

Cell-cell and cell-matrix interactions

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Talks at the “Cell–Cell/Cell–Matrix Interactions and Intracellular Signaling” Minisymposium drew on a wide range of experimental systems and approaches to explore how intracellular interactions produce complex cell shapes, and how cells interact with their neighbors and the extracellular matrix (ECM).

Intracellular interactions shape cells

Intestinal epithelial cells build a brush border of microvilli at their apical surfaces that mediates nutrient absorption and host defense. **Scott Crawley** (Tyska Laboratory, Vanderbilt University Medical Center) showed that brush border assembly is driven by *trans* interactions between protocadherin-24 and mucin-like protocadherin at the tips of adjacent microvilli. Loss of this adhesive complex causes irregularities in the length and spacing of these protrusions and contributes to the intestinal pathology seen in a mouse model of Usher syndrome.

Cells interact with their neighbors

Two talks used *Drosophila* cells to investigate the seemingly opposite behaviors of cell–cell fusion and cell–cell repulsion. Cell–cell fusions are common during development and include the union of egg and sperm and the formation of multinucleate muscle fibers. **Elizabeth Chen** (Johns Hopkins University School of Medicine) showed that an “attacking” cell penetrates its target cell using podosome-like projections. Interestingly, podosomes increase the myosin-dependent cortical tension within the target cell at the point of contact. Reducing this tension disrupts cell–cell fusion, even though the podosome invades farther. In contrast, **Andrei Luchici** (Stramer Laboratory, King’s College, London) investigated the

process of contact inhibition of locomotion. When migrating hemocytes collide during their developmental dispersal, they rapidly change direction and migrate away from one another. Pseudospindle microscopy showed that colliding cells simultaneously reorganize the actin flow in their lamellae, suggesting the cells are transiently linked through a clutch-like mechanism that then leads to the synchronous repulsive response.

Two other talks focused on cell–cell adhesion and the maintenance of tissue integrity. Vascular endothelia are continually penetrated by transmigrating leukocytes that produce micron-scale holes both through and between the cells. **Chris Carman** (Harvard Medical School) showed that these “microwounds” are rapidly healed by lamellipodia that extend from the ventral/basal cellular surface near the wound site(s). A mechanical microwounding assay revealed that the lamellipodia form in response to a loss of isometric tissue tension, which in turn triggers Rac1- and NADPH-mediated H₂O₂ production. **Jiongyi Tan** (Nelson and Dunn laboratories, Stanford University) then took a biophysical approach to understanding the most fundamental unit of cell–cell adhesion, the adherens junction. An optical trap-based single-molecule assay showed that the ternary complex of E-cadherin, β -catenin, and α -catenin binds directly to actin filaments under load by forming a catch bond. These data may explain how the adherens junction is a mechanosensitive structure.

Cells interact with the matrix

The final two talks focused on ECM interactions at the molecular and tissue levels. Vinculin is a key component of the focal adhesions (FA) that link the ECM to the actin cytoskeleton. However, because vinculin binds many other FA proteins, it has been difficult to identify the mechanisms that govern its activation and functional specificity. Focusing superresolution light microscopy on point mutations that disrupt vinculin’s interaction with individual proteins, **Lindsay Case** (Waterman Laboratory, National Heart, Lung, and Blood Institute, National Institutes of Health) showed that inactive vinculin is recruited to a plasma membrane–proximal FA region by paxillin. Once activated, the protein shifts to more distal FA regions, where it interacts with talin and actin to promote FA stabilization.

The *Drosophila* egg chamber is an organ-like structure that elongates to produce an elliptical egg. This transformation depends on a dramatic migration of the egg chamber’s epithelial layer that causes the organ to rotate within its surrounding basement membrane ECM. **Maureen Cetera** (Horne-Badovinac Laboratory, University of Chicago) showed that rotation promotes the tissue-level alignment of contractile actin bundles that drive elongation morphogenesis at later stages. Rotation induces this effect first through the motility itself and then by polarizing the basement membrane, which reinforces and stabilizes the actin pattern.

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