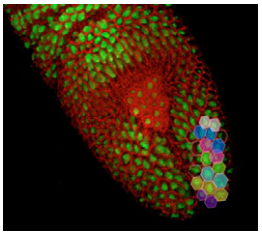
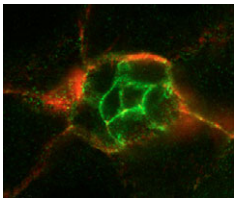


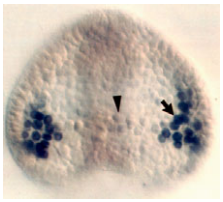
Axis elongation: flies do it without PCP

Tissue elongation is a general feature of morphogenesis. In vertebrates, embryonic axis elongation involves cell intercalation and oriented cell division, processes controlled by the planar cell polarity (PCP) pathway (which ensures that neighbouring cells adopt the correct polarity in developing tissues by promoting cell-cell communication). But what about invertebrate embryos? On p. 3049, Morais da Silva and Vincent report that both processes contribute to germband elongation (GBE) in *Drosophila* embryos. The germband, which forms the larva's trunk, doubles in length during early embryogenesis. Elongation of the anterior germband involves cell intercalation but not oriented cell division. By contrast, using time-lapse imaging of histone–GFP-labelled embryos, the researchers show that mitoses in the posterior germband are orientated along the axis of elongation during the fast phase of GBE and that cell division inhibition reduces GBE. This orientation of cell division, like cell intercalation in the anterior of the germband, requires segmental patterning, but neither process requires the PCP pathway. The authors propose, therefore, that an alternative means of planar polarisation must mediate tissue elongation in *Drosophila* embryos.



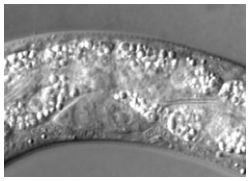
PCP pathway directs border cell manoeuvres

Directed cell migration is another essential feature of morphogenesis. But, although the migration of single cells is well characterised, less is known about the coordinated movement of groups of cells. Now, Bastock and Strutt report that the PCP pathway coordinates cell migration during *Drosophila* oogenesis (see p. 3055). During this process, motile epithelial border cells detach from the anterior of the developing egg chamber and migrate towards the oocyte, carrying two non-motile polar follicle cells with them. By examining egg chambers from flies carrying mutations in the PCP pathway proteins Frizzled, Strabismus and Dishevelled, the researchers show that the pathway acts in the border cells and the polar follicle cells to promote migration. Other experiments lead them to propose that the PCP pathway mediates communication between motile and non-motile cells and promotes the production of the actin-rich structures that are required for efficient, coordinated migration.



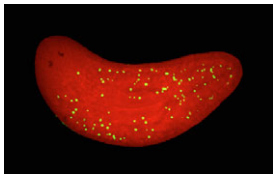
Network deployments for developmental plasticity

Cell fates in sea urchin embryos are remarkably labile. For example, mesodermal lineages can activate the transcriptional gene regulatory network (GRN) that drives skeletogenesis if the micromere precursors of the primary mesenchyme cells (PMCs, the cells that form the embryonic skeleton) are removed. To determine the molecular basis of this plasticity, Ettensohn and colleagues have examined the conversion of non-skeletogenic mesoderm (NSM) to a PMC fate during gastrulation and reveal that most, but not all, of the upstream transcription factors in the skeletogenic GRN are recapitulated by transfating cells (see p. 3077). They show that the transcription factor *alx1*, a key component of the skeletogenic GRN, is expressed in transfating NSMs, that *alx1* expression in transfating NSMs and in PMCs requires MAPK signalling, and that *alx1* expression in micromeres normally suppresses NSMs from transfating. However, the transcription factor *pmar1* (which activates the skeletogenic GRN in PMCs) is not needed in transfating NSMs. Thus, the skeletogenic GRN is activated by distinct mechanisms during normal and regulative development.



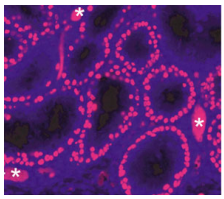
Worming a way into cell fate evolution

Studies into vulva formation in nematodes have provided insights into key developmental mechanisms and their evolution. Now, on p. 3111, Yi and Sommer reveal that different regulatory networks specify the cells that form the vulva (the vulva equivalence group, VEG) in *Pristionchus pacificus* and *Caenorhabditis elegans*. In both nematodes, establishment of the VEG from the middle of the ventral epidermis requires the Hox gene *lin-39*. In *C. elegans*, the anterior and posterior non-vulval epidermal cells fuse with the hypodermis (a process indirectly regulated by *lin-39*) but, in *P. pacificus*, these cells die. Yi and Sommer isolated *gev-2*, a vulvaless mutant of *P. pacificus*, and show that *gev-2* is the *P. pacificus pax-3* gene (*Ppa-pax-3*). *Ppa-pax-3* is a direct target of *Ppa*-LIN-39 and regulates the survival of the VEG precursors but induces the death of the posterior epidermal cells, they report. Thus, a different regulatory network in *P. pacificus* specifies the VEG than in *C. elegans*, a finding that sheds new light on the evolution of fate specification.



Gap junctional talk regulates adult stem cells

Adult stem cells are regulated by interactions with neighbouring differentiated cells, but what is the molecular basis of these interactions? Such information would be useful in the context of regenerative medicine and cancer biology. Now, Oviedo and Levin report that, in the planarian worm *Schmidtea mediterranea*, the regulation of neoblasts (adult stem cells that proliferate after injury and regenerate damaged tissues) requires *smedinx-11*, which encodes an innexin, an invertebrate gap junction protein (see p. 3121). The researchers investigated innexin transcripts in *S. mediterranea* as potential regulators of neoblasts because gap junction-permeable signals have been implicated in embryonic patterning and morphogenesis. They show that *smedinx-11* is expressed in the neoblasts and that treatment of the worms with *smedinx-11* RNAi abrogates neoblast proliferation and inhibits regeneration. It also prevents neoblast maintenance and disrupts the normal anterior-posterior gradient of mitotic neoblasts. The researchers suggest, therefore, that gap junctional communication regulates the interactions of adult stem cells with differentiated cells that control their behaviour in multicellular organisms.



Segregating new development and disease roles for cohesin

The cohesin complex ensures accurate sister chromatid segregation during cell division but it also seems to play an important role in development. For example, mutations in several cohesin components are associated with the human developmental disorder Cornelia de Lange syndrome (CdLS). Until now, there has been no animal model for this syndrome but, on p. 3191, Zhang and co-workers report that mice lacking the cohesin regulatory protein PDS5B are born with developmental abnormalities reminiscent of CdLS. *Pds5B*-deficient mice, like people with CdLS, exhibit abnormal skeletal patterning, heart defects and cleft palates, they report. Unexpectedly, however, the researchers did not find any chromosome cohesion defects in *Pds5B*$^{-/-}$ cells. Furthermore, they detected high PDS5B expression in post-mitotic neurons of wild-type mice, identified a DNA-binding domain in mouse PDS5B and showed that the protein localizes to the nucleolus. Overall, these results suggest that PDS5B and the cohesin complex might regulate multiple aspects of organogenesis by regulating developmental gene expression rather than chromosome dynamics.

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