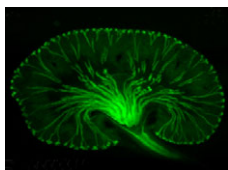


SUMO wrestling maize embryo sacs into shape

Reversible post-translational modification by small ubiquitin-related modifiers (SUMOs) regulates the activity and degradation of numerous proteins during various eukaryotic developmental processes. Now, Thomas Dresselhaus and colleagues describe the important role that a diSUMO-like protein called ZmDSUL plays in female gametophyte (embryo sac) development in maize (see p. 333). The researchers identify ZmDSUL by searching maize egg-cell-specific expressed sequence tag data for SUMO-related proteins. They show that DSUL, which has a unique structure consisting of two head-to-tail SUMO-like domains, has a specific expression pattern during maize embryo sac development and early embryogenesis. Finally, using RNAi silencing, they show that DSUL is required for female gametophyte viability and for the correct segregation and positioning of the nuclei that are produced by mitotic nuclear division in the developing embryo. Thus, they conclude, DSUL is essential for female gametophyte maturation in maize because it ensures the correct positioning of the nuclei, a prerequisite for cell specification (see also the review on female gametophyte development by Sundaresan and Alandete-Saez on p. 179).

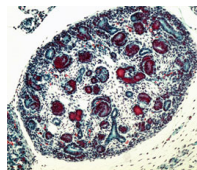


Getting renal with retinoic acid and vHNF1

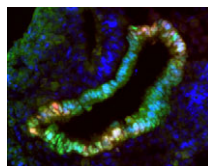
Kidney development involves reciprocal signalling between metanephric mesenchyme (which differentiates into the nephrons, the kidney's functional units), ureteric bud (UB) epithelium (which undergoes branching morphogenesis to form the urinary collecting ducts) and stromal mesenchyme (which differentiates into the renal interstitium). In this issue, two papers provide new information about how kidney development is regulated in mice.

On p. 283, Cathy Mendelsohn and colleagues describe a novel stromal-UB retinoic acid (RA) signalling pathway that is crucial for collecting duct formation. The receptor protein kinase Ret is expressed in the UB and is required for both bud formation and branching. Ret expression in the UB is controlled by RA receptors (RARs), widely expressed RA-activated transcription factors, but as RA is made in both UB and stromal cells, it has been unclear whether RA regulates Ret in an autocrine or paracrine fashion. Now, by expressing a dominant-negative RAR in mouse UB cells, the researchers show that UB RARs, rather than stromal cell RARs, are required for Ret transcription; other experiments indicate that RA from stromal, rather than from UB, cells activates UB RARs. This paracrine RA signalling pathway is probably conserved, suggest the researchers, and might be used elsewhere during development.

On p. 347, Silvia Cereghini and colleagues report that the homeodomain transcription factor vHNF1 is a key regulator of ureteric branching and early nephrogenesis. Heterozygous mutations or deletions in the gene encoding HNF1 cause 'Renal Cysts and Diabetes', a human syndrome that includes multiple kidney and genital tract abnormalities. Because *vHnf1* inactivation in mice causes early embryonic lethality, the researchers analysed tetraploid and diploid chimeras to investigate its role during early kidney development. Their experiments indicate that vHNF1 regulates several stages in renal development, including the timed outgrowth of the UB and its subsequent branching, nephric duct epithelial maintenance and early nephrogenesis. They also report that vHNF1 acts directly upstream of *Wnt9b* during the mesenchymal-epithelial transitions that underlie the initiation of nephrogenesis. Thus, they conclude, vHNF1 is a crucial component of the regulatory circuits that controls early urogenital development.

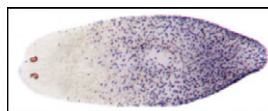


Together, these two papers shed new light on the complex signals and regulatory circuits involved in renal development.



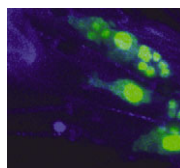
Islet cell development under Rfx6's wing

Each type of pancreatic islet cell produces a different hormone. For example, beta cells produce insulin, whereas alpha cells produce glucagon. In mouse embryos, the transcription factor neurogenin 3 (Ngn3) controls endocrine cell fate decisions in multipotent pancreatic progenitor cells, but how? On p. 203, Josselin Soyer and co-workers identify the winged helix transcription factor Rfx6 as a novel Ngn3-dependent regulator of islet cell development in mice and zebrafish. The researchers show that mouse Rfx6 and its zebrafish ortholog *rfx6* are expressed in islet progenitor cells and in all developing and adult islet cell types. Furthermore, loss-of-function experiments in zebrafish indicate that the differentiation of *glucagon-*, *ghrelin-* and *somatostatin-*expressing cells is blocked at the progenitor stage in the absence of Rfx6, whereas *insulin-*expressing cells differentiate normally but fail to form compact islets. This new information about the Ngn3-controlled genetic program during pancreas development might facilitate efforts to produce functional beta cells from human embryonic stem cells for the treatment of type 1 diabetes.



Worming into p53 evolution

The planarian *Schmidtea mediterranea* has extraordinary regenerative capacities. This long-lived non-parasitic flatworm actually reproduces by binary fission, followed by the replacement of missing body parts. The large populations of adult stem cells needed to achieve this feat make *S. mediterranea* an ideal model in which to study adult stem cell biology. Bret Pearson and Alejandro Sánchez Alvarado now report that the single planarian p53 homolog (Smed-P53) regulates proliferation and self-renewal in adult stem cell lineages in *S. mediterranea* (see p. 213). They show that Smed-p53 is predominantly expressed in newly made, postmitotic stem cell progeny and that RNAi knockdown of *Smed-p53* initially leads to stem cell hyperproliferation, which indicates that *Smed-p53* has a tumour suppressor function. However, ultimately, in the absence of *Smed-p53* expression, the stem cell population fails to self-renew. By showing that an ancestral p53-like molecule functions in both stem cell proliferation control and self-renewal, these results provide new insights into the evolution of the p53 family of transcription factors.



Nervy sexual dimorphisms

Many animals exhibit innate sexually dimorphic sexual behaviours. Male flies, for example, follow elaborate courtship rituals to secure a female mate. Nervous system sexual dimorphisms probably underlie many sex-specific behaviours, but how are these neuronal dimorphisms generated? On p. 323, David Mellert and colleagues report that one such dimorphism – midline crossing by gustatory (taste) receptor neuron (GRN) axons in male, but not female, *Drosophila* – is regulated by the sex determination genes *fruitless* (*fru*) and *doublesex* (*dsx*) and the Roundabout (Robo) axon guidance receptors. *fru* and *dsx* are regulated during pre-RNA splicing to produce male- and female-specific isoforms, with female-specific *fru* mRNA not being translated. The researchers show that, in male flies, male-specific Fru and Dsx promote midline crossing by foreleg GRN axons, whereas in female flies, female-specific Dsx represses midline crossing. They also show that male-specific Fru exerts its effect on midline crossing by directly or indirectly regulating Robo signalling. Together, these results begin to reveal how the neuronal connections that underpin *Drosophila* sex-specific behaviour are established.

Jane Bradbury