microtubule ribbon completely failed to form. Our results show that spermidine is essential for nuclear condensation and spermatid morphogenesis. Supported by NSF grant MCB-0720486 to SMW.

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Program/Abstract # 68
Noncanonical Frizzled dependent signaling controls chondrocyte polarity during cartilage morphogenesis
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A central question in developmental biology is how morphologically distinct structures are generated from similar cell types. The skeleton provides a good model system to answer this question due to the highly diverse and complex morphological differences displayed by individual elements. Analysis of the growth plate cartilage by Dodds suggested that longitudinal growth of long bones might result from the specific arrangement of chondrocytes. In the proliferative zone, chondrocytes become discoid and arrange in columns, like stacks of coins, which are parallel to the long axis of the cartilage. Although well-organized chondrocytes derive from a relatively unstructured pool of progenitor cells, it is not known whether this arrangement of cells is the result of a regulated process or is in response to physical constraints of the cartilage matrix. Here we test Dodds' model that cell columns in the growth plate cartilage form from a single progenitor cell by a process in which cells divide orthogonal to the stack then rearrange. Our work has uncovered a central role of noncanonical Frizzled dependent signaling in the regulation of chondrocyte polarity and morphogenesis in the growth plate cartilage. Funding for these studies was provided by the Searle Leadership Fund of the Chicago Community Trust.

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Program/Abstract # 69
Polarising migrating tissues during organogenesis
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Most complex organ systems arise from the directed migration of cohesive cell groups, such as sheets, chains and clusters. In this morphogenetic context, migration achieves significantly more than the simple displacement of cells from one location to another, rather it allows these cell groups to build three-dimensional shape. Interestingly, recent studies using genetic mosaics have shown that while extracellular cues clearly guide these migration events their receptors need only be expressed by cells at the very leading edge of the tissue. Here, we address the mechanisms that coordinate cell migration and polarity within one such cohesive tissue, the zebrafish lateral line primordium. This is transient migrating structure, comprising of some 100 cells, whose function is to deposit a series of mechanosensory organs throughout the skin of the fish. By combining in vivo imaging with a number of functional approaches – such as genetic mosaics, laser nanosurgery and small molecule inhibitors – we have begun to address the chemical and mechanical cues that regulate coordinated cell polarity and organization across this migrating tissue. This work reveals that many aspects of this tissue morphogenesis, including the direction of migration and the formation and deposition of organs, are based on self-organising principles.

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Program/Abstract # 70
Crag is a novel regulator of epithelial architecture and polarized deposition of basement membrane proteins in *Drosophila*
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The characteristic polarized architecture of epithelia relies on an intricate interplay between the cytoskeleton, cortical polarity complexes, the trafficking machinery, cell–cell and cell–matrix adhesion. Specifically, contact with the basement membrane (BM), a specialized extracellular matrix underlying the basal side of epithelia, is important for cell polarity. However, little is known about how BM proteins themselves achieve a polarized distribution. In a genetic screen in the *Drosophila* follicular epithelium, we identified mutations in *Crag* (Calmodulin binding protein related to a Rab3 GDP/GTP exchange protein), which encodes a conserved protein with domains implicated in membrane trafficking. Follicle cells mutant for Crag lose epithelial integrity and frequently become invasive. We demonstrate that the loss of Crag leads to the anomalous accumulation of BM components on both sides of epithelial cells without directly affecting the distribution of apical or basolateral membrane proteins. This defect is not generally observed in mutants affecting epithelial integrity. We propose that Crag plays a unique role in organizing epithelial architecture by regulating the polarized secretion of BM proteins.

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