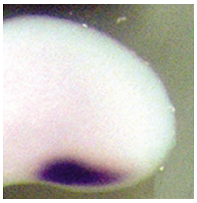
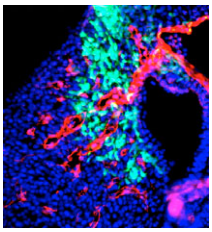
Antagonism in *Arabidopsis* mediates patterning

Auxin regulates gene expression in *Arabidopsis* through Auxin Response Factors (ARFs). While most ARF functions remain elusive, patterning functions have been assigned to some, including *MONOPTEROS* (*MP*/*ARF5*), which promotes stem cell formation in the root and shoot apical meristem. Thomas Berleth's group now show that *MP* primarily functions to counteract the activity of the carboxypeptidase *ALTERED MERISTEM PROGRAM 1* (*AMP1*), which restricts meristem size. Their analysis of single and double mutants in *Arabidopsis,* reported on p. 2561, reveals that in the absence of *AMP1* activity, *MP* patterning activity is largely dispensible, and that in *MP* mutants, meristem cells differentiate because of unimpeded *AMP1* activity. These researchers propose that *MP* represses *AMP1*'s activity and maintains niches, an idea that is supported by the two genes' overlapping expression domains: where they overlap, antagonism occurs. As *AMP1* transcript levels are normal in *MP* mutants, this antagonism is not transcriptionally regulated. Moreover, MP and AMP1 localise to different cellular compartments, so exactly how this antagonism occurs remains unknown.



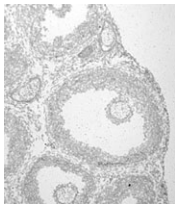
Hedgehog's fantom

Mice with the fused toes (*Ft*) mutation – a 1.6 Mb chromosomal deletion encompassing six genes – exhibit phenotypes reminiscent of disrupted Hedgehog (Hh) signalling. Vierkotten et al. have individually knocked out these genes in mice to identify which one is involved in Hh signalling. On p. 2569, they reveal that the loss of fantom (*Ftm*) accounts for most defects in *Ft* mice. Importantly, they report that Ftm localises to the ciliary basal body where it functions as a novel component of cilia-related Hh signalling – regulating the Gli3 activator (Gli3A) to processed Gli3 repressor (Gli3R) ratio (Gli transcription factors act downstream of Hh). In *Ftm* mutant mice, Gli3 levels increase; however, it remains unprocessed, altering the ratio of Gli3A:Gli3R and consequently Hh transcriptional output. Interestingly, Ftm is not required for cilia assembly; however, cilia numbers are reduced where morphogenetic processes occur and thus cilia-coupled signalling might be required for cilia maintenance. In *Drosophila*, Hh signalling occurs independently of cilia; unsurprisingly, a fly *Ftm* homologue hasn't been identified.



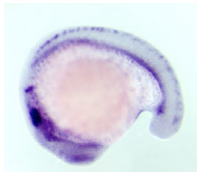
Muscle-splitting vessels

During limb development, dorsal and ventral muscles progressively separate to form individual muscles in a process called muscle splitting. In their study on p. 2579, Duprez and colleagues reveal that blood vessels regulate this process. They report that the location of endothelial cells, which are present in the developing chick limb before muscle, determines the site of future zones of muscle cleavage. By overexpressing VEGFA (a key growth factor in blood vessel development) in chick wing buds prior to muscle splitting, the researchers induced both blood vessel and connective tissue formation, while inhibiting muscle formation. Conversely, blocking blood vessel formation with a soluble VEGFR1 in chick wing buds caused muscle fusion. The authors propose that PDGFB (platelet-derived growth factor B), which is expressed in endothelial cells, is the molecular signal that regulates this process, perhaps by promoting the production of the extracellular matrix and attracting connective tissue cells to future sites of muscle splitting.



Oocytes trigger glycolysis

Mammalian oocytes cannot perform the essential metabolic process of glycolysis and so rely on their companion cumulus cells to provide them with glycolysis products. To facilitate this, oocytes secrete paracrine factors that promote the expression of glycolytic enzymes, such as platelet phosphofructokinase (PFKP) and lactate dehydrogenase A (LDHA), in cumulus cells. John Eppig and colleagues now show that oocyte-derived BMP15 and FGF8 cooperatively promote *Pfkp* and *Ldha* expression and glycolysis in mouse cumulus cells (see p. 2593). In *Bmp15*$^{-/-}$ and *Bmp15*$^{-/-}$ *Gdf9*$^{+/-}$ mutant mice, both *Pfkp* and *Ldha* expression and glycolysis are reduced in cumulus cells. Moreover, oocytes from these mutant mice are unable to promote glycolysis in wild-type cumulus cells. The co-culture of cumulus cells (without an oocyte) with BMP15 and FGF8 promotes *Pfkp* and *Ldha* expression and glycolysis, whereas the co-culture of cumulus cells with GDF9 and either BMP15 or FGF8 does not induce glycolysis. Understanding how BMP and FGF signals cooperate in this setting will clarify our knowledge of oocyte and follicular development.



Novel developmental function for cohesin

The Runx1 transcription factor – an essential hematopoiesis regulator – is involved in several leukaemia-causing chromosomal translocations. An important route to understanding the pathology of such Runx-mediated cancers is to elucidate what controls Runx expression or stability. Philip Crosier and colleagues have now discovered, from a genetic screen in zebrafish for positive regulators of *runx1*, that Rad21, a cohesin subunit, regulates *runx1* transcription in a dose-dependent manner. This depends on cohesin, a protein complex that is required for sister chromatid cohesion (p. 2639), and is the first example of cohesin-dependent gene regulation in vertebrates. In a series of genetic and morpholino-knockdown experiments, the authors show that in zebrafish *rad21* mutants, differentiated blood cells do not form and *runx3* and hematopoietic *runx1* expression is lost. Removing one copy of *rad21* reduces *runx1* and also downstream proneural gene expression. Knocking down Smc3 – another cohesin subunit – similarly reduces *runx1* expression. Thus, the cohesin complex regulates *runx1* expression in zebrafish, but exactly how, awaits future work.

IN *JOURNAL OF CELL SCIENCE*

Rho signalling delivers the milk

Milk secretion by mammary glands is established at birth by a series of coordinated switches, such as the sealing of tight junctions between secretory epithelial cells. Now, Fischer et al. report that the Rho effector protein PKN1 (protein kinase N1) contributes to this process. By making transgenic mice that express constitutively active PKN1 in the mammary epithelium, the authors show that although the mammary glands of these mice develop normally during pregnancy, they lactate poorly and their glands involute soon after parturition. By injecting radioactive sucrose intraductally, the authors demonstrate that the epithelial tight junctions in the lactating transgenic glands are poorly sealed. Furthermore, PKN1 expression in mammary epithelial cell cultures interferes with tight junction sealing. Thus, PKN1 and Rho signalling are necessary to keep tight junctions open during pregnancy, and the downregulation of Rho signalling at birth leads to tight junction sealing.

Fischer et al. (2007). Impaired tight junction sealing and precocious involution in mammary glands of *PKN1* transgenic mice. *J. Cell Science* **120**, 2272-2283.