IN BRIEF

CYTOSKELETON

Making multiple cilia

How centriole assembly and outgrowth of motile cilia are driven during multiciliate cell (MCC) differentiation is unclear, although Notch signalling is known to inhibit MCC formation in epithelia. Stubbs *et al.* observed that a gene encoding a small coiled-coiled protein, which they termed multicilin (MCI), is repressed by Notch in *Xenopus laevis* skin, so they assessed its ability to promote MCC differentiation. Depletion of MCI prevented MCC formation, whereas its overexpression induced ectopic MCC formation. But, how might MCI promote MCC differentiation? Experiments showed that MCI promotes centriole assembly by inducing cell cycle exit (centriole assembly in MCCs occurs postmitotically) and extension of motile cilia through the transcription factor Forkhead box J1 (FOXJ1). Furthermore, microarray analysis revealed that MCI upregulates genes that are expressed in MCCs, including genes encoding centriole components. Thus, MCI, which the authors validate as a transcriptional activator, is a novel regulator of MCC formation.

ORIGINAL RESEARCH PAPER Stubbs, J. L. *et al.* Multicilin promotes centriole assembly and ciliogenesis during multiciliate cell differentiation. *Nature Cell Biol.* 8 Jan 2012 (doi:10.1038/ncb2406)

CELL SIGNALLING

A Hippo signal for anoikis

The Hippo tumour suppressor pathway regulates organ size, but less is known about how the pathway is activated. Zhao *et al.* show that, in cell cultures, detachment from the extracellular matrix induces cytoplasmic localization and phosphorylation, and thus inactivation, of the transcription co-activator YAP (Yes-associated protein), which is a known oncoprotein and regulator of organ size. YAP inactivation by cell detachment depended on the actin and microtubule cytoskeletons and was mediated by the Hippo pathway. Indeed, on cell detachment, the Hippo pathway kinases Large tumour suppressor homologue 1 (LATS1) and LATS2 were shown to phosphorylate YAP. Furthermore, Zhao *et al.* found that YAP inactivation after cell detachment induced anoikis, a type of apoptosis that is repressed in cancer cells to promote cell survival and metastasis. Thus, YAP inhibition by the Hippo pathway may inhibit metastasis and may be a potential therapeutic target.

ORIGINAL RESEARCH PAPER Zhao, B. *et al.* Cell detachment activates the Hippo pathway via cytoskeleton reorganization to induce anoikis. *Genes Dev.* **26**, 54–68 (2012)

MEMBRANE TRAFFICKING

From mitochondria to lysosomes

McBride and colleagues previously observed budding of two vesicle populations from mitochondria, one of which was targeted to peroxisomes. But the fate of the second vesicle pool was unclear. Here, they define the target destination for these vesicles as lysosomes, and thus elucidate a new vesicular transport route that might influence mitochondrial quality control, perhaps by eliminating oxidized complexes that accumulate during mitochondrial respiration. They find that vesicle budding and lysosomal targeting occur both at steady state and early during oxidative stress, and that the extent of budding correlates with the level of respiratory activity. There are several indications that this mitochondrion–lysosome transport pathway is independent of the mitochondrial fission and mitophagy pathways. The questions now are how cargo is selected for removal by these vesicles, and what kick-starts vesicle formation.

ORIGINAL RESEARCH PAPER Soubannier, V. *et al.* A vesicular transport pathway shuttles cargo from mitochondria to lysosomes. *Curr. Biol.* 5 Jan 2012 (doi:10.1016/j.cub.2011.11.057).