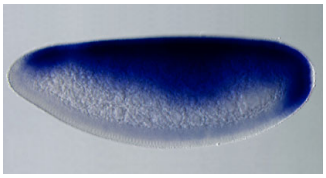
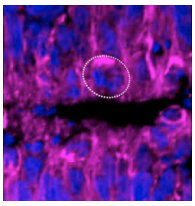


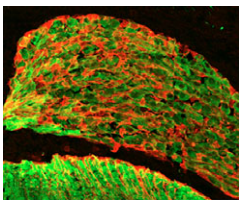
Dorsal mediates divergence

Despite the evolutionary distance between fruit flies and mosquitoes, similarities in their embryonic patterning do exist. However, differences in the morphology of their extra-embryonic membranes (EMs) have evolved: whereas *Drosophila* has one EM (the amnioserosa), the malaria mosquito, *Anopheles gambiae*, has distinct amnion and serosa tissues. Yury Goltsev and colleagues (see p. 2415) now show that changes in the expression of the dorsal gene network underlie morphological changes in the EMs of these Diptera. Specifically, the occurrence of two EMs in *A. gambiae* correlates with a broader domain of Decapentaplegic (Dpp) signalling. Studies of dorsal-ventral patterning gene expression profiles in *A. gambiae* reveal that this expanded Dpp domain arises from the restricted expression of the Dpp inhibitor, Sog. High-affinity Dorsal-binding sites in the *Drosophila sog* enhancer drives broad *sog* expression. By contrast, the mosquito *sog* enhancer contains low-affinity Dorsal-binding sites, which can restrict *sog* expression in the *Drosophila* mesoderm. Subtle changes in the Dorsal patterning network have thus altered the morphology of a single EM to form two.



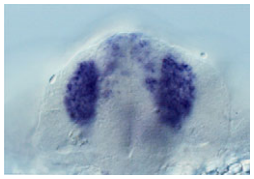
In transitin with Numb

Neurons and glia are generated in the developing CNS following asymmetric divisions of neural stem cells in the neuroepithelium – the cleavage plane is reportedly important for the localisation of cell-fate determinants. On p. 2425, Wakamatsu et al. report that in chicks, transitin, an intermediate filament protein, physically interacts with Numb, a cell-fate determinant, anchoring it to the cell cortex of mitotic neuroepithelial (NE) cells. Using biochemical, overexpression and RNAi-knockdown assays, these researchers show that transitin and Numb physically interact in the chick NE cell cortex. Cell-tracing experiments reveal that the lateral movement of this basally located transitin-Numb complex – which occurs by an unknown mechanism – asymmetrically localises Numb to one daughter cell, even when the cleavage plane is perpendicular to the ventricular surface. Interestingly, transitin gene knockdown in NE cells reduces the levels of basally located Numb and also promotes cell differentiation. Future work will probably focus on the mechanism by which transitin mediates both cellular differentiation and the lateral movement of Numb.



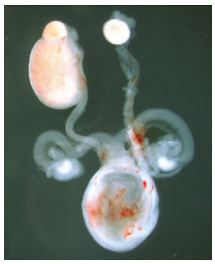
Notching up gliogenesis

Constitutive Notch (N) overexpression promotes gliogenesis, but does this reflect the true physiological role of N signalling? To answer this question, Sean Morrison's group conditionally deleted *Rbpsuh* – which encodes the DNA-binding protein RBP/J and is required for canonical signalling by all N receptors – in the mouse central (CNS) and peripheral (PNS) nervous systems. Their results on p. 2435 show that N signalling regulates gliogenesis independently of its role in neural progenitor maintenance. The conditional deletion of *Rbpsuh* in neural crest stem cells and in neuroectodermal cells of the developing CNS causes a near complete loss of gliogenesis, despite neurogenesis occurring almost as normal. These defects, the authors show, are not due to the premature depletion of neural progenitors. *Rbpsuh*, the authors report, is also required to maintain *Sox9* (a glial specification gene) expression in spinal cord progenitors, demonstrating that N signalling acts in glial specification. Together, these findings reveal a reiterative role for N signalling in neurogenesis; initially promoting progenitor maintenance and later promoting gliogenesis.



Bifunctional chemokine signalling

Cell positioning and axon pathfinding establish neural circuits in the developing nervous system. Previous fate-mapping studies in zebrafish have revealed that olfactory sensory neuron (OSN) precursors along the anterior neural plate converge – via the action of unknown factors – to form the olfactory placode. Miyasaka et al. now report on p. 2459 that, in zebrafish, Cxcl12/Cxcr4 chemokine signalling mediates both the olfactory placode assembly and the correct projection of differentiated OSN axons to the olfactory bulb. In *odysseus* (*ody*) zebrafish mutants (in which Cxcr4b receptor function is lost), olfactory placode assembly is perturbed, olfactory neurons displace ventrally and OSN axons fail to exit the placode, and instead accumulate near the placode-telencephalon border. The misexpression of the Cxcl12 ligand (ubiquitously and in mosaics) in zebrafish embryos also perturbs placode formation. The role of Cxcr4 signalling in zebrafish has some similarities to its role in mice, where it regulates motor axon projections. Perhaps, the authors suggest, Cxcl12/Cxcr4 chemokine signalling creates a favourable environment for initial axonal trajectories in vertebrates.



Kidney development: an invasion of space

Reciprocal interactions between the ureteric bud (UB) and the surrounding metanephric mesenchyme (MM) are crucial for kidney induction and development. Louis Reichardt's group have previously shown that, in the absence of α8β1 integrin (*Itga8*) expression in the MM, the UB fails to invade the surrounding MM – the first step in kidney induction – causing kidney agenesis. Now, this group identifies nephronectin (Npnt) as α8β1 integrin's essential ligand. Npnt is expressed in the UB and mediates α8β1 integrin activity during early mouse kidney development (see p. 2501). The expression of glial cell line-derived growth factor (GDNF) – an important growth factor in kidney development – in the MM, they show, is stimulated in response to Npnt/α8β1 integrin activity. Using *Npnt*-null and *Itga8*-null mice, these researchers show that, in both strains, UBs form but fail to invade the MM, as *Gdnf* expression is reduced. However, their other findings reveal that additional factors may contribute to normal *Gdnf* expression patterns during kidney development. One such factor is revealed in a study by Rolf Zeller's group on p. 2397, who report that gremlin 1 (*Grem1*), a BMP antagonist, is upregulated in the mouse MM prior to UB outgrowth. The resultant reduction in Bmp4 activity establishes the epithelial-mesenchymal (e-m) Gdnf/Wnt11 autoregulatory feedback loop that is essential for the UB's initial outgrowth and branching. Moreover, they show that failed UB outgrowth and secondary branching can be rescued in cultured *gremlin* mutant kidney primordia by recombinant gremlin. *Wnt11* expression at the epithelial tips and *Gdnf* expression in the MM is also restored by gremlin in these cultures. The regulation of Bmp4 activity by gremlin, and the subsequent establishment of the e-m feedback loop, provides insight into the mechanisms that mediate the UB's invasion of the MM, the important first step in kidney development. Further insights in kidney development are also reported by Andrew McMahon's group on p. 2533, who reveal that, at a later stage of kidney development, Wnt9b and Wnt4 signalling act via carefully modulated β-catenin-mediated canonical signalling during renal vesicle induction and mesenchymal-epithelial transition.