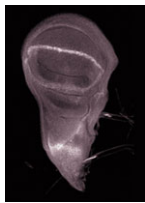


Immune system development curtails regeneration

Many animals can regenerate lost appendages, but for unknown reasons their regenerative capacity varies with species, appendage and developmental stage. Now, Takeo Kubo and colleagues reveal that the development of the immune system impacts negatively on the regeneration of *Xenopus* tadpole tails (see p. 2323). Tadpoles can regenerate amputated tails, but briefly lose this ability during the so-called refractory period (embryonic stage 45–47) for some reason. By comparing gene expression profiles at wound sites following tail amputation before, during and after the refractory period, the researchers discovered that immune responses differ between these three periods; during the regenerative periods, they are either not yet established or well matured. Intriguingly, suppressing immune responses during the refractory period, using pharmacological agents or by depleting immune cells by knocking down the transcription factor PU.1, restores regenerative capacity. These findings establish, for the first time, a direct link between an organism's regenerative capacity and its immune responses. Future studies should show whether this link is conserved in other systems.



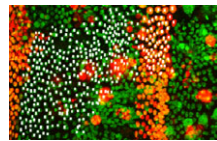
Ecdysone uncouples wing development from nutrition

In flies and other insects, starvation delays metamorphosis from larval to pupal stages, but once a certain size or weight – the critical weight – is reached, development proceeds independently of nutrition. What regulates this switch? On p. 2345, Christen Mirth and colleagues report that in *Drosophila* wing imaginal discs, it is ecdysone molting hormone signalling. They show that the expression of wing-patterning proteins is suppressed when larvae are starved pre-critical weight, but that this expression becomes nutrition-independent thereafter. Increasing the size of the prothoracic gland, which produces ecdysone, pre-critical weight through enhanced insulin signalling during starvation leads to premature nutrition-independent wing-patterning protein expression; suppressing insulin signalling delays it. Knocking down the ecdysone receptor (EcR) in the wing disc, which leads to the derepression of transcriptional targets normally repressed by unbound EcR, also results in premature nutrition-independent patterning protein expression. Together, these findings reveal that ecdysone signalling switches wing disc development to a nutrition-independent mode by derepressing genes that are repressed by unbound EcR.



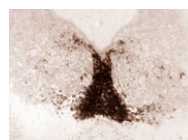
Plant PcG conservation identified

Polycomb group (PcG) protein complexes, which function in the epigenetic regulation of gene expression, control numerous developmental processes in plants and animals. For instance, the *Arabidopsis* PcG protein FERTILIZATION INDEPENDENT ENDOSPERM (AtFIE) regulates the transition of the haploid female gametophyte to the diploid sporophyte phase. Nir Ohad and co-workers now report that FIE function is evolutionarily conserved from mosses to flowering plants (see p. 2433). They identify a FIE homologue, PpFIE, in the moss *Physcomitrella patens*, which is expressed only in apical gametophytic cells and in stem cells that undergo fate transition. Deleting PpFIE leads to disrupted gametophytic cell differentiation, indicating that the function of PpFIE is similar to that of AtFIE. The fact that AtFIE can partially rescue the PpFIE loss-of-function phenotype (and vice versa) and that PpFIE can interact with other PcG proteins further support this notion. Thus, the role of the FIE PcG complex in regulating gametophytic differentiation appears to have been established early in plant evolution.



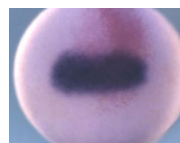
Histoblast migration gets abdomen into shape

Shaping complex tissues requires the organisation of large groups of cells, but how the behaviour of individual cells during such processes is controlled and coordinated remains elusive. During metamorphosis, the *Drosophila* abdominal epidermis is formed by the extensive migration of imaginal histoblast cells that replace the larval abdominal epithelium; on p. 2403, Bischoff and Cseresnyés analyse histoblast behaviour in unprecedented detail. By tracking individual histoblasts in three dimensions over time, the authors show that these cells move in two phases, first migrating dorsally and then turning anteriorly. More anteriorly positioned histoblasts turn earlier, whereas more posterior ones migrate faster and turn later. In other tissues, such as the embryonic germband, morphogenesis also depends on oriented mitosis, but histoblast divisions are not preferentially aligned. In addition, the authors demonstrate that histoblasts actively contribute to their own movement. These findings highlight the diversity of morphological processes involved in shaping a multicellular organism and offer new inroads into understanding the mechanisms that underpin epithelial morphogenesis.



Dopaminergic gene regulatory network comes into view

Midbrain dopaminergic (mdDA) neuron generation crucially depends on the cooperative action of the transcription factors Nurr1 and Pitx3, but little is known about their transcriptional targets. Now, on p. 2363, Marten Smidt and colleagues identify three novel Nurr1 target genes in mice. Through a combination of microarray, chromatin immunoprecipitation and gene expression studies on normal and *Nurr1*-deficient midbrain tissue, as well as on a *Nurr1*-overexpressing midbrain-derived cell line, the authors establish that *Dlk1*, *Ptpru* and *Klhl1* are Nurr1 target genes. They also show that *Ptpru* and *Klhl1* expression in mdDA neurons is *Pitx3* dependent, whereas *Dlk1* expression does not require *Pitx3*. Their gene expression analysis of several mdDA markers in *Dlk1* knockout mice revealed that *Dat*, a gene involved in dopamine metabolism, is expressed prematurely in certain dopaminergic precursor cells of these animals. Taken together, these data underscore the complexity of Nurr1-Pitx3 downstream activity and represent an important advance towards understanding the intricate regulatory networks that govern mdDA neuron development.



Self-regulating Mex3b in an RNA bind

Most developmental events require the precise control of gene expression, and RNA-binding protein-mediated post-transcriptional processes play an important role in such control. Now, Masanori Taira and colleagues identify a novel post-transcriptional control mechanism in *Xenopus* that centres on the RNA-binding protein Mex3b (see p. 2413). The *mex3b* mRNA contains a long, evolutionarily conserved 3'UTR (3'LCU), which, the authors report, promotes mRNA translation and destabilisation. Mex3b promotes the destabilisation of its own mRNA by binding to its 3'LCU, thus forming an autoregulatory negative-feedback loop, the in vivo presence of which the researchers demonstrate through *mex3b* overexpression and knockdown experiments. They also reveal a role for Mex3b in neural plate patterning that involves fibroblast growth factor (FGF) signalling. As Mex3b levels affect FGF responsiveness, and as the mRNAs of the FGF signalling components *sdc2* and *ets1* appear to be direct Mex3b targets, the authors suggest that the tightly controlled, autoregulated expression levels of *mex3b* in turn regulate FGF signalling during neural patterning.