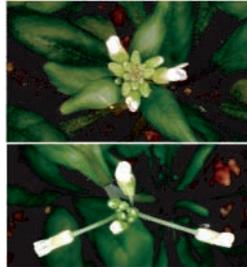


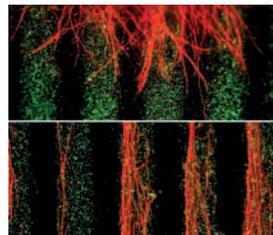
Plant talk

Body and organ size in multicellular organisms is governed by intrinsic mechanisms that coordinate cell size and number during development. On p. 1491, Torii and co-workers describe how three related receptor-like kinases interact synergistically to link cell proliferation to organ growth and flower development in *Arabidopsis*. Plants that lack ERECTA, a leucine-rich receptor-like serine/threonine kinase, have compact inflorescences and short lateral organs, which indicates that ERECTA mediates cell-cell signals that coordinate organ growth. However, a dominant-negative fragment of ERECTA has previously been shown to enhance the phenotype of null *erecta* plants, so there may be redundancy in the ERECTA signalling pathway. Torii and colleagues now identify two paralogous ERECTA-like receptors – ERL1 and ERL2. *erl1* and *erl2* mutations alone had no detectable phenotype but each enhanced the defects seen in *erecta* plants; loss of all three genes severely reduced cell proliferation, resulting in extreme dwarfism and abnormal flower development.



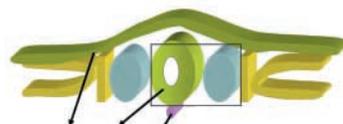
Cue Zic3: axonal guidance in the retina

During the development of the retina, ganglion cell axons project towards the optic disc, a central opening in the retina through which the axons exit to form the optic nerve. But what guides the axons to the optic disc? According to Zhang and colleagues, the zinc-finger transcription factor Zic3 helps to pattern the chick retina for intraretinal axon guidance (see p. 1553). The researchers show that *Zic3* expression is high at the periphery of the retina and low at the centre during active axon extension. Disruption of this gradient by retroviral overexpression of *Zic3* results in aberrant axon projection but does not affect retinal neurogenesis or differentiation. Finally, the pattern of axonal growth over a ‘carpet’ formed in vitro from alternating stripes of membrane fragments taken from the centre and the periphery of the retina suggests that *Zic3* upregulates the expression of a negative axon guidance cue.



Hooking up the blood supply

Multiple cellular processes are coordinated during the development of the embryonic vasculature to produce a primary vessel network that interfaces correctly with other embryonic structures. However, little is known about the signals involved in vascular patterning. On p. 1503, Hogan and colleagues propose that the neural tube is a midline signalling centre for embryonic vascular pattern formation in higher vertebrates. They show first that, in mice, ectopic neural tubes recruit a perineural vascular plexus (PNVP), a capillary bed that forms around the developing brain and spinal cord at mid-gestation. Next, they report that neural tube expression of VEGFA, a signalling molecule involved in vascular patterning, is spatially and temporally correlated with murine PNVP formation. Finally, they test the role of VEGFA in an explant model and conclude that VEGFA is a necessary component of the vascular patterning signal produced by the neural tube.



Green light for oocyte maturation

During vertebrate oogenesis, the prophase of the first meiotic division is extremely long – it lasts 4-8 months in *Xenopus* oocytes – to allow the eggs to grow and accumulate all the components necessary for early embryonic development. On p. 1543, Karaïskou et al. report that a polo-like kinase 1 (Plx1) is the critical limiting factor whose absence prevents *Xenopus* oocytes re-entering meiosis until their growth is complete. Only oocytes at stage VI of growth are competent to resume meiosis in response to progesterone, the trigger for meiotic maturation at ovulation. In these cells, progesterone induces the activation of pre-M-phase promoting factor (pre-MPF), an inactive Cdc2-cyclin B2 complex. The small amount of active MPF formed then establishes an MPF auto-amplification loop. By overexpressing Plx1 in stage IV oocytes, Karaïskou and co-workers show that the absence of Plx1 in stage IV oocytes prevents both the generation of the active MPF trigger and the establishment of an MPF auto-amplification loop, thus preventing premature entry into meiosis.

Creepy crawly developmental diversity

The adult body plan of insects is highly conserved but the developmental processes underlying its formation can vary greatly between different species. Liu and Kaufman report that *hunchback*, a gap gene involved in the subdivision of the blastoderm in *Drosophila*, has two separate roles in *Oncopeltus fasciatus*, the milkweed bug (see p. 1515). In *Drosophila*, a long germband insect, all of the body segments are specified simultaneously during the blastoderm stage. In *Oncopeltus*, an intermediate germband insect, only the anterior segments are proportionally represented in the blastoderm fate map; posterior segments are specified later, during germband elongation. In RNAi-based experiments, the researchers show that *Oncopeltus hunchback* both suppresses abdominal identity at the blastoderm stage and is required for germband growth and segmentation. Thus, although *hunchback*-depleted *Oncopeltus* and *Drosophila* both have a relatively normal head followed immediately by abdominal segments, the developmental events underlying this phenotype are very different.



In Journal of Cell Science  
Cancer without mutation?

A simple view of cancer is that cells become transformed because they acquire mutations that release them from growth constraints and allow them to become invasive. However, Maffini and co-workers show that epithelial cells can become malignant because of alterations in surrounding tissue, rather than because of mutations in their own DNA. Using a tissue recombination model that allows easy separation of epithelium and neighbouring stromal cells, they exposed epithelial cells to the carcinogen N-nitrosomethylurea (NMU) in vitro and transplanted them into rat mammary gland fat pads lacking epithelium (cleared fat pads). They also transplanted unexposed epithelial cells into cleared fat pads of rats previously exposed to NMU. Neoplastic transformation of the epithelial cells occurred only when the stroma had been exposed to NMU. The authors propose that the stroma is the target of the carcinogen and that alterations in its behaviour are necessary and sufficient to induce transformation of the neighbouring epithelium.

Maffini, M. V. et al. (2004). The stroma as a crucial target in rat mammary gland carcinogenesis. *J. Cell Sci.* **117**, 1495-1502.