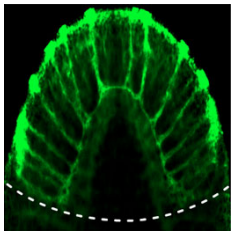
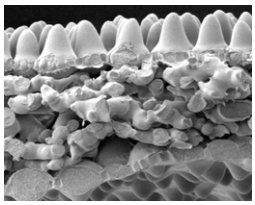


Endocytosis regenerated

The regenerative ability of planarians is truly remarkable – they can regenerate their entire body from a tiny tissue fragment. Of particular interest is how the brain regenerates. On p. 1679, Agata and colleagues now show that the planarian *clathrin heavy chain* (*DjCHC*) gene, which functions in endocytosis, is required for neurite extension and maintenance during regeneration but not for neuronal differentiation. They used a novel in vitro cell culture system in which primary cultures of planarian neurons from regenerated heads were sorted (according to neuronal marker expression) by fluorescence-activated cell sorting (FACS), following the RNAi knockdown of genes that are expressed in the regenerating CNS. This in vitro assay revealed that neurite extension but not neuronal differentiation depends on *DjCHC*. In uncut planarians, the patterning and differentiation of neural cells is normal despite the RNAi knockdown of *DjCHC*; however, neurites subsequently regress and neural cells die, resulting in an atrophied CNS. This surprising link between endocytosis and CNS regeneration will be of interest to both neurobiologists and investigators of regeneration.

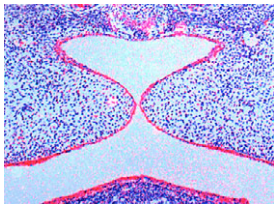


The shape of attraction

Petal colour intensity is an adaptation that flowering plants have made to lure pollinating insects. A conical-shaped petal epidermal cell has increased colour intensity because more incidental light is reflected into it. MIXTA, a MYB-related transcription factor, regulates conical cell shape development in snapdragon (*Antirrhinum majus*) petals. Cathie Martin and co-workers report that proteins closely related to MIXTA from both *A. majus* and other angiosperms have similar, but distinct, roles in petal epidermal cell shape formation (p. 1691). Cell shape contributes to the overall petal curvature, and thus flower shape, a role not previously associated with MIXTA-like proteins. Using genetic mutants from snapdragon and petunia (*Petunia hybrida*), the authors reveal the occurrence of flatter epidermal cells in *mixta* mutants and of shallow, conically shaped cells in petunia *phmyb1* mutants. They propose that an altered direction of growth, followed by growth extension, generates cell shape in this setting. This family of transcription factors, in modifying the petal epidermal cell shape, fulfil a crucial role in attracting insects, thus ensuring pollination.

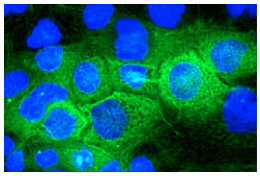
*Gli*mpsing the inner ear

The vertebrate inner ear has two main components: the dorsal vestibular structures (responsible for balance) and the ventral cochlear duct (responsible for hearing). Sonic hedgehog (Shh) signalling, which emanates from the floor plate and notochord, directs the formation of these structures, but how it does this is unknown. Reciprocal gradients of Gli activator (GliA) and Gli repressor (GliR) activity are now shown by Doris Wu and co-workers to mediate these responses (p. 1713). The formation of the ventral-most otic region, the distal cochlear duct, depends on Gli2A/3A function, whereas that of the proximal cochlear duct and saccule depends on Gli3R antagonism (due to lower Shh signalling). What is particularly novel about Shh/Gli signalling in the inner ear is the dosage requirement for Gli3R in the dorsal ear; dorsal vestibular structures are rescued in $Shh^{-/-};Gli3^{+/-}$ embryos. These findings may have important implications for other developmental events in which Hedgehog signalling is crucial; for example, during dorsoventral cell specification in the spinal cord.



Palatal fusion at a Snail's pace

The role of Snail proteins in patterning vertebrate embryos is varied and widely documented. They regulate epithelial-mesenchymal transitions by downregulating epithelial-specific genes and are involved in other cellular processes, such as neural crest delamination. Thomas Gridley's group now report that, surprisingly, *Snai1* and *Snai2*, encoding Snail and Slug, respectively, have no apparent role in neural crest generation or delamination in mice, but are crucial for proper craniofacial morphogenesis (p. 1789). They show that the deletion of both copies of *Snai1* in embryonic neural crest cells performed on a null *Snai2* genetic background results in multiple craniofacial defects, and a cleft palate defect that is very different from that seen in $Snai1^{+/-}$ $Snai2^{-/-}$ mouse embryos. This cleft palate defect arises from the failure of Meckel's cartilage to extend the mandible, a condition also seen in humans with Pierre Robin Sequence, in which a smaller mandible prevents the correct positioning of the tongue. As such, these mice provide a useful model in which to study this developmental disorder.



Placental development: attached to chaperones

A successful pregnancy depends on many events, such as the attachment of the allantois to the chorionic mesothelium. However, little is known about the genes that are expressed in the chorionic trophoblast and whose loss results in attachment defects. *Mrj*, which encodes a co-chaperone, is one of these genes, and is now shown by James Cross and colleagues to regulate the turnover of keratin in the developing mouse placenta (see p. 1809). Chorioallantoic attachment fails and keratin inclusion bodies develop in $Mrj^{-/-}$ embryos. This attachment failure is a consequence of cytotoxicity, the authors show, that is caused by keratin inclusion bodies rather than by a failure of the keratin cytoskeleton to form; keratin-deficient embryos correctly attach, and a reduction in keratin expression in $Mrj^{-/-}$ conceptuses rescues failed attachment. Mrj is known to interact with Huntington disease proteins with expanded N-terminal repeat aggregates within neurons. Because inclusion bodies are associated with other neurodegenerative diseases, the authors propose that Mrj may have a more general role in preventing intracellular inclusion bodies.

IN *JOURNAL OF CELL SCIENCE*

Wound healing goes to Rac and ruin

During wound healing, the increased proliferation of epidermal keratinocytes and their migration across wounds repairs damaged epithelium. The signalling mechanisms that underlie skin re-epithelialization are unclear, but the small GTPase Rac1 has been implicated. Ingo Haase and colleagues now provide the first clear evidence that Rac1 is required for normal epidermal wound healing in vivo in mammals. Transgenic mice expressing dominant-negative Rac1 (N17Rac1) in epidermal keratinocytes and mice with an epidermis-specific deletion of Rac1 have a normal epidermis but skin wounds repair slowly. The authors show that the proliferation and migration of N17Rac1-expressing keratinocytes are inhibited in vitro and that the migration defects of these cells are particularly pronounced on collagen. Finally, they report that decreased persistence of lamella protrusions may cause these migration defects. These findings provide much-needed insight into the role of Rac1 in re-epithelialization.

Tscharntke et al. (2007). Impaired epidermal wound healing in vivo upon inhibition or deletion of Rac1. *J. Cell Sci.* **120**, 1480-1490.