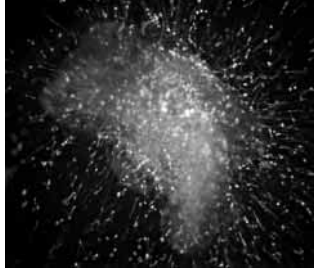


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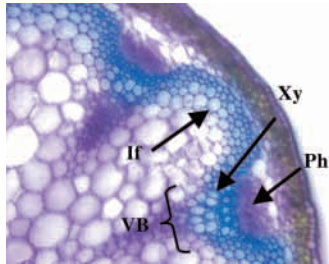
Netrin, cell repulsion and myelination

Spinal cord myelination crucially depends on the long-distance migration of oligodendrocyte precursors (OPs) that originate from a ventral ventricular zone (VVZ) domain in the developing neural tube. Tsai et al. now report that the initial dispersal of OPs from this location is mediated by a chemorepulsive response to netrin 1, which belongs to the well-known family of neuronal chemorepellants and chemoattractants. They show, on p. 2095, that netrin 1 is expressed in chick ventral spinal cord when OPs initially migrate from the VVZ. In vitro, both chick ventral spinal cord explants and netrin 1-positive cells repel migratory OPs, which express the netrin 1 receptors DCC and UNC5. This chemorepulsive response can be inhibited by a function-blocking, anti-DCC antibody. These, and other, findings reported here highlight the crucial role of netrin 1 in the process of axon myelination.



Regulating plant vascular patterning

On p. 2139, Parker et al. report the identification of a novel gene from two vascular-patterning *Arabidopsis* mutants, *cov1-1* and *cov1-2*. In the *Arabidopsis* stem, vascular bundle patterning is highly ordered, consisting of five to eight bundles of xylem and phloem that are separated by interfascicular (IF) tissue. In *cov1* mutants, this pattern breaks down in the stem, to produce plants with a continuous ring-like pattern of undefined bundles interspersed with little IF tissue. However, as the authors show, this defect is not caused by altered auxin signalling – an inducer of vascular development. They predict that *COV1* encodes an integral membrane protein, which might function in the negative regulation of xylem and phloem differentiation in stems. Future studies should provide new insights into its function and into this important, but little understood, patterning process in plants.

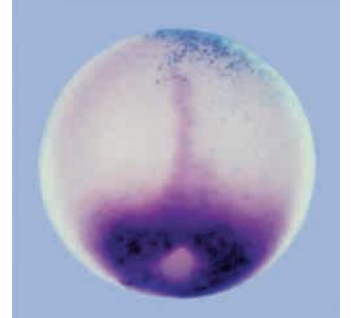


Converging Nodal and FGF signalling

Xenopus embryos have an 'odd man out' of nodal-related proteins – Xnr3. It is structurally different to other Xnrs, and, uniquely among them, induces cellular finger-like protrusions when ectopically expressed. This led Janet Heasman and colleagues to investigate the role of Xnr3 in convergent extension movements during embryogenesis with morpholino oligos. They report, on p. 2199, that Xnr3⁻ embryos have gastrulation and neurulation defects, and that Xnr3⁻ cells fail to undergo convergent extension movements in gastrulae explants. Xnr3 requires the FGF receptor FGFR1 to induce these cell elongation movements, and also interacts synergistically with FRL1 (an FGFR-binding, EGF-CFC protein), indicating that Xnr3 regulates cell movement through FGFR signalling. Importantly, because Xnr3 is a target of the maternal Wnt/β-catenin pathway, these findings link the allocation of early cell fate by this pathway to the subsequent movement of cells from the organizer.

Notch-mediated cell fate switching

To investigate the role of Notch in specifying the dorsal midline structures, the floor plate (FP) and notochord (NC), Andrés Carrasco and colleagues blocked and activated Notch signalling in early frog embryos. Their findings, on p. 2225, show that when Notch signalling is activated, *sonic hedgehog* (*shh*) and *pintallavis* (*plvs*) expression is stimulated in the FP, whereas that of the NC markers *chordin* (*chd*) and *brachyury* (*bra*) is repressed. These expression changes are accompanied by FP expansion and a reduction in NC size. From these and other findings, the authors propose that Notch activation favours FP development at the expense of that of the NC in cells in the early organiser that have the potential to develop into either of these structures. This cell-fate switch probably occurs before mid gastrula and requires Presenilin.



New role for Tbx3 in the yolk sac

To investigate the role of the T-box gene *Tbx3* in the human developmental disorder ulnar-mammary syndrome (UMS), which is caused by spontaneous mutations in *TBX3*, Virginia Papaioannou and colleagues created *Tbx3*-knockout mice. They report, on p. 2263, that although heterozygous mutant mice have only a mild developmental phenotype – UMS is a dominant disorder – the homozygous mutants recapitulate two of the main features of the disorder: abnormal forelimb and mammary gland development. Moreover, massive cell death of the yolk sac endoderm occurs in *Tbx3*-null embryos at various times in midgestation. Subsequently, and possibly as a result, mutant embryos die over several days, with none surviving to birth. These mutant mice therefore reveal a novel role for *Tbx3* in the yolk sac and provide a means for investigating the role of this gene in limb and mammary gland development.



In Journal of Cell Science Stem cell regulation in shoots

Stem cells in the shoot meristem provide a good model system for studying stem cell regulation in general. Like all stem cells, these cells must strike a balance between self-renewal and differentiation, and this is regulated in plants by signals emanating from a group of cells termed the organizing centre. In their Commentary article, Rita Groß-Hardt and Thomas Laux review our understanding of these signals and the role of the secreted peptide CLAVATA 3 (CLV3) in stem cell maintenance and differentiation, and in defining the physical boundaries of the stem cell niche despite its constantly changing population.

Groß-Hardt, R. and Laux, T. (2003). Stem cell regulation in the shoot meristem. *J. Cell Sci.* **116**, 1659-1666.