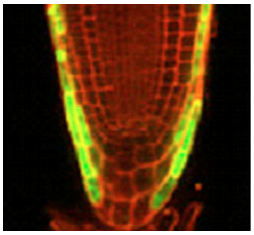
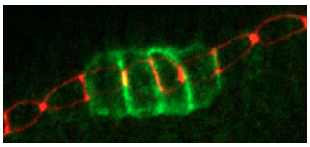


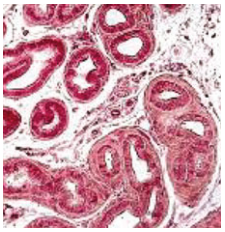
Auxin: putting down roots

The initiation of new lateral roots in *Arabidopsis* occurs at predictable distances from the growing tip and depends on auxin transport and redistribution. However, the precise mechanism that regulates the positioning of new roots has remained elusive. Tom Beeckman and colleagues now show, on p. 681, how oscillating waves of auxin accumulation and response in the cells that exit the root meristem bring about the regular left-right alternating pattern of lateral root development along the main root axis. AUX1, an auxin influx carrier, is essential for this left-right patterning in response to gravity; it transports auxin from cells exiting the root tip back to those still in the root tip. Cells between the growing tip and the meristem display an oscillatory responsiveness to auxin with a periodicity of 15 hours. The authors demonstrate that this peak in auxin responsiveness corresponds precisely with the formation of a lateral root. Thus cells are primed for root development while still in the root tip.



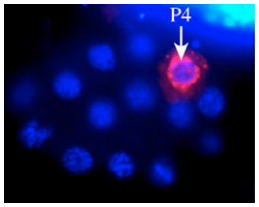
*LIN*king neurobeachin to endocytosis

Vulval precursor cells (VPCs) of *C. elegans* have the potential, through reciprocal LIN-12/Notch and LET-23/EGFR signalling, to give rise to either vulval or non-vulval fates. In VPCs in which LET-23 is activated maximally, LIN-12 is endocytosed and degraded. Now on p. 691, Iva Greenwald and co-workers report the isolation of SEL-2, a *C. elegans* homologue of mammalian neurobeachin (which is required for neurotransmission at neuromuscular junctions) and LRBA (which positively regulates EGFR in cell culture). SEL-2, they show, is required for efficient endocytosis in epithelial cells and for maintaining LIN-12 in a steady state. LIN-12 is mislocalised basolaterally in *sel-2* mutants and cannot be degraded in response to Ras activation. Moreover, endocytosis is compromised in the intestinal epithelium of these mutants, as revealed by the basolateral accumulation of a dye that marks endocytic vesicles. In VPCs, LIN-12 trafficking and stability, rather than its transcription, is modulated, and SEL-2's role may contribute to the mechanisms required for these processes and for cell fate specification.



Prostates get into shape with FGFR2

The adult prostrate depends on androgens for its growth and function – its epithelium regresses when androgens are depleted. FGF signalling, through the FGF receptor FGFR2, has been implicated in mouse prostate development, but studies of FGFR2's role in prostate organogenesis have been hampered by the early embryonic death of *Fgfr2*-null mutants. On p. 723, Fen Wang's group report, from their studies of conditional *Fgfr2* mutant embryos, that FGFR2 is required for prostate growth and morphogenesis and for certain aspects of this organ's androgen dependency. Branching morphogenesis is particularly affected in these mutants, and despite the continued ability of *Fgfr2* conditional mutant prostates to secrete proteins in response to androgen, their ability to regulate tissue maintenance in an androgen-dependent manner is compromised. As advanced prostate tumours can often grow independently of androgen, further studies into the molecular mechanisms that define how FGFR2 regulates the prostate's maintenance and growth in an androgen-dependent manner could yield new therapeutic targets for the treatment of these aggressive cancers.

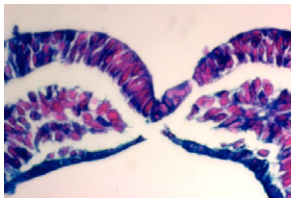


Silence please for MRG-1

Germ cells are unique in their ability to give rise to the next generation, and thus must remain undifferentiated to maintain totipotency. Mammalian mortality-factor-related MRG15 is a chromodomain protein that regulates transcription. The *C. elegans* orthologue of MRG15 is MRG-1, and now Hiroshi Sakamoto's and Susan Strome's labs (see p. 757) report that MRG-1 is required for germline development and for X chromosome silencing in *C. elegans*. Surprisingly, however, MRG-1 localizes only to autosomes and is undetectable on X chromosomes. MRG-1 shares with the maternal-effect sterile protein, MES-4, an autosomal localisation and the ability to repress genes in the germ line. However, MRG-1's autosomal localisation does not depend on MES-4 activity, and vice versa. MRG-1 might, the authors propose, act in a complex to modify chromatin organisation and gene expression through the regulation of histone acetylation. They further suggest that MRG-1 might de-repress autosomal genes that can silence genes on the X chromosome; these autosomal genes, however, have yet to be identified.

SRF: muscling in on ectoderm

The inhibition of Activin/Nodal signalling during germ layer formation is essential for ectodermal specification and for correctly positioning the endoderm and mesoderm. As serum response factor (SRF)-deficient mice die in early embryonic development, Yun et al. turned to *Xenopus* embryos, where mesodermal induction is better characterised, to investigate the contribution that SRF makes to mesoderm development, They reveal, on p. 769, that the ectopic expression of SRF RNA suppresses mesoderm induction in the marginal zone (where animal and vegetal hemispheres meet) of frog embryos, and also in cultured animal caps, by inhibiting Activin/Nodal signals. Activin signalling induces the binding of FAST-1 and Smad2 to each other. Since XSRF competes with FAST-1 to bind Smad2, it thus terminates Activin signalling. The authors demonstrate how the inhibition of XSRF function by antisense morpholinos causes the expression domain of mesodermal genes to expand within ectodermal territory, and how this enhances the inducing activity of Activin signalling. Thus, SRF ensures that correct germ layer specification occurs by regulating Activin signalling.



PCP: mind the gap

Planar-cell-polarity (PCP) signalling confers polarity on cells within an epithelium, and contributes to the correct fusion of neural folds during neural tube closure in higher vertebrates. Mutations in mouse PCP genes cause severe neural tube defects – such as are also seen in 10% of human neural tube defects. Surprisingly, whether convergent extension (CE) is involved in such defects is unknown. Now, on p. 789, Andrew Copp and colleagues reveal that CE is defective in the axial mesoderm and neuroepithelium of mice with mutant PCP genes, such as *loop-tail* (*Lp*), before the onset of neurulation. Wild-type cells occur at the midline of chimeric *Lp* mice, indicating that this process is cell autonomous. Unlike in other vertebrates, CE in mice depends on downstream RhoA, but not JNK signalling. Other findings and those reported here should lead to the search for more candidate genes that underlie human neural tube defects. (For more on PCP in vertebrates, see p. 647 for a review by Yanshu Wang and Jeremy Nathans.)