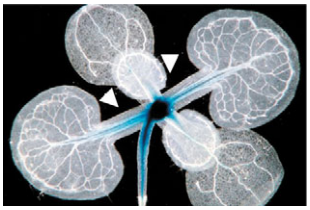
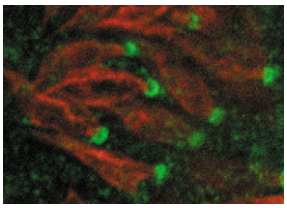


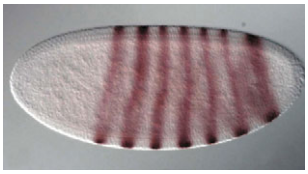
Leaf development takes no KNOX

A key question in plant development concerns what controls leaf development and leaf shape. On p. 3955, Hay et al. report that interactions between the hormone auxin and the AS1 and KNOX transcription factors control leaf development in *Arabidopsis*. In higher plants, the specification of leaf initials at the tip of the shoot apical meristem (a slowly dividing stem cell population) is facilitated by mutual repression between AS1 (which promotes leaf fate) and KNOX (which promotes meristem activity). Now, Hay and colleagues show that auxin activity (which is transported towards leaf initials, where it accumulates via the efflux facilitator PIN1) acts with AS1 to repress expression of the KNOX gene *BREVIPEDICELLUS* and thus promote leaf fate. They also show that PIN1-regulated auxin gradients control leaf shape in a KNOX-independent manner, but that ectopic KNOX expression in leaves perturbs these gradients and so alters leaf shape. Thus, the researchers suggest, regulation of auxin gradients by KNOX proteins may underlie natural variations in leaf form.



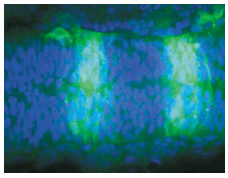
Getting a grip on special microtubules

Several developmental processes – for example, *bicoid* mRNA localisation during oogenesis and meiosis during germline development – involve special microtubule-organising centres (MTOCs). Now, on p. 3963, Christiane Nüsslein-Volhard and colleagues propose that the cap structure of the γ-tubulin ring complex (γTuRC) is essential for the function of these special MTOCs. The γTuRC, which is required for microtubule nucleation, consists of a lockwasher-like structure and a globular cap. To study the function of the cap, the researchers analysed flies that contain mutations in the cap components *Grip75* or *Grip128*. Animals with these mutations are viable but sterile, and their cells seem to contain γTuRCs that nucleate the microtubules required for essential functions in somatic cells. By contrast, the distinct microtubules that are needed for *bicoid* mRNA localisation and germline meiosis do not form properly in the mutant flies. The researchers suggest, therefore, that Grip75 and Grip128 are needed to anchor the γTuRC at special MTOCs but are not essential for microtubule nucleation.



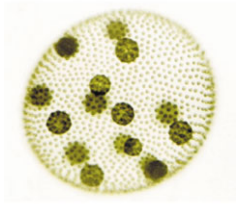
Caudal: an ancestral master organiser

The establishment of the anteroposterior axis is an important early event in embryogenesis. Many of the molecular components of this process are conserved through evolution. However, Bicoid – the master organiser of anterior development in *Drosophila* – is not present in non-dipteran insects. So, to study the evolution of body plan patterning, Olesnicky and co-workers have turned to the wasp *Nasonia* (see p. 3973). Wasps lack Bicoid but their embryos are patterned completely within a syncytial environment like fly embryos. The researchers report that a gradient of localised *caudal* mRNA directs posterior patterning in *Nasonia* embryos in contrast to *Drosophila* embryos, in which the translational repression of *caudal* mRNA by Bicoid establishes a gradient of Caudal protein. The researchers also show that *Nasonia caudal* activates the expression of gap genes, which then activate pair-rule gene expression; in *Drosophila*, *caudal* mostly regulates pair-rule gene expression. These results suggest that *caudal* is an ancestral master organiser of patterning but that its role has been reduced in dipterans.



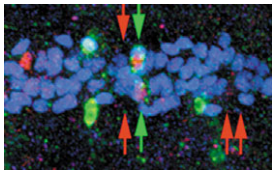
Semaphorin' neural crest signals

Neural crest cells (NCCs) are migratory cells that differentiate into several cell types, including neurons and bone cells. NCCs proliferate before and throughout their migration and differentiation. Wnt/TCF signalling helps to control this proliferation but are any other factors involved? Berndt and Halloran now report that the guidance molecule semaphorin 3d (Sema3d) acts downstream of Wnt/TCF signalling to promote NCC proliferation and development in the zebrafish hindbrain (see p. 3983). The researchers show that morpholino-mediated knockdown of Sema3d inhibits the proliferation of hindbrain neuroepithelial cells at the time of their epithelial-mesenchymal transition into migratory NCCs. It also reduces the number of migratory NCCs and disrupts the development of NCC derivatives. Other results indicate that Sema3d lies downstream of Wnt/TCF signalling; for example, Sema3d overexpression rescues the reduced NCC proliferation caused by expression of a dominant-repressor form of TCF. The researchers conclude from their experiments that Sema3d function is important for regulating the cell cycle of NCCs and for their subsequent development.



Translational control gets a shunt

The green alga *Volvox carteri*, with its two distinct cell types – somatic and reproductive cells – is an ideal organism in which to study the molecular basis of cell differentiation. Somatic cell terminal differentiation in *Volvox* is controlled by the transcriptional repressor RegA, which stops these cells from becoming reproductive cells. Now, on p. 4045, Babinger and colleagues report that, surprisingly, the translation of *regA* is controlled by ribosome shunting, a process in which the translation initiation complex dissociates from the mRNA at a stable secondary structure and then rebinds at a downstream 'landing site'. Via a systematic mutational analysis of the *regA* 5′ UTR, which contains eight start codons, the researchers discounted leaky scanning, reinitiation and internal ribosome entry site-mediated initiation as the mechanisms that control *regA* translation. Instead, their results indicate that *regA* translation is controlled by ribosome shunting, an unusual mechanism in eukaryotes for regulating translation that, the researchers suggest, might be used in other developmental situations.



Tinman needed for a good heart

In *Drosophila*, the NK homeobox gene *tinman* (*tin*) is essential for the specification of cardiac progenitors in the early dorsal mesoderm. Like its vertebrate counterpart *Nkx2.5*, *tin* is also expressed during cardiac maturation and differentiation. However, its later role in cardiac development is unclear because *tin*-null embryos have no dorsal vessel (the *Drosophila* equivalent of a heart), and die. Zaffran et al. now reveal that *tin* controls the diversification and differentiation of myocardial cells during the later stages of cardiogenesis, through regulatory interactions with *Dorsocross* and other cardiogenic factors (see p. 4073). The researchers made their discovery by making transgenic fly lines that expressed *tin* normally during early heart development, but that did not express *tin* in dorsal vessel cardioblasts at later stages. The dorsal vessel formed in the resulting embryos and was present in surviving adult flies, but myocardial diversification, differentiation and remodelling was defective. These findings provide new information about the molecular pathways that act at later stages of fly, and perhaps also mammalian, heart development.

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