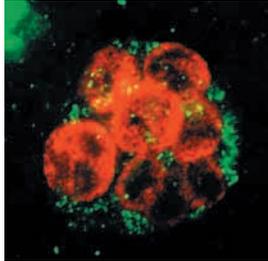


In this issue

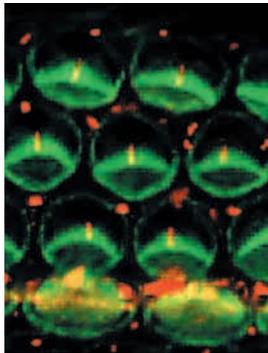
Fearing coalescence in gonad development

Two types of cellular movement underlie proper gonad formation in animals: individual cell migration, where primordial germ cells migrate from their site of origin to contact the cells of the somatic gonad; and coordinated tissue morphogenesis, where germ cells and somatic cells coalesce to form the embryonic gonad. Now Ruth Lehman and colleagues shed light on these little understood events with their phenotypic and molecular characterization of the *fear of intimacy (foi)* gene in *Drosophila* (see p. 2355). *foi* is required for gonad coalescence, but not for somatic gonad or germ cell identity. Its protein, which is predicted to belong to a novel family of conserved transmembrane proteins and is required in gonadal mesoderm for coalescence to occur, localises to the cell surface, where the authors propose that it might cooperate with E-cadherin in this morphogenetic process.



Wnts and planar polarity in mammals

Planar cell polarity – the process whereby cellular structures within the plane of an epithelium are oriented in the same direction – has been well studied in flies; studies that have revealed the role of non-canonical Wnt signalling in this process. Now Dabdoub et al. report the first study of planar polarity development in a mammalian system: the cochlea, in which stereociliary bundles on mechanosensory hair cells in the sensory epithelium must be unidirectionally oriented to ensure unimpaired hearing. Their findings, on p. 2375, show that several Wnts, particularly *Wnt7a*, are highly expressed in developing mouse cochlea. Moreover, the application of *Wnt7a*, or Wnt signalling inhibitors, to cultured cochlear explants causes disrupted bundle orientation, indicating that the molecular basis of planar polarity might have been conserved between vertebrates and invertebrates.



Regulating flowering across time

Flowers of some dicot plants, such as *Arabidopsis* and *Antirrhinum*, look very different from those of certain monocot species, such as the grasses – the grasses, for example, do not have sepal- or petal-like structures. So has the transcriptional network that regulates flower development been conserved since monocots and dicots diverged ~150 million years ago? To investigate this, John Doebley and co-workers used insertional mutagenesis to disrupt the *zfl1* and *zfl2* genes in maize, which are homologues of the *FLORICAULA (FLO)* and *LEAFY (LFY)* genes in *Antirrhinum* and *Arabidopsis*, respectively. *FLO* and *LFY* are regulators of the ABC floral organ identity genes, and Doebley and colleagues surprisingly found (see p. 2385) that *zfl1* and *zfl2* do indeed share conserved roles with these dicot counterparts (despite their evolutionary distance), as *zfl1/zfl2* double mutants have disrupted floral organ identity and fail to form normal reproductive structures.



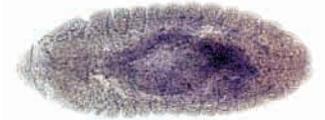
The many functions of Cyr61

The proper execution of gastrulation requires that cell signalling, cell adhesion and cell migration are all precisely coordinated and integrated. In their investigations into how this might occur, Latinkic et al. studied Cyr61, a CCN-family, secreted, extracellular matrix (ECM)-associated protein. CCN proteins are very versatile – they can mediate cell adhesion and migration, for example, and can induce signalling events – all expected features of candidate gastrulation regulators. The authors now report, on p. 2429, that Cyr61 is an important regulator of gastrulation movements: both its overexpression and inhibition disrupt gastrulation in frog embryos, perhaps because Cyr61 is required for the assembly of a fibronectin-rich ECM and because it regulates cell-cell and cell-matrix adhesion. Intriguingly, Xcyr61 also appears to both stimulate and inhibit Wnt signalling in a context-dependent manner. Future studies into the different domains of Cyr61 should reveal where its many activities reside.



Sphingolipids: emerging roles in development

Sphingolipids are complex membrane lipids, some of which, such as sphingosine-1-phosphate, act in signalling pathways that regulate cell death, survival, differentiation and migration in multicellular organisms. Herr et al. now report the identification and disruption, in *Drosophila*, of *Sply*, which encodes sphingosine-1-phosphate lyase, an enzyme that catalyses the catabolism of sphingosine-1-phosphate. Their findings, on p. 2443, reveal, for the first time, that disrupted sphingolipid catabolism can directly cause complex developmental abnormalities in a metazoan. *Sply*-null mutants have abnormal dorsal longitudinal muscles (and so are flightless), reduced egg laying and larval viability, and accumulate sphingoid bases. These defects were rescued by restoring *Sply* expression and by introducing a suppressor mutation that reduces sphingolipid synthesis and the accumulation of sphingolipid intermediates. Such findings are a first step towards elucidating the roles of these signalling molecules in development and cell function.



Spatially controlling translation

Although *glp-1* mRNA is abundantly expressed in all blastomeres of *C. elegans* embryos up to the eight-cell stage, its protein is present in only the anterior AB blastomeres of two- and four-cell embryos. Thus, its translation, which is required for gonad and embryonic development, is spatially and temporally controlled. This occurs via the spatial control (SCR) and the temporal control (TCR) regions of the 3' UTR of *glp-1*. Now Ogura et al. report, on p. 2495, that POS-1, which translationally regulates another maternal transcript, *apx-1*, represses *glp-1* translation by binding to its SCR. A yeast two-hybrid screen also identified a POS-1-interacting protein, SPN-4 (an RNP-type, RNA-binding protein), that binds to the TCR of *glp-1* and is required for its translation in anterior blastomeres. The authors propose that a balance between these two proteins controls *glp-1* translation. Given the pleiotropic phenotypes of *pos-1* and *spn-4* mutants, these proteins probably also regulate the translation of other maternal RNAs.

