disrupts MIIB polarization, suggesting that MIIA phosphorylation is part of the mechanism that transmits substrate elasticity cues to cell polarity.

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Molecular Biology of the Cell Volume 23 Page 970

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