New insights into plant development in New England

Liam Dolan[1] and Jane A. Langdale[2,*]

[1]John Innes Centre, Colney, Norwich NR4 7UH, UK
[2]Department of Plant Sciences, University of Oxford, South Parks Rd, Oxford OX1 3RB, UK
*Author for correspondence (e-mail: jane.langdale@plants.ox.ac.uk)

Development 131, 5215-5220
Published by The Company of Biologists 2004
doi:10.1242/dev.01439

Summary

This year, the biannually organized FASEB meeting 'Mechanisms in Plant Development' took place in August in Vermont, USA, organized by Martin Hulskamp (University of Köln, Köln, Germany) and John Schiefelbein (University of Michigan, Ann Arbor, MI, USA). The meeting covered numerous topics, ranging from patterning and differentiation to the evolution of developmental mechanisms. Despite apparent distinctions between the sessions, many of the talks were broad ranging and most highlighted unifying developmental concepts.

Fertilization and epigenetic regulation of development

After a pollen grain lands on the stigma of a receptive plant, it grows through the transmitting tract towards an ovule, where it penetrates the embryo sac and releases sperm cells. Given that pollen of many different species may land on a stigma, plants have complex mechanisms with which to identify compatible pollen. Ueli Grossniklaus (University of Zurich, Zurich, Switzerland) described a gene that may be involved in such a process. In *feronia* (*fer*) mutants of *Arabidopsis*, sperm cells are not released into the synergid where the *FER* gene is transcribed (Huck et al., 2003). As *FER* encodes a putative receptor kinase, it may represent the first identified part of a gametophyte-gametophyte recognition system. Following double fertilisation, flowering plants develop a diploid embryo and a triploid endosperm, the development of which is repressed prior to fertilization. Ramin Yadegari (University of Arizona, Tucson, AZ, USA) described recent progress towards understanding the epigenetic control of this repression by the Polycomb-group (PcG) proteins FIS1/MEA, FIE and FIS2. Paternal alleles of PcG genes are repressed during early endosperm development, and Yadegari is currently identifying regions of the *FIS2* gene that are required for this negative regulation. Rob Martienssen (Cold Spring Harbor Laboratories, Cold Spring Harbor, NY, USA) accounted for a further a mechanism for the epigenetic regulation of gene expression in plants – via transposable elements – which indicates that the general mechanism of heterochomatin (silent chromatin) regulation is conserved among eukaryotes (Lippman et al., 2004).

Stem cells and patterning: the root and shoot of it

As embryogenesis proceeds, the root and shoot apical meristems are generated. Three talks focussed on the genetic mechanisms responsible for spatially positioning stem cell populations within the developing root or shoot meristem. In the first, Gerd Jurgens (University of Tübingen, Tübingen, Germany) discussed how auxin plays a role in establishing the root meristem. PIN-FORMED (PIN) proteins, which are hypothesized to regulate auxin efflux from cells, are required for the formation of a polar axis during *Arabidopsis* embryogenesis (Geldner et al., 2003). PIN-regulated auxin transport then controls the expression of two transcription factors, *MONOPTEROS* (*MP*) and *BODENLOS*, which specify the basal embryonic pole that develops into the root meristem. Ben Scheres (Utrecht University, Utrecht, The Netherlands) developed this theme to discuss how coordinates along both the proximodistal (PD) and radial axes are interpreted to position the quiescent centre (QC) within the root meristem. In the radial axis, the GRAS-type transcription factor SCARECROW (SCR) is necessary and sufficient for QC specification (Heidstra et al., 2004). However, as the QC occupies only a single cell layer in the PD axis, other factors must influence specification. Using promoter trap lines, Scheres has identified *PLETHORA1* (*PLT1*), which encodes an APETALA2-type transcription factor that is specifically expressed in the QC and stem cells. Double mutants of *plt1* and its duplicate *plt2* display aberrant stem cell populations in the root, and fail to express QC markers. Notably, early spatial restriction of *PLT* expression depends on four PIN genes and later expression requires *MP*. As an auxin-responsive reporter gene construct is expressed normally in *plt* double mutants, PLT probably acts downstream of auxin to establish the PD coordinates for QC specification. Thus, QC specification is mediated by an overlap between auxin and SCR pathways.

Overlapping pathways that specify stem cell populations were also discussed by Steve Clark (University of Michigan, Ann Arbor, MI, USA). He introduced two proteins that are related to the CLAVATA1 (CLV1) receptor kinase. CLV1 acts in a feedback pathway with the homeodomain protein WUSCHEL (WUS) to specify stem cell fate in the shoot apical meristem (SAM) of *Arabidopsis* (Carles and Fletcher, 2003). WUS promotes stem cell fate and CLV3 activity, whereas CLV restricts the *WUS* expression domain and thus the size of the stem cell population. Mutational analysis has shown that the CLV1-related BAM1 and BAM2 proteins act with CLV1 in the SAM. Paradoxically, *clv1* mutants have larger meristems than do wild-type plants, whereas *bam* double mutants have smaller meristems. This observation can be explained if the CLV1 receptor is able to respond to BAM ligands. If so, loss of *bam* receptor function could lead to hyperactivation of CLV1 by its own ligand (CLV3) and by the BAM ligands. As a consequence, CLV1 would 'over'-repress WUS activity and meristem size would be reduced. The dynamics of CLV/WUS interactions were elegantly displayed by Venugopala Reddy (Caltech, Pasadena, CA, USA) using real-time confocal microscopy (Reddy et al., 2004). By using constitutively expressed *WUS* and a *CLV3*-reporter gene construct, Reddy showed that WUS-induced expansion of the *CLV3* expression domain involves a respecification of the peripheral zone of the SAM. An additional player in the WUS pathway was introduced by Cristel Carles (Plant Gene Expression Centre, Albany, CA, USA). The *ULTRAPETALA1* (*ULT1*) gene restricts the *WUS* expression domain in the inflorescence

meristem of *Arabidopsis* in a CLV-independent manner. ULT1 also acts in the AGAMOUS (AG)/WUS pathway that operates in the floral meristem. Previous work has shown that WUS acts with LEAFY to promote AG function in early floral development and that later in floral development AG acts to repress WUS activity, and thus to terminate floral development (Lohmann et al., 2001; Lenhard et al., 2001). Mutant and transgenic analysis suggest that *ULT1*, which encodes a putative transcription factor, is a novel temporal component of AG induction in the centre of the flower and of the subsequent WUS-dependent stem cell termination (Carles et al., 2004).

In addition to maintaining stem cells, the SAM also produces the aerial organs of the plant. The distinction between meristem and organs is a crucial component of the switch from indeterminate to determinate growth. In this context, Patty Springer (University of California, Riverside, CA, USA) discussed the role of *LATERAL ORGAN BOUNDARIES* (*LOB*), a gene whose expression is regulated by the homeodomain proteins SHOOTMERISTEMLESS (STM) and BREVIPEDICELLUS (BP) (Shuai et al., 2002). Interactions between STM and the 3′ region of the *LOB* gene, and between BP and the 5′ region of *LOB*, restrict *LOB* expression to the region between the stem cells and developing organ primordia. Springer has identified two further genes expressed in this region that may interact with LOB to demarcate the meristem/organ primordia boundary.

From boundaries to specialized tissues

Once boundaries are established between meristems and organ primordia, patterning can occur in an organ-specific manner. The developmental decisions that occur during organogenesis were highlighted in talks by Kathy Barton (Carnegie Institute, Washington, DC, USA) and Kay Schneitz (Technical University, Munich, Germany). Barton discussed the regulation of HD ZIP genes such as *PHABULOSA* (*PHB*), the spatial expression of which is restricted to the adaxial side of the leaf primordium, which is crucial for establishing the adaxial/abaxial axis of the leaf (Shuai et al., 2002). The localization of *PHB* mRNA reportedly involves miRNA-induced post-transcriptional gene silencing. Somewhat controversially, Barton proposes an alternative mechanism whereby miRNA binding leads to the methylation of downstream regions of the *PHB* gene and its subsequent transcriptional repression. Interestingly, this explanation is more compatible with the phenotype of dominant gain-of-function *phb* mutants. Schneitz presented an analysis of factors that facilitate differentiation along the PD axis of ovules. Three domains are apparent in this axis: the distal nucellus, the middle chalaza and the basal funiculus. In early development, *WUS* is expressed in the distal domain, yet WUS activity influences integument outgrowth from the chalaza. Therefore, WUS most probably acts non cell-autonomously to specify chalazal and hence integument fate (Sieber et al., 2004).

Within developing organs, several processes specify cell fate, and at least eight talks addressed this issue. In one of the best presentations of the meeting, Adrienne Roeder (University of California, San Diego, CA, USA) discussed fruit development in *Arabidopsis*. The *Arabidopsis* fruit comprises two large seedpod walls known as valves that are joined to the replum at the valve margins. The valve margins form narrow stripes of cells that are specialized for seed dispersal. When the fruit matures, the valve margin cells separate, allowing the valves to detach from the replum and the seeds to be released. Specification of valve margin cell fate is controlled by a group of transcription factors. SHATTERPROOF (SHP) is a MADS domain transcription factor that is initially expressed broadly, but becomes restricted to the valve margin. SHP positively regulates the expression of two basis helix-loop-helix (bHLH) transcription factors, *INDEHISCENT* (*IND*) and *ALCATRAZ* (*ALC*), which are also required for valve margin differentiation (Liljegren et al., 2004). The negative regulation of *SHP* by the homeodomain protein REPLUMLESS (RPL) in the replum and by another MADS factor, FRUITFULL (FUL), in the valves limits *SHP* expression to the valve margin (Roeder et al., 2003). The coordinated action of these transcription factors thereby leads to the formation of the line of specialized valve margin cells precisely at the border between the valve and the replum (see Fig. 1).

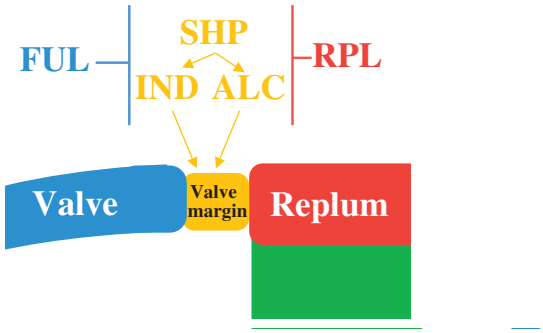

Fig. 1. Summary of genetic interactions occurring at the boundary between valve-valve margin and replum in the developing fruit. The colour of each protein indicates the region in which it is expressed. ALC, ALCATRAZ; FUL, FRUITFULL; IND, INDEHISCENT; RPL, REPLUMLESS; SHP, SHATTERPROOF.

A similar theme is found in the development of the *Arabidopsis* root epidermis, where transcription factors again control the formation of files of cells. The root epidermis comprises longitudinal rows of hair-bearing and hairless cells. The *CAPRICE* (*CPC*) gene is transcribed in developing hairless cells and the Myb-like CPC protein moves to neighbouring cells to promote hair development (Schellmann et al., 2002; Wada et al., 2002). Tetsuya Kurata (RIKEN Institute, Yokohama, Japan) identified a protein that interacts with CPC called FIC (FACTOR INTERACTING WITH CPC). Like *CPC*, *FIC* transcripts are also localized in hairless cells, suggesting that FIC may also act cell non-autonomously, possibly in a complex with CPC. Liam Dolan (John Innes Centre, Norwich, UK) described the function of ROOT HAIR DEFECTIVE6 (RHD6), a bHLH transcription factor that is a possible CPC target. *RHD6* could represent the first example of a gene that facilitates root hair development per se, as opposed to patterning hair versus non-hair cells throughout the organ.

Two further talks discussed the specification of vascular tissue. Vascular traces contain xylem strands, and a fundamental question is how individual xylem cells [tracheary elements (TE)] fit together to form a contiguous tube. Using *Zinnia elegans*, Hiroo Fukuda (University of Tokyo, Tokyo, Japan) showed that cultured cells undergoing xylem differentiation secrete a protein that induces the differentiation

of TEs in neighbouring cells. The secreted protein (xylogen) is induced by auxin and has a polar distribution in the cell. This suggests that during tube formation, developing TEs secrete xylogen and induce neighbouring cells to also develop as TEs. *Arabidopsis* plants lacking xylogen cannot develop contiguous xylem and instead form discontinuous vascular strands (Motose et al., 2004). Further insight into the mechanism of vascular cell differentiation might come from a mutagenesis screen in *Arabidopsis* described by Tim Nelson (Yale University, New Haven, CT, USA). Nelson reported the existence of two mutant classes – those with discontinuous venation and those with parallel, rather than networked, venation.

In the final talks on cell-type differentiation Fred Sack (Ohio State University, Columbus, OH, USA) and Lynn Pillitteri (University of Washington, Seattle, WA, USA) discussed how the patterning of stomata and epidermal pavement cells depends upon the balance between asymmetric cell divisions, symmetric cell divisions and terminal differentiation (Fig. 2). Pillitterri highlighted the role of ERECTA (ER)-like receptor kinases in stomatal patterning and Sack described TOO MANY MOUTHS (TMM), a different type of extracellular receptor (Nadeau and Sack, 2002). The ligand for the TMM receptor may be created through the action of the STOMATAL DENSITY AND DISTRIBUTION (SDD) protease and TMM may act upstream of YODA, a MAPKKK (Bergmann et al., 2004). Later in development, the transcription factors FOUR LIPS and FAMA limit symmetric divisions. Possibly, these transcription factors are targets of the signal transduction cascade defined by ER, TMM, SDD and YODA proteins.

Cell size and cell shape

Cellular differentiation processes modulate not just cell type but also cell size and cell shape. Increase in cell size in plants is accompanied by an increase in the amount of DNA in the nucleus (endoreduplication). Yuki Mizukami (University of California, Berkeley, CA, USA) identified a group of proteins required for endoreduplication in *Arabidopsis*. Fizzy genes control the cell cycle in *Drosophila*. *Arabidopsis* plants that lack the two *FIZZY-RELATED* (FZR) genes have reduced endoreduplication, whereas overexpression of *FZR1* induces endoreduplication in cultured cells. Neither perturbation affects cell-type differentiation, demonstrating that increased DNA content is not essential for cell differentiation.

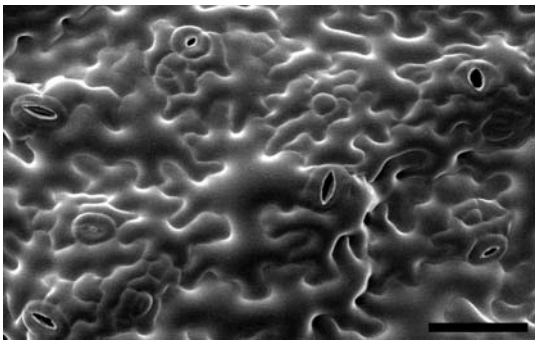

Fig. 2. Cell distribution on the abaxial surface (underside) of an Arabidopsis cotyledon (seed leaf). Stomata are pores that are surrounded by a pair of kidney-shaped guard cells – the pores appear as black voids in this image. The epidermal surface between stomata is occupied by pavement cells with undulating edges. Scale bar: 50 λm.

The cytoskeleton plays an important role in determining cell shape, and this issue was the focus of many talks. Tobias Baskin (University of Massachusetts, Amherst, MA, USA) discussed the roles of cellulose and microtubules in determining cell growth directions. Experiments with low doses of anti-microtubule drugs suggest that the degree of cellular growth anisotropy (the degree to which cell expansion is not uniform, i.e. isometric on all surfaces) is determined by how well aligned cellulose microfibrils are across fields of cells rather than in individual cells, and that microtubules somehow promote the establishment of transcellular cellulose alignments. (Baskin et al., 2004). Both Jie Le (Purdue University, West Lafayette, IN, USA) and David Oppenheimer (University of Florida, Gainesville, FL, USA) discussed the role of the cytoskeleton in the morphogenesis of *Arabidopsis* trichomes – branched epidermal hair cells. Actin filaments form bundles in elongating trichome branches, and the drug-induced depolymerisation of these bundles results in the formation of fat trichomes with short branches that resemble those that form on *distorted* (*dis*) mutants. Four of the DIS genes encode components of the Arp2/3 complex, which nucleates polymerization of actin filaments in vitro. To stimulate actin polymerization, the Arp2/3 complex must be activated. Laurie Smith (University of California, San Diego, CA, USA) described a family of WAVE/Scar-related proteins found in both *Arabidopsis* and maize that can activate the Arp2/3 complex in vitro. These proteins also bind to *BRICK 1* (BRK1), a protein required for localized actin polymerization and for the proper morphogenesis of maize epidermal pavement cell lobes (Frank and Smith, 2002). BRK1 is the plant homolog of a mammalian protein found in a complex with WAVE that regulates WAVE activity. Jie Le reported that two additional DIS genes [*PIROGI* (*PIR*) and *GNARLED* (*GRL*)] encode other components of this WAVE regulatory complex. Together, these observations suggest that a WAVE complex containing PIR, GRL and BRK1 is crucial for the activation of the plant Arp2/3 complex during trichome morphogenesis in *Arabidopsis* and during pavement cell formation in maize. The formation of epidermal pavement cell lobes in *Arabidopsis* involves both F-actin and microtubules, both of which are controlled by ROP-GTPases. So how does ROP-GTPase control two different cytoskeletal systems in the same cell? Zhenbiao Yang (University of California, Riverside, CA, USA) described two ROP INTERACTING PROTEINs (RIC2 and RIC4), one of which controls microfilament assembly and the other microtubule organization. The interaction of ROP with different proteins can coordinate the activity of these two cytoskeletal systems.

Hormonal and environmental influences on development

Several speakers highlighted advances in our understanding of both hormonal and environmental influences on plant development. In the case of hormones, brassinosteroids and auxin were discussed at length. The brassinosteroid signal transduction cascade was discussed by Jianming Li (University of Michigan, Ann Arbor, MI, USA) who presented an

impressive experimentally supported model in which the receptor kinases BRASSINOSTEROID RECEPTOR1 (BRI1) (Li and Chory, 1997) and BRASSINOSTEROID INSENSITIVE 1-ASSOCIATED RECEPTOR KINASE 1 (BAK1) are present in the plasma membrane as monomers until the steroid is present. The binding of brassinosteroid promotes receptor dimerization and its consequent activation. Activated BRI1 then phosphorylates TRANSTHYRETIN-LIKE (TTL), which is similar to the vertebrate thyroid carrier protein (Nam and Li, 2004), and inhibits BRASSINOSTEROID INSENSITIVE2 (BIN2), a GSK3 kinase (Li and Nam, 2002). As a consequence, BIN2 cannot phosphorylate BRASSINAZOLE-RESISTANT1 (BZR1) (Wang et al., 2002) and BRI1-EMS-SUPPRESSOR1 (BES2) (Yin et al., 2002; Zhao et al., 2002). In the non-phosphorylated state, BZR1 and BES2 are stable and are transferred to the nucleus, where they are hypothesized to regulate the transcription of brassinosteroid-responsive genes.

Auxin also controls development by regulating the stability of transcription factors and other proteins. For example, auxin treatment induces the degradation of AUX/IAA proteins through their ubiquitination (Gray et al., 2001). When present, AUX/IAA proteins bind to AUXIN RESPONSE FACTOR (ARF) proteins and prevent the ARFs from regulating the transcription of auxin response genes. In the presence of auxin, AUX/IAA proteins are degraded and ARF proteins promote or repress transcription. Details of how the AUX/IAA proteins feed into the ubiquitin pathway in response to auxin have emerged over the past few years (Kepinski and Leyser, 2002). Essentially, auxin stimulates an interaction between the AUX/IAA proteins and a ubiquitin protein ligase (E3) SCF^{TIR1}. Mark Estelle (Indiana University, Bloomington, IN, USA) described recent data to show that at least three *transport inhibitor response 1* (*TIR1*)-related proteins are involved in auxin signalling as part of E3 ligase complexes. Furthermore, on the basis of assays in a cell-free system, he indicated that auxin interacts directly with the SCF^{TIR} complex or with a tightly associated protein. If true, the hunt for at least one type of auxin receptor may soon be over.

With respect to environmental influences on development, both temperature and light were discussed. Rick Amasino (University of Wisconsin, Madison, WI, USA) discussed temperature effects in the context of vernalization requirements for flowering. Whilst rapid-cycling *Arabidopsis* has no vernalization requirement, winter annuals must be exposed to the cold to gain competence to respond to day length in spring. Cold treatment represses activity of FLOWERING LOCUS C (FLC), a repressor of flowering (Michaels and Amasino, 1999; Sheldon et al., 1999), and this repression is mediated by two distinct methylation events and one deacetylation event (Bastow et al., 2004; Sung and Amasino, 2004). The epigenetic nature of these modifications permits the memory of winter to be perpetuated into spring.

The effect of light was highlighted in three talks. Rob McClung (Dartmouth College, Hanover, NH, USA) and Julin Maloof (University of California, Davis, CA, USA) demonstrated how quantitative trait loci (QTL) analysis can be used to identify genomic regions responsible for natural variation in response to light (Michael et al., 2003; Borevitz et al., 2002; Maloof et al., 2001). Xing Wang Deng (Yale University, New Haven, CT, USA) went on to report that

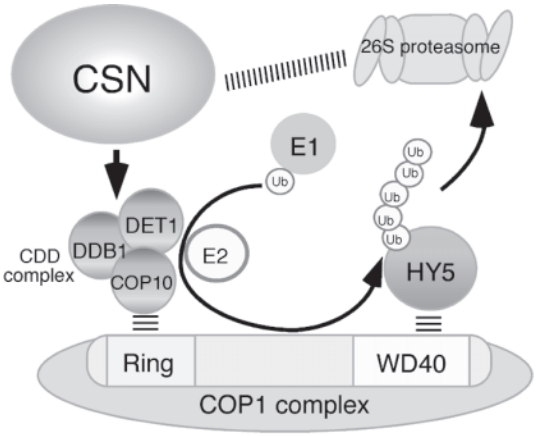

Fig. 3. A model of the functional relationships among COP9 signalosome subunit 5B (CSN), CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1) and DDB1 DET1 COP10 complex (CDD) complexes, in ubiquitin (Ub)-mediated proteolysis. In darkness, CSN, COP1 and CDD complexes work together to promote the ubiquitination of photomorphogenesis-promoting transcription factors such as HY5. The CSN directly interacts with and stabilizes the CDD complex. The CDD complex has the ability to enhance E2 activity. COP10 interacts with the Ring finger domain of COP1. HY5 interacts with the WD40 repeat domain of COP1 and is ubiquitinated by the ubiquitin E3 ligase activity of COP1. Polyubiquitinated HY5 is presumably recognized and degraded by the 26S proteasome. The CSN might regulate the function of the 26S proteasome, CDD complex and COP1 E3 ligase activity. DDB1, DAMAGED DNA BINDING PROTEIN 1B; DET1, DEETIOLATED 1.

function had now been assigned to all of the loci that were initially identified in genetic screens for de-etiolated (DET) and CONSTITUTIVELY PHOTOMORPHOGENIC (COP) mutants (Chory, 1993; Wei and Deng, 1996). In particular, he showed that in addition to the COP9 signalosome (Chamovitz et al., 1996) and the COP1 complex (Saijo et al., 2003; Seo et al., 2003), another multisubunit complex acts to degrade proteins upon exposure to light (see Fig. 3). COP10 interacts with DEETIOLATED1 (DET1) and DDB1 to form a complex that enhances ubiquitin-conjugating enzyme E2 activity (Suzuki et al., 2002; Yanagawa et al., 2004). These observations suggest that the ability to etiolate evolved as a consequence of gaining mechanisms to specifically degrade proteins involved in photomorphogenesis.

Developmental mechanisms that mediate evolutionary change

The modification of developmental mechanisms to mediate evolutionary change was discussed in four talks. Jane Langdale (University of Oxford, Oxford, UK) demonstrated that the genetic pathway required for the switch from indeterminate shoot to determinate leaf growth was conserved in two plant lineages that diverged over 400 million years ago. Kirsten Bomblies (University of Wisconsin, Madison, WI, USA) showed that one of the QTL corresponding to morphological change in ear phyllotaxy from teosinte to maize maps to one of two maize LFY genes. Although *zfl1* and *zfl2* act redundantly in the floral transition (Bomblies et al., 2003), by comparing single mutations in different maize

backgrounds, Bomblies was able to distinguish gene-specific functions. Consistent with the idea that *zfl2* underlies the previously identified QTL, ear number and ear rank are associated more strongly with *zfl2* than with *zfl1*. David Baum (University of Wisconsin, Madison, WI, USA) also discussed the role of LFY in morphological change. He used a transgenic approach to determine whether differences in LFY gene function facilitate a conversion from the ancestral inflorescence flowering phenotype to a rosette-flowering phenotype. Using LFY promoters from rosette-flowering species, he demonstrated that at least partial rosette phenotypes could be induced in transgenic *Arabidopsis*. However, conversion was not complete and differed depending on which donor species was used (Yoon and Baum, 2004). Vivian Irish (Yale University, New Haven, CT, USA) went on to discuss the ancestral and derived roles of the floral homeotic genes *APETALA3* (*AP3*) and *PISTILLATA*. In *Arabidopsis*, the two genes act together to define petals and stamens. Irish has previously reported that the ancestral (paleo) *AP3* gene found in basal eudicots differs from the two genes (eu*AP3* and *TM6*) found in core eudicots (Kramer et al., 1998). As *Arabidopsis* does not have a copy of *TM6*, gene function has not previously been ascribed. Using tomato, Irish showed that whereas eu*AP3* regulates both petal and stamen formation, *TM6* only regulates stamen development. This suggests paleo*AP3* function was restricted to stamen development.

Conclusions

As the meeting concluded, it was apparent that things had moved quickly over the past 2 years and that we are achieving an ever more in-depth understanding of developmental mechanisms in *Arabidopsis*. Over the next 2 years, we expect that the discoveries made in this plant will be translated to other organisms, providing insight into the mechanism by which morphological diversity has been generated in land plants over the past 450 million years.

We apologise to speakers whose results we could not include because of space constraints. We thank M. Yanofsky for Fig. 1, M. Pernas-Ochoa and P. Linstead for Fig. 2, and X. W. Deng for Fig. 3.

References

Baskin, T. I., Beemster, G. T., Judy-March, J. E. and Marga, F. (2004). Disorganization of cortical microtubules stimulates tangential expansion and reduces the uniformity of cellulose microfibril alignment among cells in the root of Arabidopsis. *Plant Physiol.* **135**, 2279-2290.

Bastow, R., Mylne, J. S., Lister, C., Lippman, Z., Martienssen, R. A. and Dean, C. (2004). Vernalization requires epigenetic silencing of *FLC* by histone methylation. *Nature* **427**, 164-167.

Bergmann, D. C., Lukowitz, W. and Somerville, C. R. (2004). Stomatal development and pattern controlled by a MAPKK kinase. *Science* **304**, 1494-1497.

Bomblies, K., Wang, R. L., Ambrose, B. A., Schmidt, R. J., Meeley, R. B. and Doebley, J. (2003). Duplicate *FLORICAULA/LEAFY* homologs *zfl1* and *zfl2* control inflorescence architecture and flower patterning in maize. *Development* **130**, 2385-2395.

Borevitz, J. O., Maloof, J. N., Lutes, J., Dabi, T., Redfern, J. L., Trainer, G. T., Werner, J. D., Asami, T., Berry, C. C., Weigel, D. et al. (2002). Quantitative trait loci controlling light and hormone response in two accessions of *Arabidopsis thaliana*. *Genetics* **160**, 683-696.

Carles, C. C. and Fletcher, J. C. (2003). Shoot apical meristem maintenance: the art of a dynamic balance. *Trends Plant Sci.* **8**, 394-401.

Carles, C. C., Lertpiriyapong, K., Revelle, K. and Fletcher, J. C. (2004). The ULTRAPETALA1 gene functions early in Arabidopsis development to restrict shoot apical meristem activity and acts through WUSCHEL to regulate floral meristem determinacy. *Genetics* **167**, 1893-1903.

Chamovitz, D. A., Wei, N., Osterlund, M. T., von Arnim, A. G., Staub, J. M., Matsui, M. and Deng, X.-W. (1996). The COP9 complex, a novel multisubunit nuclear regulator involved in light control of a plant developmental switch. *Cell* **86**, 115-121.

Chory, J. (1993). Out of darkness: mutants reveal pathways controlling light-regulated development in plants. *Trends Genet.* **9**, 167-172.

Frank, M. J. and Smith, L. G. (2002). A small, novel protein highly conserved in plants and animals promotes the polarized growth and division of maize leaf epidermal cells. *Curr. Biol.* **12**, 849-853.

Geldner, N., Anders, N., Wolters, H., Keicher, J., Kornberger, W., Muller, P., Delbarre, A., Ueda, T., Nakano, A. and Jurgens, G. (2003). The Arabidopsis GNOM ARF-GEF mediates endosomal recycling, auxin transport, and auxin-dependent plant growth. *Cell* **112**, 219-230.

Gray, W. M., Kepinski, S., Rouse, D., Leyser, O. and Estelle, M. (2001). Auxin regulates $SCF^{(TIR1)}$-dependent degradation of AUX/IAA proteins. *Nature* **414**, 271-276.

Heidstra, R., Welch, D. and Scheres, B. (2004). Mosaic analyses using marked activation and deletion clones dissect Arabidopsis SCARECROW action in asymmetric cell division. *Genes Dev.* **18**, 1964-1969.

Huck, N., Moore, J. M., Federer, M. and Grossniklaus, U. (2003). The Arabidopsis mutant *feronia* disrupts the female gametophytic control of pollen tube reception. *Development* **130**, 2149-2159.

Kepinski, S. and Leyser, O. (2002). Ubiquitination and auxin signaling: a degrading story. *Plant Cell* **14**, S81-S95.

Kramer, E. M., Dorit, R. L. and Irish, V. F. (1998). Molecular evolution of genes controlling petal and stamen development: duplication and divergence within the *APETALA3* and *PISTILLATA* MADS-box gene lineages. *Genetics* **149**, 765-783.

Lenhard, M., Bohnert, A., Jurgens, G. and Laux, T. (2001). Termination of stem cell maintenance in Arabidopsis floral meristems by interactions between WUSCHEL and AGAMOUS. *Cell* **105**, 805-814.

Li, J. and Chory, J. (1997). A putative leucine-rich repeat receptor kinase involved in brassinosteroid signal transduction. *Cell* **90**, 929-938.

Li, J. and Nam, K. H. (2002). Regulation of brassinosteroid signaling by a GSK3/SHAGGY-Like kinase. *Science* **295**, 1299-1301.

Liljegren, S. J., Roeder, A. H., Kempin, S. A., Gremski, K., Ostergaard, L., Guimil, S., Reyes, D. K. and Yanofsky, M. F. (2004). Control of fruit patterning in Arabidopsis by INDEHISCENT. *Cell* **116**, 843-853.

Lippman, Z., Gendrel, A. V., Black, M., Vaughn, M. W., Dedhia, N., McCombie, W. R., Lavine, K., Mittal, V., May, B., Kasschau, K. D. et al. (2004). Role of transposable elements in heterochromatin and epigenetic control. *Nature* **430**, 471-476.

Lohmann, J. U., Hong, R. L., Hobe, M., Busch, M. A., Parcy, F., Simon, R. and Weigel, D. (2001). A molecular link between stem cell regulation and floral patterning in Arabidopsis. *Cell* **105**, 793-803.

Maloof, J. N., Borevitz, J. O., Dabi, T., Lutes, J., Nehring, R. B., Redfern, J. L., Trainer, G. T., Wilson, J. M., Asami, T., Berry, C. C. et al. (2001). Natural variation in light sensitivity of Arabidopsis. *Nat. Genet.* **29**, 441-446.

Michael, T. P., Salome, P. A., Yu, H. J., Spencer, T. R., Sharp, E. L., McPeek, M. A., Alonso, J. M., Ecker, J. R. and McClung, C. R. (2003). Enhanced fitness conferred by naturally occurring variation in the circadian clock. *Science* **302**, 1049-1053.

Michaels, S. D. and Amasino, R. M. (1999). *FLOWERING LOCUS C* encodes a novel MADS domain protein that acts as a repressor of flowering. *Plant Cell* **11**, 949-956.

Motose, H., Sugiyama, M. and Fukuda, H. (2004). A proteoglycan mediates inductive interaction during plant vascular development. *Nature* **429**, 873-878.

Nadeau, J. A. and Sack, F. D. (2002). Control of stomatal distribution on the Arabidopsis leaf surface. *Science* **296**, 1697-1700.

Nam, K. H. and Li, J. (2004). The Arabidopsis transthyretin-like protein is a potential substrate of BRASSINOSTEROID-INSENSITIVE 1. *Plant Cell* **16**, 2406-2417.

Reddy, G. V., Heisler, M. G., Ehrhardt, D. W. and Meyerowitz, E. M. (2004). Real-time lineage analysis reveals oriented cell divisions associated with morphogenesis at the shoot apex of *Arabidopsis thaliana*. *Development* **131**, 4225-4237.

Roeder, A. H., Ferrandiz, C. and Yanofsky, M. F. (2003). The role of the REPLUMLESS homeodomain protein in patterning the Arabidopsis fruit. *Curr. Biol.* **13**, 1630-1635.

Saijo, Y., Sullivan, J. A., Wang, H., Yang, J., Shen, Y., Rubio, V., Ma, L., Hoecker, U. and Deng, X. W. (2003). The COP1-SPA1 interaction defines a critical step in phytochrome A-mediated regulation of HY5 activity. *Genes Dev.* **17**, 2642-2647.

Schellmann, S., Schnittger, A., Kirik, V., Wada, T., Okada, K., Beermann, A., Thumfahrt, J., Jurgens, G. and Hulskamp, M. (2002). TRIPTYCHON and CAPRICE mediate lateral inhibition during trichome and root hair patterning in Arabidopsis. *EMBO J.* **21**, 5036-5046.

Seo, H. S., Yang, J. Y., Ishikawa, M., Bolle, C., Ballesteros, M. L. and Chua, N. H. (2003). LAF1 ubiquitination by COP1 controls photomorphogenesis and is stimulated by SPA1. *Nature* **423**, 995-999.

Sheldon, C. C., Burn, J. E., Perez, P. P., Metzger, J., Edwards, J. A., Peacock, W. J. and Dennis, E. S. (1999). The *FLC* MADS box gene: a repressor of flowering in Arabidopsis regulated by vernalization and methylation. *Plant Cell* **11**, 445-548.

Shuai, B., Reynaga-Pena, C. G. and Springer, P. S. (2002). The *LATERAL ORGAN BOUNDARIES* gene defines a novel, plant-specific gene family. *Plant Physiol.* **129**, 747-761.

Sieber, P., Gheyselinck, J., Gross-Hardt, R., Laux, T., Grossniklaus, U. and Schneitz, K. (2004). Pattern formation during early ovule development in Arabidopsis thaliana. *Dev. Biol.* **273**, 321-334.

Sung, S. and Amasino, R. M. (2004). Vernalization in *Arabidopsis thaliana* is mediated by the PHD finger protein VIN3. *Nature* **427**, 159-164.

Suzuki, G., Yanagawa, Y., Kwok, S. F., Matsui, M. and Deng, X. W. (2002). Arabidopsis COP10 is a ubiquitin-conjugating enzyme variant that acts together with COP1 and the COP9 signalosome in repressing photomorphogenesis. *Genes Dev.* **16**, 554-559.

Wada, T., Kurata, T., Tominaga, R., Koshino-Kimura, Y., Tachibana, T., Goto, K., Marks, M. D., Shimura, Y. and Okada, K. (2002). Role of a positive regulator of root hair development, CAPRICE, in Arabidopsis root epidermal cell differentiation. *Development* **129**, 5409-5419.

Wang, Z. Y., Nakano, T., Gendron, J., He, J., Chen, M., Vafeados, D., Yang, Y., Fujioka, S., Yoshida, S., Asami, T. et al. (2002). Nuclear-localized BZR1 mediates brassinosteroid-induced growth and feedback suppression of brassinosteroid biosynthesis. *Dev. Cell* **2**, 505-513.

Wei, N. and Deng, X.-W. (1996). The role of COP/DET/FUS genes in light control of Arabidopsis seedling development. *Plant Physiol.* **112**, 871-878.

Yanagawa, Y., Sullivan, J. A., Komatsu, S., Gusmaroli, G., Suzuki, G., Yin, J., Ishibashi, T., Saijo, Y., Rubio, V., Kimura, S. et al. (2004). Arabidopsis COP10 forms a complex with DDB1 and DET1 in vivo and enhances the activity of ubiquitin conjugating enzymes. *Genes Dev.* **18**, 2172-2181.

Yin, Y., Wang, Z. Y., Mora-Garcia, S., Li, J., Yoshida, S., Asami, T. and Chory, J. (2002). BES1 accumulates in the nucleus in response to brassinosteroids to regulate gene expression and promote stem elongation. *Cell* **109**, 181-191.

Yoon, H. S. and Baum, D. A. (2004). Transgenic study of parallelism in plant morphological evolution. *Proc. Natl. Acad. Sci. USA* **101**, 6524-6529.

Zhao, J., Peng, P., Schmitz, R. J., Decker, A. D., Tax, F. E. and Li, J. (2002). Two putative BIN2 substrates are nuclear components of brassinosteroid signaling. *Plant Physiol.* **130**, 1221-1229.