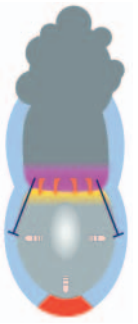
In this issue

By Jane Bradbury

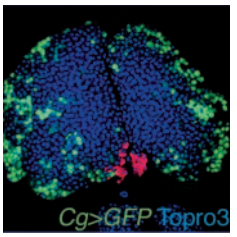
Patterning embryos from the outside in

The anteroposterior (AP) axis in mammalian embryos is established by interactions between the embryonic and extra-embryonic tissues; in particular, the extra-embryonic anterior visceral endoderm (AVE) is needed for anterior patterning in mice. However, little is known about what induces AVE formation at the distal tip of the mouse embryo or what directs its migration to the embryo's anterior. Rodriguez and colleagues now report that these processes are regulated by the extra-embryonic ectoderm (ExE; see p. 2513). By using microsurgery, grafting and video imaging, they show that the ExE restricts AVE induction to the distal tip of the mouse embryo and is required to initiate AVE migration to the prospective anterior of the embryo. The ExE also induces mesoderm markers in the posterior epiblast. Thus, the ExE has a critical role in AP specification in the mouse by patterning both extra-embryonic and embryonic tissues.



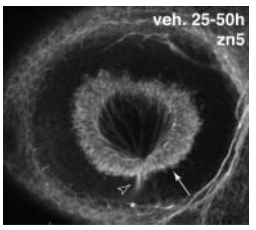
A fly's view of hematopoiesis

The development of the blood cells (haemocytes) of adult *Drosophila* occurs during larval development in a specialised organ called the lymph gland, but how haematopoiesis in flies is spatially and temporally regulated is poorly understood. On p. 2521, Jung and co-workers remedy this by analysing the structure of the lymph gland and the expression of haematopoietic and pro-haemocytic markers within the gland. They describe two previously unrecognised zones in the larval lymph gland: the medullary zone, which contains quiescent, immature haemocytes; and the cortical zone, which contains proliferating haemocytes that express maturation markers. This finding indicates that in *Drosophila*, as in vertebrates, quiescent multipotent blood precursors give rise to various mature blood cells. Additional similarities between vertebrate and fly haematopoiesis, together with the researchers' detailed model for hemocyte maturation in the lymph gland, establish *Drosophila* as a genetic model for the study of haematopoiesis.



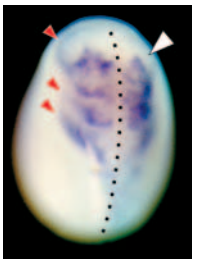
Neurogenic timing: a cell-intrinsic matter?

During nervous system development, regulating when progenitor cells stop proliferating and start differentiating produces the correct number of neurons. But is neurogenesis triggered by cell-extrinsic or cell-intrinsic signals? In the developing zebrafish retina it may be primarily the latter, report Kay and colleagues (see p. 2573). In *Drosophila* eye development, signals from newly differentiated neurons trigger neurogenesis in adjacent progenitors, and researchers have proposed that a similar sequential induction drives retinal neurogenesis in vertebrates. By manipulating the environment of developing retinoblasts, Kay et al show instead that temporally staggered, cell-intrinsic expression of the proneural gene *atonal-homologue 5* (*ath5*) is sufficient to support ganglion cell neurogenesis in the zebrafish retina. Thus, cell-intrinsic factors alone can trigger retinal neurogenesis. However, note the researchers, midline-derived Sonic hedgehog signals are part of the mechanism that sets the neurogenic timer earlier in zebrafish development.



Non-canonical signals for neural crest migration

During embryogenesis, neural crest cells migrate along specific routes to their final destinations, where they differentiate into several cell types. On p. 2587, De Calisto et al. now report that non-canonical Wnt signalling through the planar cell polarity or the Wnt-Ca^{2+} pathway is essential for the migration of these cells in *Xenopus* embryos. Their grafting and in vitro experiments with embryos carrying *Dishevelled* (*Dsh*) mutations show that non-canonical Wnt signalling controls neural crest migration; Dsh is a key component of both non-canonical Wnt signalling pathways. Other experiments uncover an essential role for the non-canonical Wnt ligand Wnt11 in this process. Finally, time-lapse analysis demonstrates that non-canonical Wnt signalling controls neural crest migration in vitro by stabilising the protrusions of migrating cells. The researchers propose that, as in mesoderm migration during vertebrate gastrulation, non-canonical Wnt signalling controls cytoskeletal behaviour or cell-adhesion properties during neural crest migration.



Cleaving Bmps with tissue specific proteases

In *Drosophila* embryos, Tolloid (Tld) – an extracellular protease related to bone morphogenetic protein 1 (Bmp1) – helps to specify dorsal structures by cleaving the Bmp inhibitor Short gastrulation (Sog) to release Decapentaplegic from an inhibitory complex. On p. 2645, Serpe and colleagues report that, during posterior crossvein (PCV) formation in *Drosophila* wings, the embryonic role of Tld in spatially restricting Bmp signalling is recapitulated by Tolloid-related (Tlr). The researchers show that, like Tld, Tlr cleaves Sog but with slightly different kinetics and that *tlr* mutants lack the PCV probably through excess Sog activity reducing Bmp signalling. However, other results indicate that, as in the embryo, Sog has both negative and positive effects on Bmp signalling in the wing. Finally, because Tld and Tlr cannot substitute for each other during development, the researchers propose that their different Sog catalytic properties match them to dorsal structure and PCV specification, respectively.

Heartfelt networks

The vertebrate heart is assembled from precursor cells in the primary and the secondary (anterior) heart field in a complex process that involves numerous transcription factors. Phan and co-workers now describe how myocyte enhancer factor 2C (MEF2C) and the transcriptional repressor BOP are involved in cardiac development (see p. 2669). Mice embryos lacking MEF2C or BOP develop malformed right ventricles and outflow tracts, which implicates these transcription factors in anterior heart field development. The researchers show that *Bop* expression in the developing heart is downregulated in *Mef2c* mutant embryos and identify a MEF2C-response element in the *Bop* promoter that controls *Bop* expression in the anterior heart field. The researchers propose a network of transcription factors involved in ventricular development that, together with other recently described transcription factor networks in the heart (see *Development* 132, part 10), provides important insights into the aetiology of human congenital heart defects.