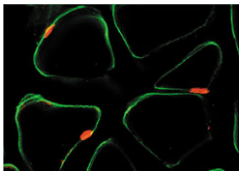
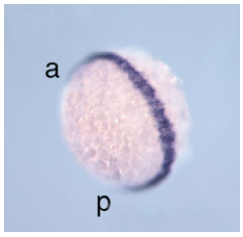


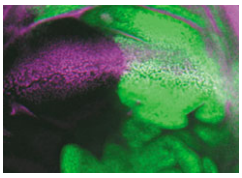
A tail of regeneration

Cut the tail off a *Xenopus* tadpole and it quickly makes a new muscle-packed tail. But are the muscles in this regenerated appendage formed from de-differentiated myofibres (as in newts) or from stem cells? On p. 2303, Jonathan Slack and colleagues provide strong evidence that muscle satellite cells – adult stem cells that repair damaged muscles in mammals – rebuild the muscles in regenerating *Xenopus* tails and reveal the role that the Pax7 transcription factor plays in this process. The researchers demonstrate that regenerating *Xenopus* tails contain many dividing muscle satellite cells, most of which express Pax7. Using Pax7 as a marker for satellite cells, they show that these cells are responsible for forming the muscle masses of the regenerated tail. Finally, they report that when *pax7* function is antagonized during tail regeneration, the tail reforms but it contains fewer satellite cells. Thus, Pax7 is needed to maintain satellite cells as a stem cell population but is not required for their differentiation into myofibres.



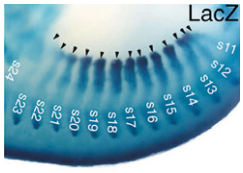
Spinning a developmental story

For all animals, the formation of the embryonic axes is an important developmental milestone. Now, on p. 2347, Akiyama-Oda and Oda report that in spiders Decapentaplegic (Dpp) is essential for the transformation of a radially symmetrical embryo to one with axial symmetry. They also report that Short gastrulation (Sog) is required for ventral patterning. In *Drosophila* embryos, Sog functions antagonistically with Dpp in dorsoventral pattern formation. To investigate the function of these two proteins in house spider embryos, the researchers used RNAi knockdown. Depletion of *dpp* stopped the embryos losing their radial symmetry; depletion of *sog* led to nearly complete loss of ventral structures, including the central nervous system. The researchers note that this mechanism for ventral specification is much more similar to that of vertebrates than to that of evolutionarily distant arthropods, like *Drosophila*, in which Sog makes only minor contributions to the development of ventral structures. Future studies in spider embryos could, therefore, provide new insights into the evolution of early development.



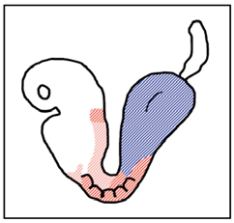
Fat and Dachsous: a signal partnership

The *Drosophila* protocadherins Fat (Ft) and Dachsous (Ds) are required for regulating imaginal disc growth, establishing planar cell polarity (PCP), and for the proximodistal (PD) patterning of appendages. Cadherins usually function as adhesion molecules, but Ft and Ds, which interact with each other, could act as a receptor-ligand pair instead. Matakatsu and Blair now report that this seems to happen in the establishment of PCP but that Ds and Ft regulate growth and PD patterning independently (see p. 2315). The researchers used a structure-function approach to test which domains of Ft and Ds mediate their various activities. They show, for example, that the extracellular domain of Ft is not needed for its functions in growth, PCP establishment and PD patterning but that the extracellular domain of Ds is necessary and sufficient for its effects on PCP. These results suggest a model, in which Ft has a receptor-like function that is mediated by its intracellular domain while Ds has a ligand-like function in PCP.



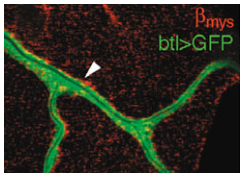
Fgf10 from somite to breast

To date, it has remained unclear what signals subdivide the surface ectoderm of embryos into different developmental fields, such as those that form mammary epithelium. But now, on p. 2325, Veltmaat et al. reveal that the induction and positioning of one of the five mammary placodes in the mouse depends on *Gli3*-mediated somitic *Fgf10* expression gradients, which activate ectodermally expressed FGF receptors and lead to *Wnt10b* expression. Using Wnt signalling and ectodermal multilayering as markers of mammogenesis, the researchers investigated mammary placode development in mouse mutants with altered somitic *Fgf10* gradients. They report that mammary line formation is impaired and placode 3 is absent in embryos in which the somitic *Fgf10* gradient is shortened (as in *Gli3* null embryos) or less *Fgf10* is expressed overall. The researchers suggest, therefore, that a combination of somitic elongation and somitic *Fgf10* gradients induces the differentiation of the surface ectoderm into mammary epithelium at the position in mice that corresponds to the position of the human breast.



PcGs get complex with mammalian Hox genes

Polycomb group (PcG) proteins repress homeotic (Hox) gene expression in specific embryonic regions of mammals but the molecular mechanism of this control is poorly understood. Fujimura and co-workers now report that, in mouse embryos, class 2 PcG components (which bind to DNA regulatory sequences) form complexes of different compositions to control spatial *Hoxb8* expression (shown in blue). The researchers on p. 2371 reveal that PcG protein binding and histone modifications around *Hoxb8* are different in tissues that do and do not express *Hoxb8*. In particular, they show that the binding near *Hoxb8* of a class 2 PcG complex that contains the E3 ubiquitin ligase Rnf2, together with trimethylation of histone H3, which marks inactive chromatin, correlates with transcriptional silencing of *Hoxb8*. Genetic impairment of Suz12 (a class 1 PcG protein that trimethylates histone tails) and of Rnf2 produces Hox gene derepression. Thus, interactions between class 1 and class 2 PcG proteins maintain repression of Hox genes outside their expression domains in mice, as in *Drosophila*.



Talin(t) for tracheal branching

Although the formation of the *Drosophila* tracheal system is partly understood, little is known about how the terminal branches of this network of epithelial tubes are maintained. Levi, Ghabrial and Krasnow now reveal that integrin-talin adhesion complexes maintain these branches and their luminal organization (see p. 2383). Tracheal terminal cells form hollow terminal branches, which adhere tightly to target tissues to supply them with oxygen. In a genetic screen, the researchers isolated *tendrils* mutants, which have fewer than normal terminal tracheal branches that contain multiple, convoluted lumens. This phenotype arises late in development from loss of branches but not their lumens and is caused by mutations in the gene encoding talin, which links integrin cell-adhesion molecules to the cytoskeleton. Terminal cells mutant for *Drosophila* β-integrins also show the *tendrils* phenotype. The researchers conclude that integrin-talin adhesion complexes anchor mature terminal branches to their target tissues and also maintain their luminal organization. Similar complexes, they suggest, may stabilize other tubular networks.

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