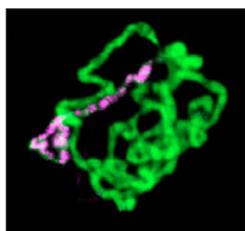


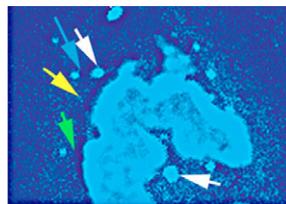
Sizing up size control

The developmental mechanisms that control the growth and final size of organs are poorly understood. What, for example, determines the different sizes of halteres and wings, homologous thoracic structures in *Drosophila*? Two papers in this issue of *Development* provide new insights into this process. On p. 4421, Martín and Morata show that the control of growth and final size is an autonomous feature of the *Drosophila* wing imaginal disc and of its anterior and posterior compartments. They used *Minute* mutations, which prolong the larval period of heterozygous animals (*M/+*) without affecting the size of the larvae or adults, in a complex genetic strategy to generate slow-growing *M/+* larvae that contained fast-growing, wild-type *Minute*⁺ (*M*⁺) discs or compartments. These wild-type, *M*⁺ tissues have ~20 hours more development time than is normal, enough to quadruple in size, yet they grow only to a normal size in *M/+* larvae. And although adjoining *M/+* and *M*⁺ wing disc compartments in *M/+* larvae initially grow at different rates, they form adult wings of the correct shape and size. The researchers conclude that an autonomous mechanism within the wing disc compartments arrests their growth once they reach the right size, probably by lengthening the cell division cycle. On p. 4495, de Navas, Garaulet and Sánchez-Herrero tackle the question of what controls the size difference between halteres and wings, and show that the *Ultrabithorax* (*Ubx*) Hox gene controls haltere size by regulating Decapentaplegic (*Dpp*) signalling. *Ubx* is expressed in the haltere disc but not in the wing disc, and *Ubx* mutations increase haltere size, transforming them into wings. Because changes in *Dpp* signalling affect wing size, the authors wondered whether *Ubx* fixes haltere size by modifying the *Dpp* pathway. Their results indicate that *Ubx* downregulates *dpp* expression and alters *Dpp* activity in halteres. It also reduces *Dpp* spread, they report, by controlling the expression of the *Dpp* receptor *thick veins* and of *division abnormally delayed*, which encodes a cell-surface molecule. Thus, they suggest, changes in *Dpp* signalling that are induced by *Ubx* might partly account for the different size of halteres and wings.



SCF opens up for dosage compensation

In *Drosophila*, equal gene expression from the sex chromosomes in males and females (dosage compensation) is achieved by hypertranscription from the single X chromosome in male flies. Furuhashi and colleagues now identify DNA supercoiling factor (SCF), a protein that generates negative supercoils in DNA, as an important player in this process (see p. 4475). They show that SCF knockdown by RNAi causes male-specific reduction of X-linked gene expression and male lethality. SCF, they report, colocalizes along the X chromosome with the male-specific lethality (MSL) complex, which is required for dosage compensation, and overexpression of SCF gives the X chromosome a bloated appearance. This phenotype depends on the MSL component MOF, a histone acetyltransferase, and is suppressed by overexpression of the chromatin remodelling protein ISWI, which antagonizes MOF activity. The researchers conclude that by counteracting ISWI action, SCF helps to form and/or maintain the transcriptionally active open chromatin that is needed for X chromosome hypertranscription in male flies.



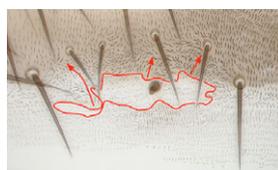
For plastic synapses – start recycling

Synaptic connections in the mammalian nervous system are established during development but are refined during adult life. In the CNS, synaptic plasticity is partly regulated by recycling postsynaptic neurotransmitter receptors. Bruneau and Akaaboune now describe the dynamics of acetylcholine receptor (AChR) recycling at the neuromuscular junction (NMJ) in mice and reveal that tyrosine dephosphorylation controls the insertion and maintenance of recycled AChR at this accessible synapse (see p. 4485). Previously, the researchers had shown, by using biotin-bungarotoxin and labelled streptavidin, that many AChRs reappear in the postsynaptic membrane after internalization and intermingle with AChRs that have not been internalized – so-called pre-existing AChRs. Now they show that recycled AChRs are removed from functional synapses much faster than pre-existing receptors. Denervation of the NMJ increases their removal rate; conversely, muscle stimulation prevents their loss. These findings shed light on how receptors at less accessible synapses are recycled and thus how synaptic plasticity is regulated in the CNS.



A new twist to LR asymmetry

The establishment of the embryonic axes is a crucial developmental event. Anteroposterior and dorsoventral axis determination is reasonably well understood, but in some organisms the earliest steps of left-right (LR) axis formation remain unclear. Now Danilchik and co-workers describe an intrinsic chirality in the cortex of *Xenopus* eggs that might predetermine this animal's LR asymmetry (see p. 4517). They report that one-cell *Xenopus* embryos and parthenogenetically activated eggs undergo a twisting motion in which the animal pole rotates counterclockwise past the vegetal cortex after treatment with 2,3-butanedione monoxime (BDM), which disrupts actin-myosin interactions. BDM treatment, they report, induces a shear zone parallel to the egg's equator in which long actin fibres develop in a microtubule-independent manner, and randomizes the LR orientation of visceral organs in affected tadpoles. The researchers suggest that the consistent chirality of the BDM-induced twisting movement reveals a cryptic asymmetry in the egg's cortical actin cytoskeleton that could play an early role in LR axis determination.



Planar polarity: a radical rethink

Planar cell polarity (PCP) – asymmetry in the plane of epithelial tissues – controls the arrangement of structures such as insect hairs and ommatidia. The prevailing model for PCP establishment in *Drosophila* proposes that a morphogen gradient organizes the expression of two atypical cadherins, Dachsous (*Ds*) and Fat (*Ft*), and of the Golgi protein Four-jointed, to set up *Ds* system gradients. These then act via the Stan system – the cadherin receptor-like molecule Starry Night (*Stan*) and the Wnt receptor Frizzled (*Fz*) – to orientate hairs and ommatidia. On p. 4561, José Casal, Peter Lawrence and Gary Struhl challenge this model by reporting that the *Ds* and *Stan* systems act independently to orientate abdominal hairs. For example, they show that ectopic expression of *ds* repolarizes surrounding cells even in flies that lack both *Stan* and *Fz*. Other experiments indicate that cells that send polarity information need either *Ds* or *Ft* but responding cells need both proteins. From these findings, the researchers propose a radical new model that suggests how *Ds*-*Ft* bridges between cells might propagate PCP.

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