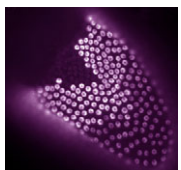


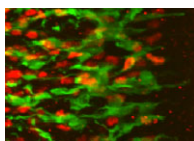
miR172: double duty in potato development

In plants, many developmental events, such as flowering and tuberisation (the formation of thickened underground stems), are controlled by day length, a means of determining when they occur in the year. The molecular mechanisms involved are somewhat understood for flowering, but remain largely unknown for tuberisation. Now, on p. 2873, Paula Suárez-López and co-workers demonstrate that in potatoes, the microRNA *miR172* regulates both flowering and tuberisation. The authors show that *miR172* levels increase when potato plants are grown under tuber-inducing short-day conditions. At the onset of tuberisation, *miR172* levels increase specifically in stolons, the stems from which tubers arise. *miR172* overexpression promotes not only flowering, as seen in other plants, but also tuberisation, and it increases the mRNA levels of the tuberisation promoter *BEL5*. The depletion of the photoreceptor PHYB, a repressor of tuberisation, increases stolon levels of both *BEL5* mRNA and *miR172*. Future work should shed light on whether the dual function of *miR172* in flowering and tuberisation is conserved in other plant species.



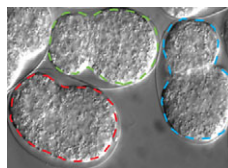
Egfr signalling out of the (feedback) loop

A subset of cells in the *Drosophila* follicular epithelium, a popular model for studying cell fate determination and morphogenesis, forms respiratory structures called dorsal appendages. Patterning within this tissue depends on Egfr signalling: dorsal appendage cell fate requires Gurken, an Egfr ligand, and Gurken-Egfr signalling is widely believed to be regulated in an autocrine feedback loop by another Egfr ligand, Spitz, and the Egfr inhibitor Argos. On p. 2893, however, Laura Nilson and colleagues challenge this view by showing that the Spitz-Argos feedback loop is not required for dorsal appendage patterning and that, instead, the necessary spatial information might be provided directly by Gurken. Mirror and Pointed, two transcription factors that function downstream of Egfr signalling, and Sprouty, an Egfr signalling pathway inhibitor, they report, are crucial for this process. Together, these findings indicate that graded Gurken-Egfr signalling patterns the follicular epithelium directly rather than through an autocrine feedback loop, a notion also supported by Stanislav Shvartsman and co-workers (see p. 2903).



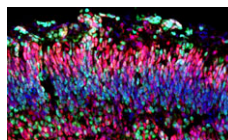
LIMiting concentrations for motor neurons...

During nervous system development, a multitude of different neuronal cell types are generated in a process partly regulated through the combinatorial expression of LIM homeodomain transcription factors. Now, on p. 2923, Song, Pfaff and co-workers investigate the effects of changing the concentrations of the LIM homeodomain proteins *Islet1* and *Islet2*, and of the putative *Islet* protein antagonist *Lmo4*, on the differentiation of motor neurons (MNs) and of V2a interneurons in mice. By combining different mutations that affect *Islet* expression, the authors demonstrate that reducing *Islet* protein levels leads to increased V2a interneuron differentiation at the expense of MN formation. Unexpectedly, depleting *Lmo4* levels alone has no effect on MN or V2a interneuron specification; however, the loss of MNs caused by *Islet* protein depletion is rescued in mice that lack *Lmo4*. These findings indicate that both *Islet* protein and *Lmo4* levels contribute to the make-up of LIM complexes in vivo, and the authors speculate that *Lmo4* might facilitate fate decisions when LIM protein concentrations are low.



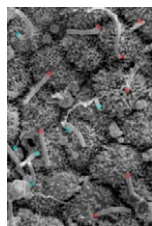
...while *nhr-67* specifies neurons at every step

The animal nervous system contains many different types of neurons, but what mechanisms underpin the generation of such diversity? Now, Oliver Hobert and colleagues reveal that in *C. elegans*, the Tailless/TLX transcription factor (TF) *NHR-67* controls both the neuronal cell fate and left-right (L/R) identity of the ASE class of gustatory neurons (see p. 2933). ASE neurons differentiate in stages: first, overall ASE identity is adopted; then, distinct left-hand (ASEL) and right-hand (ASER) cell types are specified. The authors isolate *nhr-67* from a screen for mutants with perturbed L/R asymmetry and discover, surprisingly, that it also regulates overall ASE identity, in conjunction with the fate determinant *che-1*. Subsequently, *nhr-67* promotes ASER specification by activating the homeobox gene *cog-1* in both prospective ASER and ASEL neurons; in ASEL neurons, however, *cog-1* activity is inhibited by the microRNA *lxy-6*. Such reiterative deployment of TFs, the authors suggest, might constitute a general feature of TF activity in the specification of neuronal subtypes.



MARCKS leaves its mark on radial glia

Radial glial cells (RGCs), which span the entire width of the developing cerebral cortex, act as neural progenitors and provide a polarised scaffold for the migration of newborn neurons. But what are the molecular factors that regulate their polarisation? Eva Anton and co-workers now reveal that in mice, the calmodulin-binding protein MARCKS, a PKC substrate, is involved in this process (see p. 2965). In *Marcks*^{-/-} mice, they report, the RGC scaffold is disrupted. *Marcks*^{-/-} RGCs proliferate aberrantly and are frequently abnormally localised. The polarised distribution of cell-polarity markers, such as PAR complex proteins, in RGCs is also disrupted, as is RGC-guided neuronal migration, leading to defects in cortical lamination. Surprisingly, the phosphorylation of MARCKS by PKC is not required for its function in RGCs, but its myristoylation-mediated targeting to the plasma membrane is. The authors conclude that myristoylated MARCKS is crucial for RGC development and suggest that MARCKS might serve as an anchor for the membrane localisation of crucial signalling complexes.



Thinking BicC about cilia orientation

Cilia defects are often linked to disturbed left-right asymmetry and to kidney cysts, a symptom also seen in mice and frogs that lack the conserved RNA-binding protein Bicaudal C (BicC). Now, on p. 3019, Daniel Constam and colleagues identify a role for BicC in cilia biology. The authors find that disrupting BicC function in either mouse or *Xenopus* embryos randomises left-right asymmetry by disturbing the regular planar orientation of motile cilia and, hence, cilia-driven fluid flow. Further, the researchers find that in kidney cell lines, the BicC sterile alpha motif (SAM) domain guides the localisation of BicC to cytoplasmic structures that contain the canonical Wnt signalling component Dishevelled 2 (Dvl2), as well as RNA-processing bodies known as P-bodies. BicC interferes with Dvl2-mediated canonical Wnt signalling; such interference has been suggested to promote planar cell polarity (PCP) signalling. Thus, the authors propose, BicC might link cilia orientation with PCP signalling by modulating P-body-mediated RNA silencing or by regulating Dvl2 function.