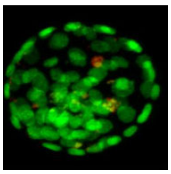
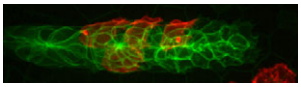


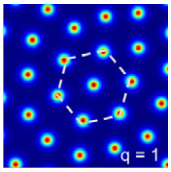
CTCF gets maternal

Maternal effect genes are transcribed in the oocyte and are essential for embryonic development. Few are known in mammals, but now Marisa Bartolomei and co-workers add *Ctcf* to this short list (see p. 2729). In vertebrates, CTCF regulates transcription at genomic loci by binding to enhancer and insulator sequences. In an earlier study into CTCF binding and activity at the maternally imprinted *H19/Igf2* locus, the Bartolomei lab generated a transgenic mouse in which growing oocytes are specifically depleted of CTCF by RNAi. Using microarrays, they have now identified hundreds of genes that are misregulated in these CTCF-depleted oocytes. Most genes are downregulated; moreover, downregulated genes occur closer to CTCF-binding sites. Oocyte CTCF depletion, they report, delays not only meiosis onset but also the second, post-fertilisation division; it also perturbs zygotic genome activation, alters nuclear morphology and causes apoptotic early embryonic death. These abnormalities, further experiments show, are very likely to be a maternal effect caused by transcriptional, rather than chromatin, defects. Together, these findings reveal new and independent CTCF functions in oocyte and embryonic growth.



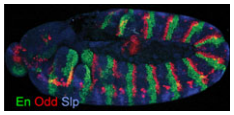
Fgf signals followers from leaders

During organogenesis, the migration of epithelial tissues can be controlled by their leading edge, a region that detects external guidance cues and directs cohesive tissue movement. An example of this is the migrating primordium of the zebrafish lateral line, from which mechanosensory organs arise as a result of rosette-like structures being deposited by the trailing cells of the primordium. On p. 2695, Darren Gilmour and colleagues show that, in this tissue, cells behind the leading edge become assembled into sensory organ progenitors in response to Fgf signalling. By using *fgf3;fgf10* double mutant fish and by inhibiting Fgf signalling, the authors demonstrate that this pathway is necessary for organising prospective sensory organs and for driving cells towards an increasingly epithelial, non-leader fate as they fall behind the leading edge. While Fgf signalling has been shown to select 'leader' cell fate in several different contexts, this new work reveals that the same signalling pathway can drive this fate transition in the reverse direction, turning leaders into followers.



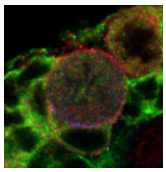
Dusting off BMP roles in feather development

In birds, various signalling pathways are implicated in the dermis-epidermis crosstalk that leads to feather morphogenesis. Among these is the BMP pathway, which is believed to inhibit feather formation, yet the evidence for it doing so is somewhat contradictory. On p. 2797, Michon and colleagues shed some light on this issue with their study and mathematical modelling of BMP signalling during feather formation in chicks. Their findings reveal that rather than acting just as inhibitors, different BMPs have distinct, sometimes antagonistic, roles in feather development. To distinguish these roles, the authors investigated the expression of BMP target genes and studied in vitro dermal fibroblast behaviour in response to different BMPs. From these and other findings, they generated a mathematical model – a reaction-diffusion model – that simulates feather patterning and accounts for the negative effects that excess BMP2 and BMP7 have on feather formation. From their results, the authors propose a new view of BMPs and dermis organisation that agrees with previous findings but not with their interpretation.



Hh and Wg: inducers or stabilisers of cell fate?

As Jeff Axelrod and co-workers state in their paper (see p. 2767), 'a fundamental concept in development is that secreted molecules such as Wg and Hh generate pattern by inducing cell fate'. They now cast doubt on this concept by reporting that, in *Drosophila* embryos, Wg and Hh generate pattern by inhibiting specific switches in cell identity rather than by specifying cell identity. They reached this conclusion by studying the specification and patterning of the segmental grooves that develop immediately posterior to the Hh-secreting, *en*-expressing cell stripes in the fly embryonic epidermis. By identifying Odd as a groove cell (GC) marker, the authors traced GC lineage and found that Wg, by maintaining En in these stripes, inhibits the development of Odd-expressing GC precursors. Thus, Wg signalling stops cells from switching to a GC identity, while cells beyond its reach can make this switch. Similarly, Hh, in a subsequent step, refines the GC pattern by blocking another transition towards a more posterior fate. How general this inhibition of cell identity progression by patterning signals is, awaits further investigation.



Novel aPKC regulator rises to the bait

The atypical protein kinase C (aPKC) is required for the polarisation of many cell types and has important roles in neural stem cell identity and proliferation. Yet, despite its importance for cell polarity and growth, little is known about how its activity is regulated. Now Chabu and Doe (on p. 2739) report that, in *Drosophila*, Dynamin-associated protein 160 (Dap160) positively regulates aPKC, and that aPKC requires it to establish neuroblast (NB) cell polarity and cell cycle progression. The authors identified Dap160 by performing immunoprecipitation experiments coupled to mass spectrometry using aPKC as the bait. They show that Dap160 directly interacts with aPKC and stimulates its activity in vitro. In vivo, it colocalises with aPKC at the apical cortex of embryonic NBs; in *dap160* mutants, aPKC delocalises from here and has reduced activity. In addition, in both *dap160* and *aPKC* mutants, fewer proliferating NBs with a prolonged cell cycle are found. Exactly how Dap160 localises aPKC and promotes cell cycle progression remain questions for the future.

IN JOURNAL OF CELL SCIENCE

New axon regeneration Tr(i)x

The regeneration of an axon after injury is enhanced by prior 'conditioning' lesions, which are believed to stimulate the expression of regeneration-associated genes (RAGs). Conditioning lesions have typically been introduced in vivo, but Tonge et al. now show in *Journal of Cell Science* that *Xenopus* dorsal root ganglia with attached peripheral nerves (PN-DRG) can be 'conditioned' in vitro by a 3-day incubation in serum-free medium. The authors demonstrate that BDNF treatment stimulates axonal outgrowth in conditioned PN-DRG, even in the presence of the transcriptional inhibitor actinomycin D; thus, de novo mRNA synthesis occurs during in vitro conditioning, as it does in vivo. Moreover, inhibiting protein synthesis specifically in the distal nerve impairs the conditioning response, and the authors identify 32 proteins that are synthesised and undergo retrograde transport during conditioning. Notably, inhibitors of Trx and Trx-specific morpholino oligonucleotides inhibit the conditioning response. Trx might, therefore, promote axonal regeneration, perhaps by upregulating RAGs.

Tonge, D. et al. (2008). Enhancement of axonal regeneration by in vitro conditioning and its inhibition by cyclopentenone prostaglandins. J. Cell Sci. 121, 2565-2577.