

Cells on the move in Philadelphia

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The Minisymposium “Cell Migration and Motility” was attended by approximately 500 visitors and covered a broad range of questions in the field using diverse model systems. Topics comprised actin dynamics, cell polarity, force transduction, signal transduction, barrier transmigration, and chemotactic guidance.

The session started with the 2010 American Society for Cell Biology Early Career Life Science awardee, **Anna Kashina** (University of Pennsylvania), who reported on the role of arginylation in the dynamics of lamellipodial actin. In contrast to γ -actin, β -actin preferentially localizes to the leading edge of migrating cells and is arginylated. Surprisingly, the reason for this differential arginylation is not inferred by differences at the amino acid level but by different mRNA codon usage that results in faster β -actin translation and protein folding kinetics, which in turn affect arginylation, protein stability, and turnover rates.

The talk was followed by a presentation on the chemotropic response of budding yeast—the basis of mate recognition. **Jayme Johnson** (Duke University) showed data supporting a new model in which the “polarity patch,” a polarity complex that is condensed by

positive feedback and directs polarized growth, searches the inner membrane for active receptors. Local, receptor-mediated signaling slows patch motility and thereby biases the growth response toward the pheromone gradient.

Cochair **Carole Parent** (National Cancer Institute, National Institutes of Health) then reported on a new chemotactic signalling pathway in neutrophil granulocytes that shows partial homology with attractant sensing in *Dictyostelium*. The pathway involves a mammalian target of rapamycin complex 2, protein kinase C, and cyclic adenosine monophosphate-dependent signalling cascade that regulates myosin II-dependent contractility at the trailing edge of the cell and thereby balances leading-edge protrusion with trailing-edge retraction during directed motility.

Daria Siekhaus (Skirball Institute, New York University) followed with a presentation on *Drosophila* immune cell distribution during early embryogenesis. The path of these cells involves the penetration of an epithelial layer, and a genetic screen revealed that, in analogy to the leukocyte extravasation process in vertebrates, this step involves inside-out signalling of integrins, while migration through the rest of the interstitium is independent of these receptors. Integrin activation is mediated by the guanosine 5'-triphosphate-binding proteins Rap1 and RhoL, and the weakening of epithelial junctions is sufficient to rescue the immune cell autonomous penetration defects.

In the next presentation cochair **Michael Sixt** (Institute of Science and Technology, Austria) presented data supporting the idea that the presentation of guidance cues determines the migratory mode of leukocytes. When dendritic cells are exposed to soluble chemokines, they migrate in a nonadhesive manner, and consequently the extracellular substrate does not play instructive roles for cell guidance. When the chemokine is immobilized to surfaces, integrin activation is triggered, the cells become adhesive, and they migrate along the chemokine-decorated matrix structure.

The session concluded with a talk by **Matthew Paszek** (University of California, San Francisco), who proposed a new role of the glycocalyx in cell adhesion. A combination of immunofluorescence and fluorescence interference contrast microscopy demonstrated that at sites of high glycoprotein density the plasma membrane of adherent cells is elevated from the substrate, while the spatially separated adhesion sites are in close proximity to the substrate. Consequently, enhanced glycoprotein expression causes irregular membrane topography and imposes a vertical pull on integrin receptors, causing enhanced force coupling and adhesion site maturation.

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