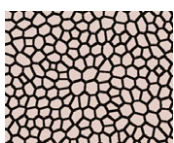


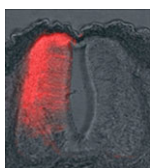
Gata-way to trophoblast development

The first developmental decision in mouse embryos establishes the embryonic and the extraembryonic tissue lineages. The transcription factor *Cdx2* promotes the extraembryonic trophoblast fate but, suggest Amy Ralston and colleagues, it does not act alone. Rather, they report, the transcription factor *Gata3* regulates trophoblast development in parallel to *Cdx2* (see p. 395). The researchers identify *Gata3* as a trophoblast-specific gene by microarray analysis. Using functional genomic strategies, they show that *Gata3* induces the trophoblast fate in embryonic stem cells and drives the differentiation of trophoblast stem cells. Although *Gata3* is coexpressed with *Cdx2* in the blastocyst, *Gata3* expression does not depend on *Cdx2*. Instead, the expression of both *Gata3* and *Cdx2* depends on the expression of *Tead4*, a third transcription factor. Thus, the researchers suggest, *Gata3* and *Cdx2* act in parallel pathways downstream of *Tead4* to induce the expression of common and independent targets in the trophoblast lineage. The characterisation of these targets should provide additional insights into early lineage restriction during embryogenesis.



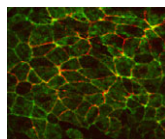
Pack 'em in: establishing epithelial topology

Epithelia consist of tightly packed cells in which the apical cell surfaces resemble polygons with different numbers of neighbouring cells. But how is this topology, which ensures the structural integrity of epithelia, established during development? On p. 499, Tinri Aegerter-Wilmsen and colleagues propose that growth regulation by mechanical stress might help to establish epithelial topology. Previously, it has been proposed that cell division alone, or cell division plus cell rearrangements, is sufficient to explain epithelial topology. Now, the researchers formulate two variant models in which cellular growth rates are dependent on mechanical force. A comparison of the predictions of these models with experimental data on the clustering and the polygon distribution of mitotic cells in the *Drosophila* wing imaginal disc shows that only the models that include mechanical feedback fit the experimental data. These models also show a reduction in cell rearrangements and eliminations during epithelial growth. Thus, growth regulation by mechanical stresses could help to ensure the structural integrity of growing epithelial tissues.



Sensing one's place with retinoic acid

During vertebrate development, retinoic acid (RA) signalling helps to shape the neural tube and consequently the central nervous system. Although several retinaldehyde dehydrogenases can synthesize RA from vitamin A, the most important of these during development is *Raldh2*. Now, José Xavier-Neto and colleagues identify a conserved intronic enhancer that drives *raldh2* expression in the roof plate and dorsal-most (dl1) interneurons of frog, mouse and chicken embryos (see p. 507). This enhancer is activated dorsally through Tcf- and Cdx-homeodomain binding sites, they report, and repressed ventrally through Tgif-homeobox and Lim-homeodomain binding sites. Other experiments reveal a novel transient expression domain for *raldh2* in dl1 interneurons, which suggests that RA signalling regulates the development of spinocerebellar and intraspinal proprioceptive (movement perception) circuits. Finally, the researchers report that *raldh2* is also expressed in dorsal spinal cord interneurons in lamprey embryos. Because the lamprey is an agnathan, an ancient order of vertebrates, this observation reveals ancestral roles for RA signalling during the development of intraspinal proprioception.



Focus on induction and morphogenesis

Morphogenesis is important throughout embryogenesis, but the mechanisms that underpin it are poorly understood. On p. 405, Richard Lang and co-workers partly remedy this situation by reporting that the expression of the actin-binding protein *Shroom3* regulates apical constriction (AC; a cellular shape change from cylindrical to conical) during lens placode invagination in mice. Several types of epithelial cells undergo AC during embryonic development, and *Shroom3* has previously been associated with AC during neural plate morphogenesis in mouse and frog embryos. Lang and colleagues now show that, during lens placode invagination, *Shroom3* is required for the apical localisation of F-actin and myosin II, both of which are required for AC, and for the apical localisation of Vasp, another protein involved in actin dynamics. The researchers also show that *Shroom3* expression is dependent on *Pax6*, a transcription factor that is required for lens placode induction. Together, these results provide new insights into the mechanisms of epithelial morphogenesis and reveal a link between lens induction and lens morphogenesis.



CHALLAH stems stomatal production

In *Arabidopsis*, plants mutant for the receptor-like protein *TOO MANY MOUTHS (TMM)* produce excess stomata (tiny epidermal valves that regulate gas exchange) in leaves, but none at all in stems. What controls this tissue-specific difference? Now, on p. 447, Emily Abrash and Dominique Bergmann report that the putative stomatal ligand *CHALLAH (CHAL)* regulates stomatal production in stems. Through a *tmm* suppressor screen, the authors identified *CHAL* as a recessive repressor of the *tmm* stem phenotype. Ubiquitous *CHAL* overexpression, they report, reduces stomatal production, but its normal expression and repressive function are limited to certain tissues (including stems). *CHAL* is related to the universal stomatal regulators *EPF1* and *EPF2*, and all three probably bind to *ERECTA* family receptors. Unlike these *EPF* proteins, however, *CHAL* signalling is not enhanced by *TMM*; instead, *TMM* appears to dampen the stomatal-inhibitory effects of *CHAL*. This, the researchers suggest, might indicate that the *CHAL/EPF* superfamily has diverged into both universal and tissue-specific regulators, a process that could also have occurred in other developmental contexts.

Jane Bradbury

IN JOURNAL OF CELL SCIENCE

Wee1: preventing polarity

Wee1 is a well-characterised cell-cycle checkpoint kinase. Now, Andreas Püschel and colleagues uncover a novel role for *Wee1* in establishing neuronal polarity regulated by the kinases *SadA* and *SadB* (*SadA/B*). They show that *Wee1* is expressed early during hippocampal development, when neurons have several undifferentiated neurites, but that its expression declines as the cells become polarised and form a single axon. Maintaining high *Wee1* expression throughout differentiation disrupts neuronal polarisation: instead of a single axon, neurons form multiple neurites expressing axonal, and sometimes also dendritic, markers. In contrast to *Wee1*, the expression of *SadA/B* (known to be involved in axon formation) increases during neuronal differentiation. *SadA* physically interacts with *Wee1* and mediates an inhibitory phosphorylation event necessary for the decrease in *Wee1* expression. Accordingly, developing neurons from *Sada^{-/-} Sadb^{-/-}* mouse embryos continue to express *Wee1* and develop abnormally. Therefore, the authors conclude, *SadA/B* regulate *Wee1* activity and expression during neuronal differentiation and thus control proper neuronal cell polarity.

Müller, M. et al. (2010). Persistence of the cell cycle checkpoint kinase *Wee1* in *SadA/B*-deficient neurons disrupts neuronal polarity. *J. Cell Sci.* 123, 286-294.