Inner ear induction by FGFs

Ectodermal otic placode (OP) development, which gives rise to the inner ear, is believed to be directed by signals, such as the FGFs, that emanate from the mesenchyme and neuroectoderm that lie underneath and adjacent to this tissue, respectively. However, whether FGF's signal directly to the prospective placode or are required for hindbrain-expressed OP inducers has remained unclear. Wright and Mansour now shed light on this question with their study of OP induction in Fgf3/Fgf10 double knockout mice (see p. 3379). These mice lack otic vesicles and show aberrant patterns of OP marker-gene expression, but have normal hindbrain expression patterns. The authors conclude that FGF3 and FGF10 signals from the neuroectoderm and mesenchyme, respectively, act directly on the ectoderm to bring about OP gene expression. Notably, FGF signalling was not required for ectodermal proliferation or survival.

CNS development: an engulfing story

Gene inactivation by homologous recombination is a relatively new tool to become available to the fly community, which Sears et al. have put to good use on p. 3557 to inactivate Pvr, which encodes a receptor tyrosine kinase of the PDGF/VEGF family and is required for hemocyte/macrophage migration. By examining loss-of-function Pvr mutants, created by both gene targeting and chemical mutagenesis, the authors have discovered that Pvr is required for fly CNS morphogenesis – in its absence, axon scaffold formation and glial FSGP positioning defects occur in the CNS. By studying two other fly mutants with similar CNS defects – serpent, which lacks hemocytes, and flies mutant for the macrophage scavenger receptor, Croquemort – the authors conclude that the CNS defects of Pvr mutants are caused by the failure of macrophages to engulf cell corpses within the CNS, leading to disrupted glial and axon positioning.

How the zebrafish gets its stripes

Colour patterning is incredibly important in the animal world – it can influence everything from survival to mate choice.

With its growing collection of pigment-patterning mutants, the zebrafish is fast becoming a popular organism in which to study this important process. On p. 3447, Maderspacher and Nüsslein-Volhard analysed four such mutants to investigate stripe formation in zebrafish. In one experiment, they transplanted wild-type cells into mutant embryos lacking one of the two cell types that form stripes: melanophores or xanthophores. Their results show that the juxtaposition of these cell types is both necessary and sufficient to form stripes, as stripes were rescued in tissue patches where both cells were present. From their findings, the authors propose that pigment cell–cell interactions are the driving force behind the formation of the zebrafish’s stripes, so ruling out the possibility of prepatterning.

Screening border cell migration

Border cell migration in the Drosophila ovary is an ideal system for studying cell migration in vivo. These somatic cells delaminate from the epithelium that surrounds the germline and migrate through the nurse cells of the ovary to the anterior end of the oocyte. Three signalling pathways are probably involved: edysone, acting through its receptor Taiman (Tai), possibly regulates the timing of border cell migration; while PVF1 (a VEGF/PDGF receptor ligand) and Gurken (an EGF receptor ligand), possibly act as guidance cues. In a screen for genes involved in this process (see p. 3469), McDonald et al. identified Pvf1, and tested its ability, and that of other factors including Gurken, to guide border cells to new targets – only PVF1 was able to do so. Both Tai and PVF1 were found to regulate E-cadherin localization in border cells, possibly accounting for the interaction between these pathways.