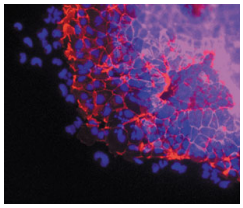
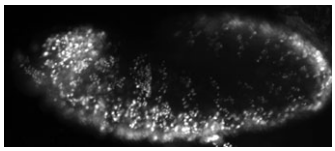


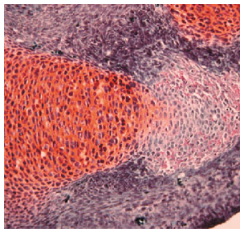
Mouse development: a moving WAVE

The dramatic cell movements that occur during gastrulation are influenced by extracellular signals. Because the highly conserved WAVE complex – which regulates the actin cytoskeleton – couples extracellular signals to cell migration, Rakeman and Anderson investigated the role of the WAVE complex in early vertebrate development (see p. 3075). They identified mice lacking Nap1 – a regulatory component of the complex – and found that without Nap1 the WAVE complex in embryos is unstable. Moreover ~25% of Nap1 mutants had a duplicated anteroposterior axis (other embryonic defects included the slowed migration of endoderm and mesoderm). This axis is determined by the position of the primitive streak, which is itself determined by anterior visceral endoderm (AVE) movement. The authors suggest that Nap1 is required for the normal polarisation and active migration of AVE cells, and conclude that, during mammalian development, the WAVE complex is vital for the regulation of actin during tissue organisation and the establishment of the body's main axes.



Cyclin to the terminal

Several cyclins are responsible for regulating the cell cycle, but their regulatory plasticity makes their distinct roles difficult to define. On p. 3201, Jacobs and colleagues discuss the particularly puzzling case of Cyclin A, which is essential for *Drosophila* embryonic viability but is needed only for certain types of mitoses. In the embryonic ectoderm, Cyclin A is only required for the very last division before cells become post-mitotic. The researchers have found that this is because during normal mitotic cycles, Cyclin A and Cyclin E function redundantly to prevent the premature activity of Fizzy-related/Cdh1 (Fzr), which targets the B-type Cyclins and String/Cdc25 for degradation. By contrast, before terminal mitoses, Cyclin E is inactivated early, leaving Cyclin A to work alone – this means that in Cyclin A mutants untimely Fzr activation prevents completion of the division programme. Observations from other labs, and those made in this paper, indicate that Cyclin A is also crucial for terminal mitoses in neuroblast lineages.



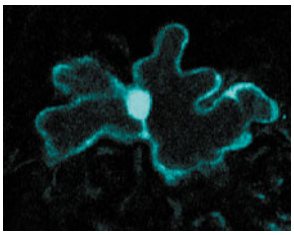
Canonical Wnt to the bone

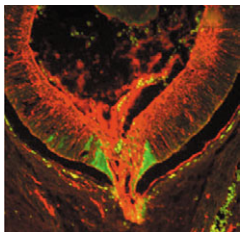
Previous studies have implied that the specification of osteoblasts – the cells that secrete bone matrix – involves Hedgehog (Hh) and canonical Wnt signalling. On p. 3231, Rodda and McMahon test this idea directly by genetic manipulation within osteoblast progenitors and find that these pathways have sequential roles in osteoblast specification. By conditionally removing β-catenin in specific cell types, they show that canonical Wnt signalling is necessary to stop osteoblast precursors from becoming chondrocytes (cells that produce cartilage). Conversely, using a stabilised form of β-catenin, they found that too much canonical Wnt signalling causes the overproliferation of osteoblast precursors and excessive mineralisation. They have also found that osteoblast specification requires only transient Hh signalling, and that Hh is not required during the formation of mature osteoblasts. The authors go on to discuss how Hh and Wnt might sequentially regulate osteoblast differentiation, speculating that the programmes of chondrocytes and osteoblast development are regulated by a balance between Sox9 and β-catenin.

Flowering: the hows and whens

Two papers in this issue discuss flowering – one addresses its timing, and the other the specification of flower organ identity. On p. 3213, Hoecker and colleagues have investigated how the SPA protein family helps plants to adjust their development to the environment. The researchers have previously shown that SPA proteins are required for skotomorphogenesis (the growth that occurs when seedlings are kept in the dark); now they investigate the role of SPA proteins in photoperiodic flowering (the timing of flowering in response to day length). They show that all four SPA proteins interact with CONSTANS (CO), which is essential for the early flowering that occurs in response to long days. CO transcription is regulated by the circadian clock and its protein is stabilised by light, which together allow CO protein to accumulate only when days are long. The presence of high levels of CO protein in *spa* triple mutants lead the authors to speculate that SPA proteins control the stability of CO in response to light.

The mechanisms by which floral organs develop are largely well characterised – A, B, C and E class genes combine to direct the development of the four floral whorls into stamens, carpals, petals and sepals – but some crucial aspects remain unclear. On p. 3159, Liu and colleagues describe a new model for the regulation of the class C gene AGAMOUS (AG), which specifies stamen and carpel development in the inner two whorls. The researchers previously identified two transcriptional co-repressors called SEUSS (SEU) and LEUNIG (LUG) that prevent ectopic expression of AG. The authors now show that these co-repressors interact with the MADs box proteins SEPALLATA3 (SEP3) and APETALA1 (AP1), converting them from activators into repressors of AG expression. So although previous models hold that AG is activated in all four whorls and repressed in the outer two, these researchers suggest that SEU and LUG repress AG in all four whorls, but that this is then antagonised by AG activation in the inner whorls.





Shedding light on a blind spot

The optic disc (often called the 'blind spot') is where optic fibres converge and leave the retina, and, on p. 3179, Bovolenta and co-workers shed light on its formation. It develops from the edges of a groove (called the optic fissure) at the ventral pole of the rudimentary eye, and its position is determined by the juncture of two distinct regions of the optic fissure – the retinal fissure and the optic groove. Having found that retinal fissure precursors express a unique combination of markers (including PAX2), the researchers studied these precursors in mice that lack BMP7. This analysis revealed that BMP7 is required for the specification of retinal fissure precursors and that it regulates their subsequent proliferation and apoptosis. SHH, they report, is also required for retinal fissure precursor maintenance during development. And because the microphthalmic (small eye) phenotype of *BMP7*-null mice resembles a rare congenital disease in humans, the authors also speculate on the role of BMP7 in this type of abnormality.

Jenny Bangham