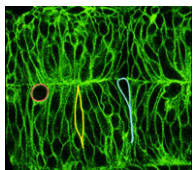


Stem cell maintenance taken down a Notch

Adult tissues are maintained throughout life through the renewal of differentiated cells by adult stem cells, but what controls the competing processes of stem cell self-renewal and differentiation? On p. 705, Allison

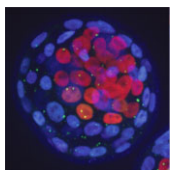
Bardin, François Schweisguth and colleagues present a new model for the transcriptional control of stem cell maintenance in the *Drosophila* intestine. When *Drosophila* intestinal stem cells (ISCs) divide, one daughter cell retains the ISC fate while the other becomes an enteroblast, which differentiates into two mature cell types. Using lineage mapping and genetic manipulation, the researchers show that ISC maintenance requires the transcriptional repression of key Notch target genes – in particular, the *Enhancer of split* complex (*E(spl)-C*) genes – by a Hairless–Suppressor of Hairless complex. Furthermore, Daughterless, a bHLH transcriptional activator, is also needed for ISC maintenance. From their results, the researchers propose that Daughterless and *E(spl)-C* factors act antagonistically to maintain the balance between ISC self-renewal and differentiation.



Relaxing into hindbrain development

Brain ventricles (fluid-filled cavities formed from the lumen of the neural tube) are essential for normal vertebrate brain function. Jennifer Gutzman and Hazel

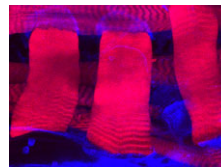
Sive now uncover a role for the myosin phosphatase inhibitory regulator *mypt1* in the expansion of the hindbrain ventricle in zebrafish embryos (see p. 795). Myosin phosphatase regulates non-muscle myosin II contractility by regulating the phosphorylation of the myosin regulatory light chain (MRLC). The researchers report that a mutation in *mypt1* produces a small hindbrain ventricle and disrupts the morphogenesis of the hindbrain rhombomeres (structures that differentiate into specific cranial nerves). In addition, although MRLC phosphorylation levels change during hindbrain morphogenesis in wild-type zebrafish embryos, they are consistently high in *mypt1* mutants, and inhibition of myosin II function rescues all of the defects observed in *mypt1* mutants. The researchers suggest, therefore, that myosin phosphatase activity ‘relaxes’ the neuroepithelium by reducing myosin contractility, thereby facilitating hindbrain morphogenesis and ventricle expansion. Epithelial relaxation, they add, might also facilitate tube inflation in other organs.



FGFs, not lineage, set early embryonic fates

Primitive endoderm (PE) and epiblast (EPI), which give rise to extra-embryonic tissues and the foetus, respectively, are derived from the inner cell mass (ICM) at day 3.5

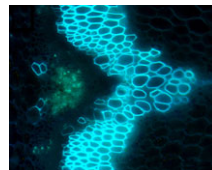
(E3.5) of mouse embryogenesis and sort into distinct layers by E4.5. How the PE and EPI are initially specified is unclear, but one theory is that cell lineage history drives their fate. Now, using live cell tracing, Yojiro Yamanaka and co-workers report that they find no clear correlation between cell lineage history and PE/EPI segregation in mouse embryos; instead, FGF signalling directs this important segregation event (see p. 715). FGF/MAP kinase pathway inhibition shifts ICM cells towards an EPI fate, they show, whereas exogenously applied FGF shifts ICM cells towards PE. Furthermore, modulating FGF signalling during blastocyst maturation can still shift ICM cell fate, even though the mutually exclusive expression of lineage-specific transcription factors has begun. Thus, the researchers propose, stochastic and progressive specification of PE and EPI lineages occurs during blastocyst maturation in an FGF/MAP kinase signal-dependent manner.



Slowdown in muscle integrity

During development, adhesion between distinct cell types is crucial for tissue assembly. Now, on p. 785, Eliezer Gilsohn and Talila Volk show that the novel tendon-derived protein Slowdown (Slow)

promotes muscle integrity in *Drosophila* embryos by ensuring that myotendinous junctions (MTJs) form correctly between muscle and tendon cells to ‘glue’ them together. MTJs, a type of hemi-adherens junction, consist mainly of muscle-specific integrin receptors and their tendon-derived extracellular matrix ligand thrombospondin. The researchers identified Slow in a microarray screen for target genes of the tendon-specific transcription factor Stripe. They show that muscle and/or tendons rupture upon muscle contraction in *slow* mutant larvae and that *slow* mutant flies are unable to fly. These defects, they report, are the result of improper assembly of the embryonic MTJ. Other experiments indicate that Slow normally forms a complex with thrombospondin that can alter the morphology and directionality of muscle ends. Thus, Gilsohn and Volk conclude, Slow promotes muscle integrity by modulating integrin-mediated adhesion at MTJs.



Receptor-ligand pair divides plant cells

Orientated cell divisions are an essential part of development in many multicellular organisms. In particular, in plants, morphogenesis is determined

solely by differential growth, which depends on the orientation of cell division planes. But how are these planes correctly lined up? On p. 767, J. Peter Etchells and Simon Turner report that signalling through the receptor-like kinase PXY and its peptide ligand CLE41 regulates the rate and orientation of cell division in the vascular meristem of *Arabidopsis*, which forms both the xylem and the phloem (the plant’s water and sugar conducting tissues). The researchers show that PXY is expressed within dividing meristematic cells, whereas CLE41 localises to the adjacent phloem cells. Importantly, alterations in the CLE41 expression pattern, but not overexpression of CLE41 in the phloem, disrupt cell division orientation and ordered vascular development. Given that localised signalling from adjacent cells also defines cell division planes in animal systems, the researchers suggest that this might be a general mechanism for regulating orientated cell division during development.



Insect evolution: filling in the gaps

In the long germband insect *Drosophila*, all of the body segments are specified simultaneously, but

most insects undergo ‘short’ or ‘intermediate’ germ segmentation in which the anterior and posterior segments are specified at different times. In *Drosophila*, gap genes both specify, and provide identity to, several contiguous segments, so what is their function in short/intermediate-germband insects? On p. 835, Paul Liu and Nipam Patel report that the homolog of the *Drosophila* gap gene *giant* in *Oncopeltus fasciatus* (an intermediate germband insect) is a bona fide gap gene. RNAi knockdown of *Oncopeltus giant*, they report, results in deletion of the maxillary and labial segments and of the first four abdominal segments. By contrast, previous work indicates that in *Tribolium castaneum*, a short germband beetle, *giant* controls gnathal (jaw) segment identity, rather than its formation, and is required for the segmentation of the entire abdomen. Liu and Patel suggest, therefore, that *giant* was a true gap gene in the common insect ancestor but lost this role during *Tribolium* evolution.

Jane Bradbury