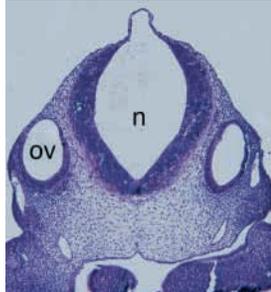


In this issue

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 FGFs 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.



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.

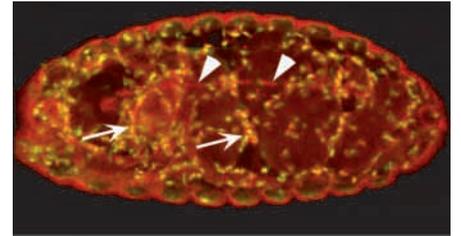
Screening border cell migration

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: ecdysone, 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.



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



mispositioning 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.

Chordin: new role in pharyngeal development

Chordin (*Chrd*) is a BMP antagonist that has well-characterised roles in vertebrate dorsoventral patterning. Now Bachiller et al. report that *Chrd* is also required for vertebrate head and neck patterning, as revealed by its inactivation in mice (see p. 3567). Surprisingly, many of the defects in *Chrd*^{-/-} mice – such as those affecting the pharyngeal arches, palate, parathyroid gland and heart – are characteristic of DiGeorge syndrome (DGS), despite the fact that *Chrd* maps outside of the microdeletion that causes this human disease. Clues to its involvement, however, came from studies of *Tbx1* and *Fgf8*, which are implicated in the aetiology of DGS: both are downregulated in *Chrd*^{-/-} pharyngeal tissue and can be induced in frog explants by *Chrd* mRNA. These findings thus uncover a role for *Chrd* in regulating *Tbx1* and possibly other factors important for pharyngeal development. Whether it is involved in other human head and neck congenital abnormalities awaits further study.

In *Journal of Cell Science*

Epithelial polarity – Stardust memories

The tight junctions (TJs) that link neighbouring epithelial cells and demarcate their apical and basolateral membranes play a crucial role in establishing epithelial cell polarity – as do signalling complexes containing the PDZ-domain proteins Par6 and Par3/ASIP, which regulate TJ assembly. Roh et al. now reveal that the components of another TJ-associated complex are important in this process: Crumbs 3 (CRB3) and its partner, the PDZ-domain protein Pals1 (mammalian homologue of the fly protein Stardust). They show that CRB3 overexpression disrupts TJ assembly and apicobasal polarity in MDCK epithelial cells: this requires the C terminus of CRB3, which interacts with the Pals1 PDZ domain. These and other results reported in this study highlight the importance of TJs and of the Pals1-CRB3 interaction in establishing mammalian epithelial cell polarity.

Roh, M. H. et al. (2003). The Crumbs3-Pals1 complex participates in the establishment of polarity in mammalian epithelial cells. *J. Cell Sci.* **116**, 2895-2906.