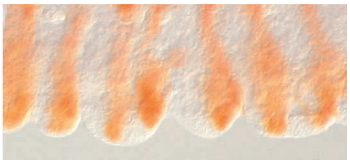
In this issue

By Jane Bradbury

Setting the boundaries: segmentation in *Drosophila*

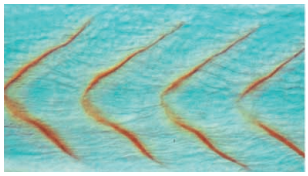
For patterning and growth to occur in well-defined embryonic regions during development, boundaries need to be established between groups of cells. On p. 5625, Larsen et al. report that both Hedgehog signalling and Engrailed expression play distinct roles in promoting the morphological changes associated with the formation of epidermal segment boundaries in *Drosophila* embryos. These boundaries, which are visible as deep grooves in the epidermis, form at the posterior edges of stripes of *engrailed* expression.



The researchers describe how *engrailed*-expressing cells at the boundary undergo apical constriction, move inwards and become bottle shaped. The results of genetic analyses reveal that for these events to occur, Hedgehog signalling and Engrailed expression are needed at the posterior and anterior, respectively, of each boundary, and that Wingless signalling at the anterior of the segments prevents boundary duplication.

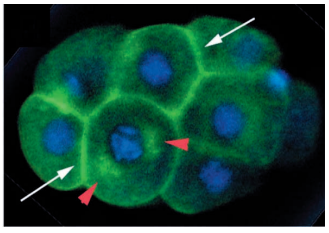
Fish model for Duchenne muscular dystrophy

In zebrafish, the recessive lethal mutation *sapje* causes progressive degeneration of skeletal muscles, reminiscent of human muscular dystrophies. Bassett and co-workers show that the *sapje* mutation disrupts the zebrafish orthologue of the human *Duchenne muscular dystrophy* (*DMD*) gene (see p. 5851). In a detailed study of the structure of the muscles throughout embryonic development, the researchers report that the progressive muscle degeneration in the mutant fish is caused by the separation of somitic fibres from their attachment points on tendon-like sheets of extracellular matrix, called myosepta. These attachment points resemble mammalian myomuscular and myotendinous junctions. However, although structural deficits of myotendinous junctions have been seen in mouse models of Duchenne muscular dystrophy, dystrophin has not previously been implicated in the maintenance of either structure. Thus, say the researchers, the *sapje* mutant may be a good model for a hitherto unsuspected pathological mechanism in muscular dystrophies.



Initiation of handedness in *C. elegans*

In most animals, the placement of the internal organs has left-right (LR) asymmetry with an invariant handedness. How handedness – whether an organ lies on the right or the left – is initiated during development is unclear. On p. 5731, Bergmann and co-workers identify GPA-16, a component of a heterotrimeric G protein, as the first *C. elegans* protein that affects handedness. LR asymmetry in *C. elegans* becomes evident sometime between the four- and six-cell stages, and is determined by a shift in the orientation of specific mitotic spindles. The researchers show that loss-of-function of GPA-16 affects spindle orientations during the third cleavage and nearly randomises handedness among the resulting adult worms. Heterotrimeric G proteins are also involved in the control of asymmetric cell division, and on p. 5717, Tsou et al. show that G-protein signalling interacts with LET-99, a protein whose localisation pattern is dependent on polarity cues, to regulate spindle orientation, and thus asymmetric cell division, in early *C. elegans* embryos.

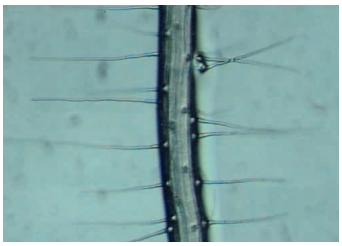


Driving neural cell migration

Organised cell migrations transform embryos from featureless spheres into recognisable animals. For a cell to move, cytoskeletal and other proteins need to be localised to its leading and trailing edges. Yaniv et al. (see p. 5649) examine whether this localisation is driven by RNA-binding proteins (RBPs). They report that one such RBP – Vg1 RBP – is localised to the extended processes of migrating neural crest cells in *Xenopus* embryos. When Vg1 RBP expression was reduced by antisense morpholino oligonucleotide injection, neural crest cells, although correctly specified, did not migrate normally and the neural tube roof plate failed to form properly because of defective neuroepithelial cell migration. Vg1 RBP, conclude the researchers, could facilitate normal cell migration by mediating the sorting of specific RNAs to the leading edge of migrating cells.

Hairy tips for plant development

Many developmental processes in plants are controlled by the hormone auxin (indole-acetic acid) via regulation (both at the level of transcription and of protein degradation) of the abundance of the Aux/IAA family of transcriptional regulators. Surprisingly, similar mutations in different but highly homologous Aux/IAAs can have very different phenotypic effects, but how this specificity is achieved is unknown. On p. 5769, Knox et al. describe how a gain-of-function mutation that makes AXR3 resistant to auxin-mediated degradation blocked root hair initiation and elongation in *Arabidopsis*, while a similar mutation in the homologous SHY2 stimulated early root hair initiation and prolonged elongation. When the mutant forms of AXR3 and SHY2 were co-expressed, aberrant root hairs were initiated but failed to grow. On the basis of their results and the known dimerisation properties of the Aux/IAAs, the researchers propose that the relative abundance of different Aux/IAAs, rather than their absolute amounts, is the key for determining which developmental process auxin induces.



In *Journal of Cell Science*

Bony growth: the importance of JunB

Most skeletal bones are formed by endochondral ossification, during which osteoblasts replace the cartilage scaffold laid down by chrondrocytes with bone. Exactly how the proliferation and differentiation of chrondrocytes and osteoblasts is controlled during ossification is not known. However, Hess and co-workers now report that, in mice, this is regulated by JunB, a member of the AP-1 transcription factor family. The JunB knockout is embryonic lethal; so the researchers developed two *junB*$^{-/-}$ Ubi-*junB* mouse lines, in which expression of JunB was controlled by the human ubiquitin C promoter. This rescued the embryonic lethal phenotype but resulted in reduced JunB expression in several adult tissues, including bone. Longitudinal bones were shorter in these mice than in wild-type mice, and the expression of several key cell-cycle regulators was deregulated in chrondrocytes and osteoblasts. The researchers suggest that studies on conditional knockout mice will help to determine the exact role of JunB in the osteoblastic lineage.

Hess, J. et al. (2003). Defective endochondral ossification in mice with strongly compromised expression of JunB. *J. Cell Sci.* **116**, 4587-4596.