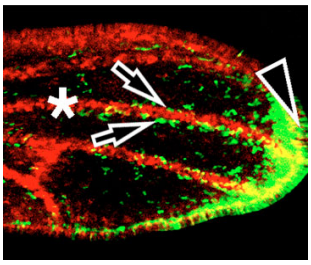
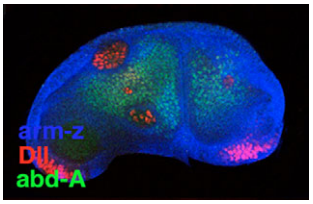
Vg1: a mystery solved

Twenty years ago, researchers discovered that the mRNA for Vg1, a transforming growth factor β family member, localizes to the vegetal cytoplasm of *Xenopus* oocytes, making Vg1 a candidate for the mesoderm-inducing signal released by vegetal cells. However, its role in vivo has remained mysterious until now. On p. 15, Birsoy and colleagues now show that maternal Vg1 is an essentially required signalling molecule during *Xenopus* development. They report that gastrulation is delayed, and that anterior and dorsal development is reduced, in embryos depleted of Vg1 using an antisense oligonucleotide. The mystery of the role of Vg1 in vivo arose, they explain, because the original Vg1 clone encodes proline at position 20. This means it is inefficiently processed to active Vg1 in vivo and so fails to rescue Vg1-depleted embryos. By showing that a Vg1 allele with serine at this position rescues Vg1-depleted embryos, the researchers demonstrate conclusively that Vg1 is an essential maternal regulator of embryonic patterning.



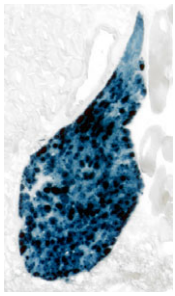
Location matters for MAP kinase

Some proteins have very different functions in different subcellular locations. Mitogen-activated protein kinase (MAPK), for example, is usually transported into the nucleus immediately after it is phosphorylated and, once there, it promotes cell proliferation. But, report Marenda et al., in the differentiating vein cells of developing *Drosophila* wings, phosphorylated MAPK (pMAPK) is held in the cytoplasm, where it controls cell fate (see p. 43). At the same time, note the researchers, pMAPK moves into the nuclei of other wing cells and promotes cell proliferation. Thus, MAPK phosphorylation can signal two different cellular outcomes in developing fly wings – differentiation or proliferation – based on the subcellular localization of pMAPK. Bifurcation of the Ras signalling pathway through this holding of pMAPK in the cytoplasm, suggest the authors, may be generally required for the cell cycle arrest that precedes differentiation.



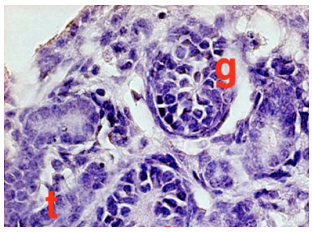
Breaking the Hox posterior prevalence rule

Hox genes pattern the anteroposterior axis of animals, determining where limbs and other structures form. Usually, Hox genes expressed in posterior regions of developing organisms suppress or downregulate the expression of more anterior genes. But, on p. 117, Foronda and co-workers report an exception to this posterior prevalence rule: in the *Drosophila* female genital disc, posteriorly expressed Abdominal-B (Abd-B) maintains Abdominal-A (Abd-A), rather than repressing it as in the embryonic epidermis. *Drosophila* genitalia derive from the genital disc, which forms from cells of the abdominal segments A8-10. The researchers report that the Abd-B isoforms M and R are expressed in A8 and A9, respectively, and that Caudal, which directs analia formation, is expressed in A10. Additional findings provide a detailed analysis of the unexpected ways in which Hox genes interact with each other and with Distal-less, which specifies appendages, to form genitalia.



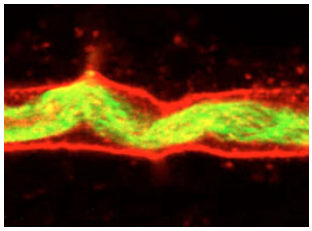
Developing a nervous sweat

Like other parts of the nervous system, the assembly of sympathetic neuronal circuits (which control autonomic bodily functions, including sweating) involves a series of differentiation steps. The final step in sweat gland innervation is a noradrenergic to cholinergic neurotransmitter phenotype switch. Stanke and co-workers now report that target-dependent signalling through the cytokine receptor subunit gp130 mediates this developmental change in the sympathetic neurons that innervate mouse sweat glands (see p. 141). They show that cytokines that act through gp130 are present in sweat glands and that the conditional deletion of gp130 in sympathetic neurons prevents the switch from a noradrenergic to cholinergic neurotransmitter phenotype; the differentiation of cholinergic sympathetic neurons innervating other targets is also mediated by gp130. Surprisingly, gp130-depleted mice have functional sweat glands, indicating that cholinergic neurotransmission is not needed for the acquisition and maintenance of sweat gland secretory responsiveness, as previously believed.



Cultured insights into kidney differentiation

For some stem cells – haematopoietic progenitors, for example – culture systems in which their differentiation can be investigated are well established. Now, on p. 151, Osafune and colleagues describe a colony-forming assay that allows them to isolate renal progenitors from embryonic mouse kidneys for the first time. Their culture system uses a feeder layer of fibroblasts that stably express Wnt4, which is required for the epithelial differentiation of metanephric mesenchyme. Single metanephric mesenchymal cells that strongly express *Sal1* (a zinc-finger nuclear factor essential for kidney development) seeded onto this feeder layer form colonies that contain several types of epithelial cells present in glomeruli and renal tubules. In addition, the researchers report that the planar cell polarity pathway acts downstream of Wnt4 to direct renal progenitor differentiation, a novel insight into the mechanisms underlying kidney development.



Chitin support: scaffolding for new tubes

Insects breathe through a network of epithelial tubes, called trachea, that transport gases around their bodies. In *Drosophila*, the uniform expansion of these tubes during development requires the assembly of a transient intraluminal chitin matrix. Now, on p. 163, Moussian et al. report that chitin filament assembly depends on Knickkopf (Knk) and Retroactive (Rtv), proteins that are also involved in the formation of the fly cuticle. The researchers describe how *knk* and *rtv* mutants develop severe tracheal tube size defects similar to those seen in chitin-deficient embryos, and show that Knk, an apical GPI-linked protein, is mislocalized in tube expansion mutants in which septate junction proteins are disrupted. The researchers propose that septate junctions, which resemble vertebrate tight junctions, ensure the correct distribution of the components needed for chitin filament assembly and so ensure the uniform expansion of the trachea.

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