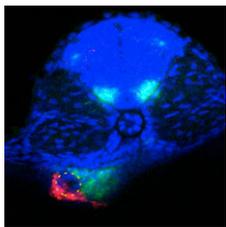


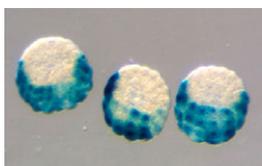
Mesoderm migration lights up

A pivotal event during gastrulation is mesoderm migration. In *Drosophila* embryos, presumptive mesoderm cells invaginate, undergo an epithelial-to-mesenchymal transition (EMT), and then spread over the ectoderm to form a monolayer. On p. 3975, Murray and Saint investigate what types of cell rearrangements occur during this example of mesoderm spreading. They express photoactivatable GFP fused to α -Tubulin in all the cells of fly embryos, photoactivate sections of mesoderm or small numbers of mesodermal cells as gastrulation begins, and then follow the migration of these fluorescent cells over non-fluorescent ectodermal cells. The researchers find that those cells in contact with the ectoderm immediately after the EMT migrate dorsolaterally as a group, but are sometimes overtaken by cells not in contact with the ectoderm. Murray and Saint conclude that mesodermal cells use a combination of strategies to form a monolayer: directional dorsolateral migration (presumably towards chemoattractants expressed in the dorsal ectoderm), strong adhesion between mesodermal and ectodermal cells, and some intercalation during the final stages.



Two-way signals for pancreatic development

The pancreas, a gland that secretes digestive juices into the stomach and insulin into the blood, develops from two endodermal buds. In mice embryos, fibroblast growth factors (FGFs) secreted by the pancreatic mesenchyme control the development of these buds, but how does the pancreatic mesenchyme form? Manfried and colleagues now show that, in zebrafish embryos, reciprocal endoderm-mesoderm interactions mediated by FGFs control pancreas development (see p. 4011). The researchers identify an area next to the ventral pancreatic bud – the pancreatic lateral plate mesoderm (LPM) – that corresponds to the pancreatic mesenchyme in mice and that is essential for ventral bud development. They show for the first time that transient expression of *fgf24* in the endodermal precursor of the ventral bud patterns the pancreatic LPM and report that subsequent expression of *fgf10* and *fgf24* by the pancreatic LPM controls the specification and growth of the ventral pancreas. Thus, they conclude, sequential signalling between the endoderm and mesoderm drives pancreas development in zebrafish.



Gene regulatory networks: maternal code deciphered

Maternal factors initiate development in many animal embryos but the zygotic gene combinations activated by these factors are poorly understood. Now, on p. 4023, Rothbächer and co-workers reveal that the maternal transcription factor Ci-GATAa (orthologue of vertebrate GATA4, 5 and 6) controls the ectodermal regulatory network in *Ciona intestinalis*. The ectoderm, which develops into epidermis and nervous tissue, occupies the animal half of ascidian embryos. The researchers report that Ci-GATAa activity is initially found throughout the embryo before being repressed in the vegetal hemisphere by maternal β -catenin. In the animal hemisphere, Ci-GATAa first activates the pan-ectodermal gene *Ci-fog*. After neural induction, it also activates *Ci-otx* in neural progenitor cells. The researchers show that this activation pattern is achieved by Ci-GATAa and maternal Ets1/2 binding to the *Ci-otx* enhancer and synergistically activating *Ci-otx* in neural territories; in non-neural territories, maternal Ets1/2 prevents *Ci-otx* activation by Ci-GATAa. Overall, these results identify a precise combinatorial code of maternal factors that initiates ectodermal development in *Ciona*.



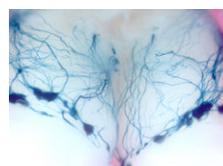
FGF keeps segmentation clock ticking

The segmented pattern of the vertebrate spine is established during embryogenesis by the formation of somites (blocks of mesoderm that form the vertebrae and back muscles) at regular temporal intervals. In the clock and wavefront model for somitogenesis, pulses of Notch, FGF and Wnt signalling in the presomitic mesoderm (PSM) are translated into the periodic array of somites at the so-called wavefront. Olivier Pourquié and colleagues now provide the first genetic evidence that FGF signalling positions the wavefront and controls the segmentation clock in mouse somitogenesis (see p. 4033). They show that conditional deletion of the FGR receptor gene *Fgfr1* in the mesoderm abolishes FGF signalling and disrupts normal cyclic gene expression in the PSM. It also arrests somite formation. In addition, pharmacological inhibition of FGF signalling blocks the oscillations of both Wnt and Notch signalling in the PSM, but with different kinetics. The researchers conclude, therefore, that FGF signalling acts upstream of Wnt and Notch signalling to control the segmentation clock during mouse somitogenesis.



From fragments to whole: BMP signals regeneration

Planarians (flatworms) can regenerate from irregularly shaped fragments. During regeneration, new tissue forms at the wound and existing tissues are remodelled to make a complete, symmetrical animal. How is this amazing feat achieved? On p. 4043, Alejandro Sánchez Alvarado's group report that BMP signalling regulates several aspects of regeneration in the planarian *Schmidtea mediterranea*. The researchers use RNAi knockdown to investigate the role of *smedolloid-1* (a BMP1/Tolloid-like gene), *smedsmad4-1* (a SMAD4-like gene) and *smedbmp4-1* (a BMP2/4/DPP-like gene) in regeneration. These experiments show that BMP signalling is involved in the formation and dorsal-ventral (DV) patterning of new tissues at the reset midline of a regenerating fragment. Additional knockdown experiments indicate that BMP signalling also maintains the DV pattern in undamaged adult tissue. These and other results lead the researchers to propose that BMP signalling regulates the dorsal midline of planarians and that the midline has to be reset by BMP activity before injured animals without left-right symmetry can regenerate.



Wired for smell

The mouse olfactory system, which detects numerous environmental chemical stimuli, is divided into subsystems. Sensory neurons in each subsystem within the olfactory epithelium have different receptor molecules and project their axons to different parts of the olfactory bulb. Now, on p. 4063, Peter Mombaerts and colleagues report that semaphorin 3F (Sema3f) and its co-receptor neuropilin 2 (Nrp2) are involved in the axonal wiring of guanylate cyclase-D (GC-D⁺)-expressing sensory neurons. The axons of these neurons, which are not known to express odorant receptors, project to the necklace glomeruli, which sit between the main and accessory olfactory bulbs. To visualize the axonal projections of GC-D⁺ neurons, the researchers generated mice that co-express GC-D with labelled tau (an axonal marker). They show that in *Sema3f*-null (but not *Sema3b*-null) mice and *Nrp2*-null mice, GC-D⁺ neurons form ectopic glomeruli in the main olfactory bulb, as well as the necklace glomeruli. Thus, the researchers conclude, an Nrp2-Sema3f interaction helps to determine the axonal wiring of this relatively poorly studied olfactory subsystem.

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