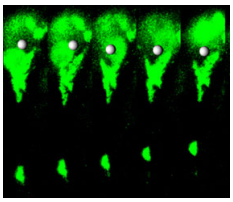
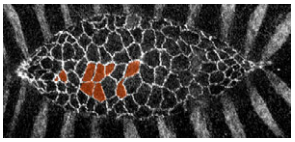


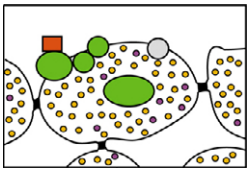
Neural crest emigrates in numerous ways

Neural crest cells (NCCs) give rise to various tissues, including pigment cells and certain neurons. They arise from the embryonic dorsal neural tube (NT) through a so-called epithelial-mesenchymal transition (EMT), during which polarised neuroepithelial cells turn into mesenchymal cells that emigrate from the NT. NCC EMT is generally thought to occur via a linear cascade of events, but on p. 1801, Ahlstrom and Erickson report findings that challenge this assumption. The authors tracked chick NCCs undergoing EMT through confocal time-lapse imaging and observed that although most NCCs detach from the NT lumen, retract their tail and translocate their cell body out of the neuroepithelium, this is not what happens in all cells. The order of events might vary, the tail might be ruptured instead of retracted, or cell body translocation might occur when the cell is morphologically rounded up during cell division. Based on these and other findings, the authors propose that EMT-associated events are mainly independently regulated and only cooperate loosely to allow NCC emigration from the NT.



Straining to reach (dorsal) closure

An organism's shape depends on morphogenetic cell and tissue movement, but how do individual cells contribute to tissue behaviour? To find out, Gorfinkiel, Martinez Arias and colleagues have quantitatively analysed cell-shape changes during *Drosophila* dorsal closure (DC) (see p. 1889). Halfway through embryogenesis, the embryo's epidermis has a gap that is covered by an extraembryonic tissue, the amnioserosa (AS). During DC, concerted tissue movements of the epidermis and the AS close this gap. Using a novel image-analysis method, the authors track the cell-shape changes of each individual AS cell (ASC) during DC and quantify them in normal embryos and in embryos in which apoptosis, microtubule dynamics or adhesion is disturbed in the AS and/or epidermis. In all cases, they observe spatial and temporal differences in ASC deformation and a correlation between ASC deformation rates and the zippering speed of the two epidermal sheets that close the gap. They suggest, therefore, that mechanical constraints from tissues outside the AS can modulate AS cell-shape changes and thus collective tissue behaviour.

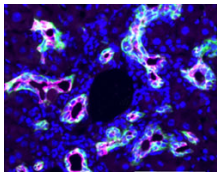


cGMP holds oocytes under arrest

In mammals, oocytes are kept from completing meiosis by a signal that comes from the surrounding somatic cells until oocyte cAMP levels drop in response to luteinising hormone (LH). What is the nature of the somatic cell signal? The answer, claim Nikolaev, Jaffe and colleagues on p. 1869, is cGMP. The authors measure cyclic nucleotide concentrations in intact follicle-enclosed mouse oocytes using FRET-based fluorescent indicators and observe that, in meiotically arrested oocytes, cGMP passes through gap junctions into the cells. There, it inhibits cAMP hydrolysis by the phosphodiesterase PDE3A, thus maintaining high cAMP levels and blocking meiotic progression. LH acts by lowering somatic cell cGMP levels and by closing the gap junctions of the somatic cells, which results in a decrease in oocyte cGMP levels. This, in turn, allows PDE3A to hydrolyse oocyte cAMP, leading to meiotic progression. Thus, the authors conclude, somatic cell cGMP maintains meiotic arrest in oocytes, and LH causes meiotic progression by interfering with this somatic cGMP signal.

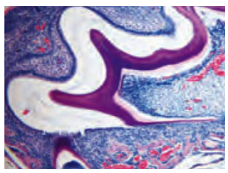
NEVERSHED sends cargo that makes petals drop

At certain developmental stages, plants shed their leaves, flowers and fruit in a process known as abscission. Even though abscission is crucial for the reproductive success of flowering plants, little is known about the genes that control this process. Now, Sarah Liljegren and co-workers demonstrate that the *Arabidopsis NEVERSHED* (*NEV*) gene is required for floral abscission (see p. 1909). The authors identified *NEV* through a screen for mutants that retain their floral organs indefinitely and establish that it encodes an ADP-ribosylation factor GTPase-activating protein (ARF-GAP), a type of protein that regulates membrane trafficking and remodelling of the actin cytoskeleton. NEV localises to the trans-Golgi network (TGN) and to endosomes; *nev* mutations disrupt the structure of the Golgi apparatus, alter the location of the TGN and cause paramural vesicles to accumulate near cell walls in the abscission zones of flowers. The researchers propose, therefore, that a crucial role of NEV is to facilitate the transport of cargo molecules required for abscission through the TGN and endosomal pathways.



EpCAM identifies hepatic stem cell potential

The mammalian liver has remarkable regenerative capacities, but whether adult hepatic stem cells (HepSCs) exist is debated. Now, on p. 1951, Minoru Tanaka and colleagues identify a cell-surface marker for mouse liver oval cells (OCs) and show that isolated OCs contain potential HepSCs. Liver regeneration generally does not require stem cells; instead, hepatocytes, which perform most of the liver's metabolic functions, proliferate. Blocking injury-induced hepatocyte proliferation, however, leads to the proliferation of OCs (facultative progenitor cells that probably generate both hepatocytes and cholangiocytes, the bile-duct-forming liver cells). The authors find that the adhesion molecule EpCAM is expressed in both OCs and cholangioblasts, whereas the EpCAM-related protein TROP2 is only present in injury-activated OCs. EpCAM-positive cells isolated from either injured or normal liver form colonies in vitro, and these clonally expanded cells can differentiate into hepatocytes and cholangiocytes, indicating that the EpCAM-positive cell population contains potential HepSCs. Future research will show whether these potential HepSCs can self-renew and repopulate the liver in vivo.



The APC of toothiness

Mice and humans form one or two sets of teeth, respectively, but mutations in the adenomatous polyposis coli (*Apc*) gene triggers additional tooth formation in both species. *Apc* encodes a negative regulator of Wnt signalling, and studies in embryonic mice have shown previously that activating Wnt signalling also induces additional teeth. On p. 1939, Richard Maas and colleagues now reveal that deleting *Apc* in the oral epithelium of adult mice has a similar effect. Using several transgenic mouse lines, the authors show that without Apc, many different embryonic and juvenile jaw areas generate extra teeth. Additional teeth also form in adult mice, mainly in the incisor region, which contains dental epithelial stem cells. The effects of *Apc* deficiency are non-cell-autonomous and mediated by β-catenin, with *Fgf8*, an early tooth initiation marker, being one target of Wnt/β-catenin signalling. Surprisingly, extra teeth can form in the absence of the homeobox gene *Msx1*, which is required for normal tooth formation. Future studies could see the application of these findings to tooth repair and regeneration in humans.