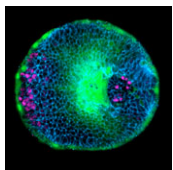


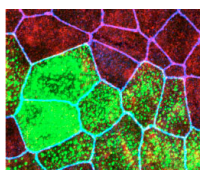
Sertoli cell sex determination secret(ion)

In mammalian embryos, the supporting cells in the developing gonads (which differentiate into the Sertoli and granulosa cells of the testes and ovaries, respectively) 'tell' the germ cells which sex to become. Retinoic acid is thought to control this process by inducing meiosis in the developing ovaries but not in the testes where it is metabolised. However, on p. 1415, Best and colleagues' results support an alternative theory: that a meiosis inhibitor exists in mouse embryonic testes. The researchers identify *Sdmg1*, a conserved transmembrane protein that is expressed in mouse embryonic Sertoli cells at the time of germ-cell masculinization. Knock-down of *Sdmg1* in Sertoli cell lines disrupts membrane trafficking and impairs their ability to masculinize germ cells, they report. Furthermore, perturbing secretion with brefeldin A in male embryonic gonads in organ culture causes male-to-female germ cell sex reversal. The researchers propose, therefore, that Sertoli cells communicate male-sex determining decisions to the germ cells during development by secreting either a meiosis inhibitor or other signalling molecules.



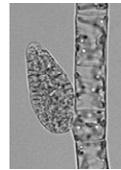
JAK/STAT signalling fixes neuroblast numbers

During *Drosophila* development, some neuroepithelial cells become neuroblasts (neural stem cells) and generate the neuronal and glial cells of the fly's nervous system. But what controls the transition from neuroepithelial cell to neuroblast? In the *Drosophila* optic lobe, report Yasugi and colleagues, a wave of proneural gene expression that is negatively regulated by JAK/STAT signalling triggers neuroblast formation (see p. 1471). During optic lobe development, neuroepithelial cells at the medial edge of the outer optic anlage develop into neuroblasts, which generate the medulla neurons; those at the lateral edge develop into lamina neuron precursors. The researchers show that a wave of expression of the proneural protein Lethal of scute sweeps from the anlage's medial edge to its lateral edge and induces neuroblast formation. JAK/STAT signalling in the most lateral neuroepithelial cells negatively regulates this wave, they report. Thus, JAK/STAT signalling balances neuroblast and lamina neuron numbers in the optic lobe and allows the formation of a precise topographic map in the visual centre.



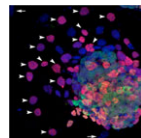
Epithelial remodelling: a ROCK-hard interaction

During embryogenesis, epithelial cell apical constriction (via actomyosin contraction) forces epithelial sheets to invaginate – a morphological change that can generate tissues with a complex architecture. Now, on p. 1493, Nishimura and Takeichi report that Shroom3 (an actin-binding protein that regulates epithelial apical constriction) and Rho kinases [ROCKs; kinases that phosphorylate myosin regulatory light chain (MLC), leading to actomyosin contraction] interact to regulate epithelial sheet remodelling. The researchers show that in epithelial cells in vitro and in invaginating chick neural tube, Shroom3 binds to and recruits ROCKs to the epithelial apical junctions. They also show that the expression of RII-C1 (the Shroom3-binding site on ROCKs that they identify) or Shroom3 depletion removes apically localized ROCKs and blocks neural tube closure. Finally, epithelial cells in the closing neural plate have a planar-polarized distribution of phosphorylated MLC, they report, that is abolished by RII-C1 expression. This distribution might drive tube closure, suggest the researchers. Together, these results provide new insights into how epithelial sheets bend during embryogenesis.



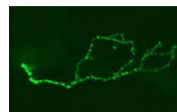
Insights into algae's alternative life style

Brown algae evolved multicellularity independently of animals and higher plants, and so are of considerable interest to evolutionary and developmental biologists. The life cycle of the model brown alga *Ectocarpus siliculosus* consists of two independent, alternating multicellular forms (generations): sporophytes and gametophytes. This alternation of generations involves an alternating pattern of cell division in the 'initial' cell: asymmetric initial-cell division occurs in gametophyte development and symmetric division in sporophyte development. Now Peters et al. (see p. 1503) provide a detailed account of the markedly different patterns of early development of these two generations, and correlate these differences with the mode of initial-cell division. Importantly, they also report a rare life cycle mutant, called *imm*, in which gametophyte characteristics develop in the sporophyte generation and in which genes normally expressed during the gametophyte stage are upregulated, while sporophyte-generation genes are downregulated. These insights into the development and genetic control of the *E. siliculosus* life cycle will enlighten future studies of this organism, the genome of which is currently being sequenced.



Running Rings around ES cell pluripotency

The maintenance of embryonic stem (ES) cell pluripotency depends on a core transcriptional regulatory network that represses key transcription factors involved in differentiation and development. The Polycomb group (PcG) of proteins mediate the heritable silencing of developmental regulators, but are the PcG proteins and this transcriptional network functionally linked? Endoh and co-workers now show that the Polycomb repressive complex 1 (PRC1) components Ring1A/B act downstream of the core transcriptional regulatory circuitry that maintains mouse ES cell self renewal (see p. 1513). Ring1A/B, they show, are essential for maintaining undifferentiated ES cells through their repression of certain developmental regulators that direct ES cell differentiation. This silencing, which is achieved through the inhibition of chromatin remodelling, depends on a key component of the core transcription network: Oct3/4. But Oct3/4 also binds to these targets independently of PRC1. These and other results clearly show that a functional link between Ring1A/B-mediated PRC1 silencing and the core transcriptional regulatory circuitry acts to maintain ES cell self renewal.



Fragile X: a developmental disease?

Fragile X syndrome (FraX), a common inherited mental retardation and autism disorder, is caused by the loss of FMRP, an mRNA-binding protein that regulates mRNA stability and translation. Individuals with FraX have immature neuronal processes and decreased plasticity of mature synapses. So is this a disease of development, neuronal plasticity or both? On p. 1547, Tessier and Brodie reveal a prominent role for FMRP in activity-dependent neural circuit refinement during brain development in *Drosophila*. They show that in the fly FraX model, brain RNA and protein levels are increased during late brain development and during early-use refinement, a period of activity-dependent process pruning. FMRP expression normally peaks during this pruning period, they report, and is positively controlled by sensory input activity. Most importantly, FMRP expression is required for activity-dependent pruning during neural circuit refinement in the Mushroom Body, the brain region where learning and memory are consolidated. Together, these results reveal a critical late development role for FMRP and suggest that FraX is primarily a developmental disease.

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