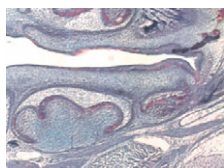




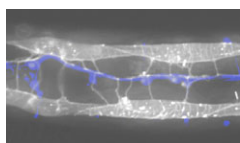
Wnt double duty in regenerating planaria

Planaria flatworms can regenerate an entire organism from tiny body fragments, making them an ideal organism for the study of regeneration. β -catenin is important for the regeneration of posterior body parts in the planarian species *Schmidtea mediterranea*, which suggests that canonical Wnt signalling functions in this process, but this has yet to be confirmed experimentally. Now, on p. 905, Kerstin Bartscherer and co-workers demonstrate that the Wnt secretory protein Wntless/Evi and several planarian Wnt ligands regulate *S. mediterranea* regeneration. Through RNAi-mediated silencing of *Smed-wntless* and of all putative planarian Wnt genes, the authors show that, similar to *Smed- β -catenin1*, *Smed-wntless*, *Smed-wnt11-2* and *Smed-wntP-1* are required for posterior regeneration. Surprisingly, they also report that *Smed-wntless* and *Smed-wnt5* are required for the proper regeneration of the planarian nervous system, and that this requirement is β -catenin independent. Thus, the authors suggest, planarian Wnts are important regulators of regeneration that signal through β -catenin-dependent and -independent pathways, all of which depend on *Smed-Wntless/Evi*.



Less cilia for more bite

In mammals, the number of teeth that form an animal's dentition is strictly controlled. In mice, the diastema, a toothless region between the incisors and the molars, contains tooth primordia during embryonic development, but these primordia are suppressed by apoptosis. On p. 897, Paul Sharpe and colleagues surprisingly reveal that a defect in primary cilia results in increased hedgehog signalling and in the formation of ectopic teeth in the diastema. Mice mutant for the *lft88/polaris* gene, which encodes a cilia intraflagellar transport protein, display ectopic tooth formation that correlates with ectopic sonic hedgehog (Shh) activity, and tissue-specific mutants reveal that *polaris* is required in the dental mesenchyme, but not the ectoderm, for normal tooth development. The authors also demonstrate that mice mutant for the Shh antagonist *Gas1* display increased Shh activity and ectopic diastema teeth. Taken together, these data indicate that, contrary to prior reports, primary cilia negatively regulate Shh activity in the diastema mesenchyme, resulting in the suppression of tooth formation.



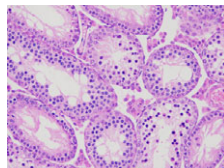
The long arm of axon guidance factors

Guided cell migration and postsynaptic membrane expansion (PME) are both important developmental processes, but much remains to be learned about their regulation. Now, Peter Roy and colleagues report that in *C. elegans* neuromuscular junction formation, during which muscle cells extend membrane processes called muscle arms towards the motor axons, several previously identified cell and axon guidance genes also direct PME (see p. 911). In a genetic screen for mutants with fewer muscle arms, the authors identified 10 genes, including *unc-40/Dcc*, which encodes a transmembrane receptor that guides cell and axonal migration in response to UNC-6/Netrin. They find that UNC-40 is enriched in muscle arms and directs muscle arm extension to motor axons independently of UNC-6. Among the factors that lie downstream of UNC-40, the authors report, are the guanine-nucleotide exchange factor UNC-73/Trio, members of the WAVE actin-polymerisation complex and the focal adhesion component homologue UNC-95. Together, these data suggest that many genes required for guided cell and growth cone migration have related roles in directing PME.



Maternal control goes up in Smaug

Following fertilisation, animal development is initially regulated by maternal factors that are present in the unfertilised egg. Developmental control then transfers to the zygotic genome, in a process known as the maternal-to-zygotic transition (MZT). Now, on p. 923, William Theurkauf and colleagues identify the RNA-binding protein Smaug as an essential regulator of MZT in *Drosophila*. The researchers report that Smaug, which participates in maternal mRNA destruction during MZT, is also required for other MZT-associated events, such as slowing of the cleavage divisions, cellularisation, DNA replication checkpoint activation and the initiation of zygotic transcription, including that of the *miR-309* cluster of microRNAs. This cluster directs the degradation of certain maternal mRNAs during MZT, indicating that Smaug mediates mRNA destruction both directly and indirectly. Furthermore, when the researchers transgenically expressed Smaug in an anterior-to-posterior gradient in the zygote, many MZT-associated events were initiated in a concentration-dependent manner. From their results, the authors propose that Smaug accumulation drives a maternal clock that controls MZT timing.



Piwi and fertility hit by Sun1 down

Sun1, an inner nuclear membrane protein, locates to telomeres and participates in postmitotic chromatin decondensation, but its functions in vivo have been subject to debate. Here, Kuan-Teh Jeang and co-workers report that Sun1 is required for the expression of coding and non-coding RNAs that are crucial for male gametogenesis (p. 965). *Sun1*^{-/-} mice, they find, are born and grow normally, but are reproductively sterile; the males' gonads are significantly smaller than those of their wild-type counterparts, and spermatogenesis is disrupted. *Sun1*^{-/-} testes also show reduced expression of genes that are associated with reproduction, including that of *Mili* and *Miwi*, two members of the Piwi gene family. *Mili* and *Miwi* participate in the biogenesis of Piwi-associated RNAs (piRNAs), which are crucial for germline development. The authors show that piRNA expression is also strongly reduced in *Sun1* mutant animals, and propose that compromised RNA expression, as well as a previously recognised loss of telomeric attachments, in these mice contributes to their sterility.



How plant defence gets hairy

Many plant species respond to damage from herbivore grazing by generating leaves with more trichomes (hair-like epidermal cells). Interestingly, damage at mature leaves affects newly forming leaf primordia and increases the frequency with which epidermal cells adopt a trichome fate. What controls this fate choice systemically? The answer, report Kiyotaka Okada and co-workers on p. 1039, is the plant hormone jasmonic acid. In *Arabidopsis* mutants that lack endogenous jasmonate (JA) signalling, trichome formation is normal, but the trichome patterning wounding response is absent. The authors isolate two additional wounding response mutants, *unarmed 9 (urm9)* and *urm23* (which is allelic to *tgt1*). In both mutants, the subnuclear localisation of the bHLH transcription factor GLABRA3 (GL3), a component of the Myb-bHLH-WD40 complex that regulates trichome development, is disrupted. In addition, JA treatment enhances GL3 expression prior to trichome initiation in both wild-type and *urm9* backgrounds. Based on these and other results, the authors propose that the activation of GL3 by JAs constitutes the link between the wounding response and trichome patterning in *Arabidopsis*.