In this issue

By Jane Bradbury

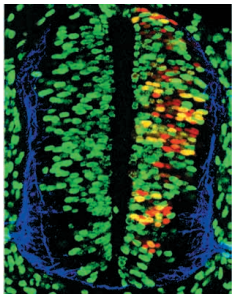
Meiosis checked out in plants

Unlike animals, plants have no primordial germline. Instead, specialised meiotic cells (sporocytes), which are required for sexual reproduction, are derived de novo from subepidermal cells in the anthers and ovules. Sporocytes then undergo meiosis to form multicellular haploid gametophytes, but little is known about the control of meiotic progression in plants. Now, Reddy et al. identify *DUET*, a gene that is required for chromosome organisation and meiotic progression in *Arabidopsis*, and which encodes a putative plant homeo domain (PHD) finger protein (see p. 5975). They describe how the *duet* mutation causes male sterility, defective chromosome organisation and the arrest of male meiocytes at metaphase I. The *duet* mutation interacts genetically with the *dyad* mutation to produce very strong defects in male meiosis. *DYAD* is a gene required for female meiotic progression, and for chromosome cohesion during male and female meiosis. Further analysis of meiosis defective mutants should, say the researchers, provide information on chromosomal checkpoints in plants and indicate how these checkpoints compare with those in animals.

Putting a lid on the brain

The mammalian skull vault develops from neural crest and mesodermal-derived mesenchymal cells that migrate over the cerebrum before proliferating and differentiating along an osteogenic pathway. On p. 6131, Ishii et al. report that the basic helix-loop-helix gene *Twist* and the homeobox gene *Msx2* – mutations in which cause craniosynostosis (premature fusion of the skull bones) and calvarial foramina (holes in the skull vault) in humans – cooperatively control the differentiation and proliferation of skeletogenic mesenchyme in mice. They show that the calvarial foramen defect in *Msx2* homozygous mutant mice is caused by fewer neural crest cells committing to osteogenesis, followed by a reduction in their proliferation. By analysing skull vault defects in *Msx2-Twist* double mutants, the researchers conclude that *Msx2* and *Twist* act in parallel to direct the correct patterning of the frontal bone.

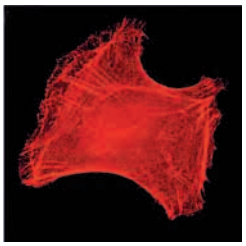
Ebf genes find their place in neurogenesis



Neurogenesis requires the coordination of numerous events, including the commitment of cells to specific fates, cell-cycle exit, migration and differentiation. In the mouse and frog, helix-loop-helix transcription factors of the Ebf family have been implicated in neurogenesis but their precise roles are unknown. On p. 6013, Garcia-Dominguez et al. describe a functional analysis of the chick orthologs of mouse Ebf1 and Ebf3. Electroporation of a dominant-negative form of Ebf1 into the chick neural tube shows that Ebf is required for neuronal differentiation and migration, but not for cell-cycle exit, indicating that Ebf genes couple cell-cycle exit with neuronal differentiation and migration. Additional experiments indicate that Ebf genes are also involved in the stabilisation of the committed state, and in neuronal subtype specification. The researchers conclude that Ebf genes lie downstream of proneural gene expression and cell-cycle exit, but upstream of neuronal differentiation and migration in central nervous system neurogenesis in higher vertebrates.

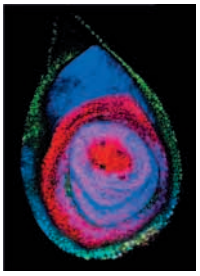
MCSP keeps epidermal stem cells together

Stem cells within the human interfollicular epidermis occur in clusters but the molecular basis for this distribution is poorly understood. Legg et al. propose that melanoma chondroitin sulphate proteoglycan (MCSP), a new stem-cell marker, contributes to the spatial organisation of epidermal stem cells by regulating their adhesive properties (see p. 6049). The researchers show that expression of MCSP, a cell-surface proteoglycan, is restricted to those cells in the interfollicular epidermis that express the highest amounts of β1 integrin. These cells are non-cycling in vivo but form self-renewing clones in vitro, characteristics of epidermal stem cells. Interference with endogenous MCSP function by expression of a CD8/MCSP chimera, consisting of the extracellular domain of CD8 and the cytoplasmic domain of MCSP, did not affect the proliferation or differentiation of keratinocytes, but reduced their cohesiveness. Legg et al. conclude that MCSP is functionally important in promoting epidermal stem-cell clustering.



Giving flies a leg up

On p. 5929 Estella et al. shed new light on how the morphological distinction between ventral and dorsal structures is achieved during development. In *Drosophila*, legs and antennae develop from the ventral imaginal discs, and Estella and co-workers report that the product of a single gene called *buttonhead* (*btd*) can trigger the entire genetic network needed for leg and antennal development. RNAi downregulation of *btd*, which encodes a zinc-finger transcription factor, together with an adjacent related transcription factor, *Sp1*, resulted in underdeveloped legs and antennae. Conversely, when *btd* was ectopically expressed in dorsal imaginal discs, the normal products of these discs – eyes, wings and halteres – were replaced by antennae and legs. This transformation involved the de novo activation of the *engrailed/hedgehog/wingless/decapentaplegic* cascade responsible for the growth and pattern of ventral imaginal discs.



Be still my beating heart

Changes in functional demand can alter the morphology of the adult heart; for example, pressure overload can induce ventricular hypertrophy. Now, two groups of researchers provide evidence that blood flow also affects cardiac morphology during embryogenesis. Berdougo et al. have investigated the effect of a mutation in the gene *atrial myosin heavy chain* on heart development in zebrafish (see p. 6121). Although this gene is expressed only in the atrium, mutations in it result in not only atrial contractile defects but also ventricular morphological defects. Similarly, Huang et al. report that inactivation of the gene for atrial myosin light chain 2 in mice causes severely diminished atrial contraction and secondary abnormalities in cardiac morphogenesis (see p. 6111). Both research teams conclude that alterations in blood flow caused by the mis-functioning of one embryonic heart chamber epigenetically influence the morphogenesis of the other chamber, and they discuss how these findings may provide clues to the causes of human congenital heart defects.