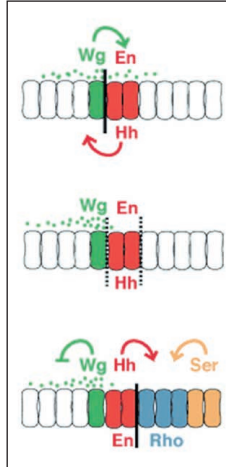


### Hedgehog dallies with segment polarity

Loss of Wingless or Hedgehog signalling in *Drosophila* results in the loss of both segment polarity and naked cuticle in the embryonic epidermis. Genetic studies have implicated the glypicans *dally* and *dally-like (dlp)*, cell-surface heparan sulphate proteoglycans, in Wingless signalling. Now Desbordes and Sanson report that neither glypican is necessary for Wingless signalling in the embryonic epidermis but that Dally-like is required for Hedgehog signalling (see p. 6245). They show that silencing of *dally-like* but not of *dally* by RNA interference produces the same segment polarity phenotype as *wingless* or *hedgehog* null mutations. In experiments in which they uncouple the normally tightly coupled Hedgehog and Wingless signalling pathways, the researchers reveal a specific requirement for Dally-like in the Hedgehog pathway. Finally, they report that Dally-like is required for reception of the Hedgehog signal, acting upstream or at the level of *patched*, which encodes the Hedgehog receptor.



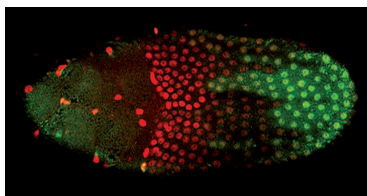
### Genes and the evolution of complexity

Differences in the protein repertoires between two organisms should reflect differences in their anatomical and physiological complexities. On p. 6317, Vogel et al. analyse the immunoglobulin superfamily (IgSF) genes, which encode proteins involved in cell-cell recognition and communication, in *Caenorhabditis elegans* and *Drosophila melanogaster*. They identify 142 IgSF proteins in the fly and 80 in the worm, and show that three-quarters of the fly's IgSF repertoire and one half of the worm's repertoire have emerged since their evolutionary divergence. The fly genome encodes fewer proteins in total than that of the worm, and the researchers suggest that the expansion of particular protein families, including the immunoglobulin superfamily, has contributed to the evolution of the more complex physiology of the fly.

### Cracking open the *Drosophila* eggshell

Late in *Drosophila* oogenesis, a flat epithelium consisting of somatically derived follicle cells is remodelled into two tubes that secrete proteins that form the long tubular dorsal appendages (DAs), the specialised respiratory structures of the eggshell. On p. 6273, Tran and Berg describe how *bullwinkle (bwk)*, a germline-expressed transcription factor, and *shark*, a newly identified enhancer of *bwk* expressed in somatic squamous stretch cells, regulate DA morphogenesis. *shark*, which encodes a non-receptor tyrosine kinase, was identified as an enhancer of *bwk* in a genetic-modifier screen. The researchers then used loss- and gain-of-function strategies to show that *shark* expression in the stretch cells requires *bwk* activity and that *shark* is required in the follicle cells for migration of the DA cells.

The researchers conclude that *shark* plays an important downstream role in the *bwk*-signalling pathway and suggest that this pathway may be conserved in vertebrates.

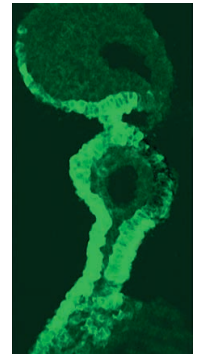


### Epimutation in plant culture

Cultured plant cells show phenotypic variation, some of which results from epigenetic changes – mitotically heritable but reversible alterations in gene expression that do not result from permanent genetic modifications. Some epigenetic changes are also transmitted during meiosis; these are called epimutations, and Meins and Thomas now report that epimutations can arise frequently in cultured tobacco plant cells (see p. 6201). Leaf cells normally require the cell-division factor cytokinin for continuous growth in culture – they have a C<sup>-</sup> phenotype. However, when cultured in media containing cytokinin, some of these cells rapidly alternate between a C<sup>-</sup> and a C<sup>+</sup> (cytokinin-independent) state, a phenomenon called pseudodirected variation. Meins and Thomas show that in plants regenerated from most C<sup>+</sup> clones, leaf tissues retained the C<sup>+</sup> phenotype. This trait was meiotically transmitted but rapidly reverted to the C<sup>-</sup> phenotype during successive sexual generations, leading the researchers to conclude that pseudodirected variation is a new form of epimutation.

### FGF8 signalling in development and disease

Mice in which FGF8 expression is globally reduced throughout development have severe cardiovascular and pharyngeal defects at birth, similar to those seen in human 22q11 deletion syndromes such as Di George syndrome. On p. 6361, Macatee et al. suggest that these defects result from a failure in local FGF8 signalling from specific domains of epithelial cells in the pharyngeal arches to the mesenchymal cells that populate and migrate through the arches. To test their hypothesis, the researchers generated *Fgf8* conditional alleles and Cre recombinase-expressing drivers designed to ablate FGF8 in different spatiotemporal domains. Ablation of ectodermal FGF8 expression caused defects in aortic arch and coronary vessel formation, whereas ablation of expression in the pharyngeal ectoderm and endoderm caused glandular and valvar defects. These results begin to reveal how local disruptions in FGF8 signalling can produce a spectrum of birth defects.



### In *Journal of Cell Science*

#### A fateful change in cell-cycle length

Neuroepithelial cells are the progenitors for all the neurons in the mammalian central nervous system. At the onset of neurogenesis, the G1 phase of the cell cycle in neuroepithelial cells lengthens, but is this a cause or an effect of neurogenesis? Calegari and Huttner treated 9.5-day-old mouse embryos in culture with olomoucine, which inhibits cyclin-dependent kinases and lengthens the G1 phase of the cell cycle. In these cultures, TIS21, a marker for neuroepithelial cells that have switched from proliferative to neuron-generating divisions, was expressed prematurely and neurons were made earlier than expected. The only observable effect of olomoucine in these cultures was a lengthening of the cell cycle by about 2 hours, so the researchers conclude that this change is sufficient to induce neuroepithelial cell differentiation. They therefore propose a model whereby cell-cycle length can be linked to the effects of cell-fate determinants.

Calegari, F. and Huttner, W. B. (2003). An inhibition of cyclin-dependent kinases that lengthens, but does not arrest, neuroepithelial cell cycle induces premature neurogenesis. *J. Cell Sci.* **116**, 4947-4955.