Cilia that sense

Forests of cilia wave fluids in desired directions. But recent excitement about primary cilia—which are unique in occurring at just one copy per cell—has focused on their possible signal transduction abilities. Now, on page 811, Iomini et al. have rediscovered primary cilia on endothelial cells and found that the growth of these cilia responds to outside signals.

Buried in papers from the 1980s, the existence of primary cilia in endothelial cells was all but forgotten. The team detected the primary cilia in cultured human umbilical vein endothelial cells (HUVECs) using antibodies to capillary morphogenesis gene-1 product (CMG-1), the human orthologue of Chlamydomonas IFT71. They exposed the cells to laminar shear stress and found that this terminates intraflagellar transport (IFT) and causes disassembly of the primary cilia. Such a response suggests that primary cilia on endothelial cells have not resolved whether the structures are usually found on the luminal side of the endothelial cell or toward the basal lamina connecting to other tissues. The answer to this question will suggest whether primary cilia might be acting as a mechanosensor of fluid flow, chemosensor, or both.

Bigger equals faster

Size matters, but how biological processes are scaled to match the size of their target—for organisms, organs, cells, and organelles—is largely a mystery. Now, Rzadzinska et al. (page 887) find that the turnover rates of neighboring actin structures depend on their respective sizes, thus renewing the entire superstructure synchronously.

The structures are stereocilia—the mechanosensitive actin pillars sticking out of sensory hair cells of the inner ear. Adjacent parallel rows of stereocilia of graded height form a staircase-like pattern. Long regarded as the epitome of a stable cytoskeleton ensemble, the current authors recently showed that stereocilia bundles are in fact continuously turning over. This may be necessary to maintain the stereocilia’s function over a lifetime.

They now find that the actin paracrystal that makes up the core of each stereocilium rebuilds itself via polymerization at the tip, treadmilling downwards, and dismantling at the base, whereas the ensemble maintains an overall steady-state structure. Turnover rate correlates with stereocilia length. How that turnover rate is controlled remains a mystery. The simplest model involves the accumulation of a regulatory component along the length of each stereocilium, with longer cilia providing more surface area to acquire more of the component. The component may then traffic to one end of the stereocilium and contribute to either faster polymerization or faster depolymerization.

So far the only component with such a graded distribution is a myosin that is present near sites of polymerization, with more present on longer stereocilia. The authors are now looking at the distribution of other regulators of actin dynamics. In addition, they suggest that size-regulated processes may be buried in more complex actin networks such as those found in filopodia.