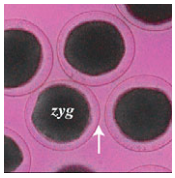
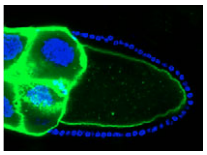


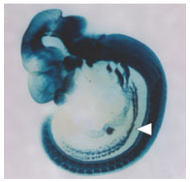
Radical ECM changes at fertilization

When an animal egg is fertilized, the zygote rapidly transforms the extracellular matrix (ECM) of the egg into a protective structure in which embryogenesis can proceed. On p. 431, Wong and Wessel provide new insights into the biochemistry of this important transformation in the sea urchin fertilization envelope. Soon after insemination, they report, the fertilization envelope becomes impermeable to molecules larger than 40,000 daltons (medium-sized proteins; for example, serum albumin) via a peroxidase-dependent protein crosslinking mechanism. Using a new in vivo technique to label and isolate the modified ECM components, they show that four major components of the fertilization envelope are selectively crosslinked in a manner that distinguishes between protein isoforms derived from the same gene. The authors speculate that this selectivity is partly due to the local clustering of target proteins within the fertilization envelope and conclude that the free-radical crosslinking of specific proteins is essential for establishing the embryonic microenvironment that is needed for early development.



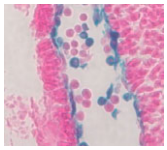
Par-sing the regulatory circuitry of oocyte polarity

In *Drosophila* oocytes, polarization of the microtubule cytoskeleton localizes the maternal RNAs that subsequently specify the anteroposterior (AP) and dorsoventral axes, but how is cytoskeletal polarity established and regulated? On p. 463, Tian and Deng report that the tumour suppressor Lethal (2) giant larvae (Lgl) and atypical protein kinase C (aPKC) play important roles in regulating microtubule polarity and in setting up the AP axis in *Drosophila* oocytes. They show that the loss of *lgl* in germline cells disrupts the normal localization of oocyte polarity markers. Restriction of Lgl activity to the posterior of the oocyte by anterior aPKC (Lgl is inactivated by phosphorylation by aPKC) is also needed for the correct localization of these markers, they report. Furthermore, active Lgl regulates the posterior enrichment of Par-1, a serine/threonine kinase that controls microtubule polarity in *Drosophila* oocytes. Together, these results indicate that a regulatory circuit that involves Lgl and its phosphorylation by aPKC establishes oocyte polarity.



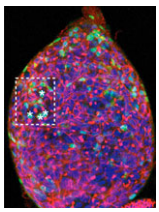
Insm1: crucial for sympatho-adrenal differentiation

Neural crest cells generate numerous different cell types in vertebrate embryos, including the 'sympatho-adrenal' precursors. These cells mature into sympathetic neurons (which control the 'fight or flight' response) and adrenal chromaffin cells (which make adrenalin and noradrenalin). Now, Carmen Birchmeier and colleagues report that the insulinoma-associated 1 (*Insm1*) gene, which encodes a zinc-finger transcription factor, is a central component of the transcriptional network that controls the differentiation of the sympatho-adrenal lineage (see p. 473). The researchers show that the differentiation of sympatho-adrenal precursors is delayed and their proliferation reduced in *Insm1* mutant mice. The expression of Mash1, Phox2 and other components of the transcriptional network that controls the differentiation of the sympatho-adrenal lineage is deregulated in *Insm1* mutant mice, they report. In addition, *Insm1* and *Mash1* mutant mice have similar phenotypes. Other data indicate that *Insm1* acts downstream of *Mash1* and *Phox2b* genetically and that *Insm1* represses *Mash1*. The researchers speculate, therefore, that Insm1 mediates certain aspects of Mash1 function during the differentiation of sympatho-adrenal precursors.



BRG1 opens up primitive erythropoiesis

During development, ATP-dependent chromatin-remodelling complexes control certain temporal and spatial changes in gene expression that drive differentiation. Mammalian SWI-SNF-like chromatin-remodelling complexes contain one of two ATPase subunits: brahma (BRM) or brahma-related gene 1 (BRG1). Now, Griffin and colleagues report that BRG1-containing SWI-SNF-like complexes are required for primitive erythropoiesis and early vascular development in mice (see p. 493). The researchers show that when *Brg1* is conditionally deleted in developing haematopoietic and endothelial cells, mouse embryos die at midgestation from anaemia. The primitive erythrocytes in these embryos fail to transcribe embryonic α- and β-globins, they report, and subsequently undergo apoptosis. Vascular remodelling in the extra-embryonic yolk sac of *Brg1* mutant embryos is also abnormal, but the additional loss of *Brm* does not exacerbate their erythropoietic or vascular abnormalities. These results extend previous experiments in which hypomorphic *Brg1* mutations prevented the transcription of adult but not of embryonic β-globin genes, note the researchers, and reveal non-redundant roles for BRM and BRG1 during primitive erythropoiesis and early vascular development.



Jak/Stat signalling finds its niche

Specialized regulatory microenvironments (niches) sustain stable stem cell populations in many tissues, but how the support (stromal) cells that function within these niches sustain stem cells is poorly understood. Now, on p. 533, López-Onieva and co-workers implicate Jak/Stat (Janus kinase/Signal transducer and activator of transcription) signalling in germline stem cell (GSC) maintenance in the *Drosophila* ovary. GSCs in the *Drosophila* ovary are supported by terminal filament cells, cap cells and escort stem cells; these last two cell types make Dpp, a BMP-like molecule that is needed for GSC maintenance. The researchers show that the Jak/Stat pathway is normally active in cap cells and that GSCs are lost through differentiation when Jak/Stat signalling is switched off in these support cells. Conversely, ectopic activation of Jak/Stat signalling in the support cells induces stem cell tumours and increases *dpp* transcription in the niche's stromal cells. These results suggest that Jak/Stat signalling regulates *dpp* transcription in the support cells of the GSC niche to maintain ovarian GSCs in an undifferentiated state.

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Axon identity: a question of timing

During the development of multipolar neurons, many neurites extend from the cell body, one of which eventually differentiates to form the mature axon; the remainder become dendrites. But how and when is the axon-to-be selected? Some studies suggest that this occurs when one of the two neurites first extends from opposite poles of the cell body. But evidence also indicates that stochastic selection occurs at a later stage, when more neurites have sprouted. Now, Carlos Dotti and co-workers show that in bipolar-stage neurons in situ, only one of the first two neurites contains the Tau-1 protein (an axonal marker) and that, in most cases, one of these neurites develops into the axon. The polarised organisation of microtubules and membrane traffic at the bipolar stage also promote axonal growth at one of the two neurites, and severing the axon of a multipolar neuron causes axon formation at the opposite pole of the cell, findings that together shed new light on dendrite morphogenesis.

Calderon de Anda, F. et al. (2008). Pyramidal neuron polarity axis is defined at the bipolar stage. J. Cell Sci. 121, 178-185.