# Cell–cell and cell–matrix interactions

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Cell–cell and cell–extracellular matrix (ECM) interactions are fundamental to the complexity of multicellularity achieved in the metazoa. These interactions share many conceptual similarities, the most prominent being a dependence on transmembrane adhesion receptors, binding of adhesion receptors to specific extracellular ligand partners, and the linkage of receptor cytoplasmic domains to intracellular cytoskeletal systems via protein complexes. In both cases, these protein complexes also integrate intracellular signaling, cytoskeletal organization, and regulation of multiple cellular functions.

The 2011 Minisymposium on Cell–Cell and Cell–Matrix Interactions brought together current topics and model systems within this broad area of cell biology.

### Molecular mechanisms that coordinate assembly and function of cell–cell and cell–ECM adhesions

Two presentations addressed how integrin- and cadherin-dependent adhesions intersect. **Kris DeMali** (University of Iowa) showed that the function of vinculin, a shared cytoplasmic component of both adhesions, is differentially regulated in cell–cell and cell–ECM adhesions. Data were presented showing that ablation of a single tyrosine residue in vinculin resulted in opposite effects on cell–cell and cell–ECM adhesions. **Fangliang Zhang** from Anna Kashina's laboratory (University of Pennsylvania) presented his research on talin, a protein that historically has been thought to be present only in integrin-based adhesions as a proximal mediator of integrin activation and of focal adhesion assembly in integrin outside–in signaling. Data were presented on a novel proteolytic processing of talin that is associated with its recruitment and function in cadherin-mediated adhesions.

### Roles of cell protrusions as initial and dynamic mediators of cell–cell and cell–ECM interactions

Filopodia have well-established roles in sampling the local environment and making initial cell–ECM contacts at the leading edge of many cell types. Filopodial-like structures, termed "epithelial zippers," can mediate initial contacts between epithelial cells that culminate in assembly of cell–cell junctions. However, the molecular composition of these structures and their relationship to filopodia has remained unclear. **Katy Liu** from Richard Cheney's laboratory (University of North Carolina at Chapel Hill) showed localization of myosin-X in initial, filopodial contacts between epithelial cells. Whereas myosin-X has a known role in filopodial assembly, the data presented revealed additional important functions in assembly of cell–cell junctions and epithelial morphogenesis. **Brad Davidson** (University of Arizona) presented data from *Ciona intestinalis* embryos, a tractable system for the analysis of cell specification in an intact organism (Cooley *et al.*, 2011). Specification of precardiac cells is directed by polarized, invasive filopodial protrusions that localize their response to an ungraded signal.

### Cell–ECM mechanisms that modulate cell motility

Syndecan-4 is a transmembrane proteoglycan that functions in cell–ECM interactions through its glycosaminoglycan side chains and by activating intracellular signaling that reinforces integrin-dependent focal adhesion assembly (Couchman, 2010). **John Couchman** (University of Copenhagen, Denmark) presented data on a new role for syndecan-4 in the protrusive invadopodia of breast carcinoma cells that regulate invasive behavior. **Jo Adams** (University of Bristol, United Kingdom) described recent phylogenetic studies that show components of the ECM (unlike integrins and cadherins) to be specific to the metazoa (Ozbek *et al.*, 2010). Among the most conserved ECM components are the thrombospondins, which participate in ECM homeostasis and remodeling processes. ECM accumulation of thrombospondin-1 facilitates migration of multiple cell types. Data were presented on L-lectin domain-dependent mechanisms that mediate ECM accumulation of thrombospondins.

## REFERENCES

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