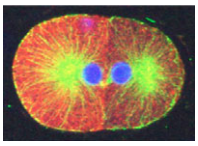
UFO that regulates flowering

Multiple endogenous and environmental signals control when plants flower. In *Arabidopsis*, the LEAFY (LFY) transcription factor integrates these signals by activating the expression of several floral homeotic genes, including *APETALA3* (*AP3*). Activation of *AP3* expression also requires the activity of UNUSUAL FLORAL ORGANS (UFO), an F-box component of an ubiquitin E3 ligase that labels proteins for proteasome-mediated degradation. Chae and colleagues now unexpectedly reveal that UFO regulates floral development in *Arabidopsis* by acting as a transcriptional co-factor (see p. 1235). They show that UFO interacts physically with LFY in vitro and in vivo, and that this interaction is needed to recruit UFO to the *AP3* promoter. UFO engineered to act as a transcriptional repressor reduces endogenous LFY activity, they report, and the disruption of proteasome activity interferes with LFY-dependent *AP3* activation. Together, these results suggest a new mechanism for regulating flower development in which UFO is a co-factor for LFY-induced *AP3* transcription that regulates the activity of LFY in a proteasome-dependent manner.

Dnmt1: a direct transcription repressor?

The regulation of gene expression during development often involves epigenetic changes. In vertebrates, for example, the cytosine methyltransferase Dnmt1 is essential for gene silencing in early embryos. It is widely thought that Dnmt1 fulfils this role by maintaining DNA methylation at inactive genes. However, on p. 1295, Richard Meehan and colleagues challenge this dogma by showing that xDnmt1 regulates gene silencing in early *Xenopus* embryos independently of its DNA methyltransferase activity. The researchers show that a partial reduction of xDnmt1 protein levels by morpholinos prematurely activates zygotic gene expression before the pre-midblastula transition, when these genes are normally activated in *Xenopus* embryos. However, this premature gene activation occurs without any decrease in DNA methylation, either globally or at specific loci. Furthermore, the injection of an mRNA encoding a catalytically inactive form of human DNMT1 rescues the morpholino-treated embryos. These and other data suggest that xDnmt1 (and possibly mammalian Dnmt1) regulates embryonic gene silencing both through its catalytic activity and by acting as a direct, non-catalytic transcription repressor protein.

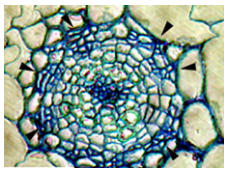


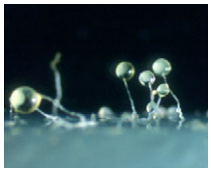
PLK-1 asymmetry makes time for cell division

During metazoan development, different cell lineages divide at different rates but how is the timing of cell division coupled with embryonic development? In *C. elegans*, lineage-specific cell-cycle duration is already apparent at the two-cell stage, when the anterior AB blastomere divides before the posterior P_1 blastomere. The preferential activation of a DNA-replication checkpoint in P_1 partly controls this asynchronous cell division. Now, Budirahardja and Gönczy (see p. 1303) report that the asymmetric distribution of the polo-like kinase PLK-1 (a positive mitotic regulator) also contributes to this asynchronous division. These researchers have discovered that anterior-posterior polarity cues cause PLK-1 to preferentially accumulate in AB. *plk-1*'s mild inactivation by RNAi does not delay mitotic entry in AB but does so in P_1, presumably because PLK-1's lower level in P_1 makes this blastomere more sensitive to PLK-1 depletion. The researchers propose, therefore, that the PLK-1-dependent mitotic advancement in AB and the checkpoint-dependent mitotic delay in P_1 together couple polarity cues and cell-cycle timing during worm development.

Ribosomal proteins turn over a new developmental leaf

The development of the characteristic planar shape of leaves depends on the establishment of an abaxial-adaxial axis in the leaf primordia. Many different genes are involved in this crucial developmental process. *REVOLUTA* [*REV*, which encodes a class III homeodomain-leucine zipper (HD-ZIPIII) transcription factor], *ASYMMETRIC LEAVES1* (*AS1*, a Myb-domain transcription factor gene) and *ASYMMETRIC LEAVES2* (*AS2*, which encodes a leucine-zipper transcription factor) are among the genes that specify the adaxial fate. KANADI family transcription factors, by contrast, help specify the abaxial identity by antagonizing HD-ZIPIII genes. In this issue, two papers unexpectedly reveal that ribosomal proteins are also involved in leaf polarity establishment in *Arabidopsis*. On p. 1315, Pinon et al. describe three genes – *PIGGYBACK1* (*PGY1*), *PGY2* and *PGY3* – that, when mutated, enhance the mild leaf polarity defects of *Arabidopsis as1* mutants. All three *pgy* mutants develop ectopic outgrowths on the adaxial (upper) side of leaves when on an *as1* mutant background. This phenotype is enhanced by *REV* mutations and is suppressed by *KANADI* gene mutations, highlighting the possibility that *PGY* genes might influence leaf development by interacting with the HD-ZIPIII-KANADI pathway. Surprisingly, the researchers show that *PGY1*, *PGY2* and *PGY3* encode the cytoplasmic large subunit ribosomal proteins RPL10a, RPL9 and RPL5, respectively. On p. 1325, Yao et al. report that several other *Arabidopsis* ribosomal protein genes are also involved in leaf patterning. The *ae5* and *ae6* mutants, they report, develop abaxial-like photosynthetic tissue (mesophyll) in the adaxial mesophyll domain and produce severely abaxialised leaves when crossed to *as1/2* mutants. Again, surprisingly, *AE5* and *AE6* were found to encode the ribosomal large subunit proteins – in this case, RPL28A and RPL5A, respectively. Yao et al. also report that plants with mutations in two other ribosomal protein genes (*RPL5B* and *RPL24B*) have similar phenotypes to the *ae5* and *ae6* mutants. Like Pinon et al., they also provide evidence that *AE5* and *AE6* interact with the HD-ZIPIII-KANADI pathway. Taken together, the results of these two papers strongly suggest that ribosome-mediated translational control is an important, and unexpected, regulator of leaf patterning.





STAT activation: DIF-ining a new mechanism

The accepted mechanism of STAT activation involves its phosphorylation by a ligand-activated tyrosine kinase, which induces STAT dimerisation and its nuclear entry. Now, though, Jeffrey Williams and colleagues reveal that STAT activation – an important regulator of gene expression – can be driven by phosphatase inhibition rather than just by kinase activation. In *Dictyostelium*, they report, the prestalk cell inducer DIF-1 activates STATc by inhibiting the protein tyrosine phosphatase PTP3 (see p. 1347). DIF-1 controls the differentiation of certain prestalk cells by inducing the nuclear localisation of STATc in these cells. The researchers show that PTP3 interacts directly with STATc and that its inhibition causes the constitutive tyrosine phosphorylation of STATc and its nuclear localisation throughout the developing slug. The treatment of developing *Dictyostelium* cells with DIF-1 or their exposure to hyper-osmotic shock (which also induces STATc nuclear localisation) reduces PTP3's activity; significantly, both treatments induce the serine-threonine phosphorylation of PTP3. Together, these results provide important new insights into STAT activation.

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