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Use of *Petunia* to unravel plant meristem functioning

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In the past decade, enormous progress has been made in our understanding of the molecular and genetic control of meristem growth, maintenance and differentiation into plant organs. Several model plants have contributed to our current knowledge of meristem function. Research using *Petunia* has had a substantial share in this progress. Integration of information obtained from this species gives clues about the common and diverged pathways underlying the formation and functioning of plant meristems.

Diversity and conservation in plant architecture

Higher plants display an amazing variation in body plan. Although they are all made up of similar organs (e.g. leaves, stem, petals, stamens and carpels), they vary extensively in the way that these organs are arranged on the plant body. For example, leaves can be arranged along the stem in a spiral (at ~137° angles), alternate (180° angles) or opposite (pairs at 90° angles) pattern. The variation in architecture is most dramatically seen in the inflorescence, the structure that carries the flower(s). In some species, the inflorescence consists of a single flower, whereas other species generate more complex inflorescences with multiple flowers arranged in various patterns. Because distinct plant architectures arose from each other (or from a common ancestor) by evolution, it seems likely that many of the genes that dictate their body architectures are conserved and that the diversification results from alterations in a few of those genes. However, the identity of those genes, how they evolved and how that affected the development of the body plan is still largely unknown.

Analysis of the mechanisms that result in the diversification of plant architecture requires a comparative developmental approach using at least two species with distinct architecture. *Petunia* is ideal for such studies because its body architecture is different from that of other common plant models (e.g. maize, *Antirrhinum* and *Arabidopsis*; Figure 1) and because it lends itself well to molecular genetic studies. The aerial plant body is generated by the continuous development of new organs throughout the life cycle. This requires specialized tissues, the meristems, where pluripotent cells differentiate. Although the basic mechanism of meristem organization is widely conserved among higher plants, species differ in their pattern of branching and timing of meristem termination, which determines to a great extent the architecture of the plant. Here, we review current knowledge about the molecular and genetic control of meristem functioning in important model species and discuss their impact on plant form.

Meristem maintenance

The vegetative shoot apical meristem (SAM) and, in many species, the inflorescence meristem (IM) can generate an unlimited number of leaves or flowers, respectively. This ability resides in a few pluripotent cells at the summit of the meristem. These summit cells produce daughter cells for the generation of various tissues but do not themselves differentiate, hence these summit cells have been termed stem cells (see Glossary) by analogy to animal systems. The mitotic daughters of the stem cells undergo several divisions until they are displaced into the periphery of the meristem and differentiate as lateral organs (i.e. leaves and flowers) or stem.

Genetic analyses in *Arabidopsis* have revealed aspects of regulatory circuits that govern stem cell

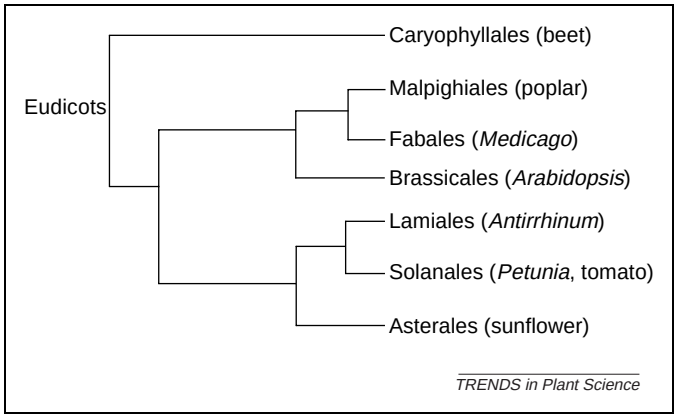

Figure 1. Phylogenetic relationships of major orders of the eudicots. The relationship between the major orders of the eudicots is illustrated in a phylogenetic tree (modified from Refs [53,54]). Examples of representative species for each order are indicated (between brackets). *Petunia* is, from an evolutionary perspective, more related to the model species *Antirrhinum* than to *Arabidopsis*, in spite of the similarity in structure of the inflorescences (raceme) of *Arabidopsis* and *Antirrhinum*. The existence of species with different types of inflorescence structures within many orders indicates that these distinct structures were already present in ancient angiosperms.

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Glossary

ABC floral organ identity genes: the identity of the floral organs is determined in a combinatorial manner by homeotic genes, which mainly belong to the MADS box transcription factor family. In the early 1990s, a model was proposed with three classes of genes (ABC) [64]; this has since been extended to include D and E functions [43,65]. The classes that are relevant here are C (which determines stamen and carpel identity), D (specifying ovule identity) and E (which is essential for proper formation of all organs in the flower).

Acropetal: undergoing development from base to apex; for example, acropetal development of an inflorescence, with flowers arising in a sequence beginning at the base and proceeding towards the apex.

Basipetal: undergoing development from apex to base; in this case, the first branch is formed at the youngest nodes and then additional branches form progressively from older nodes.

Cymose: inflorescence structure in which the inflorescence meristem transforms into a floral meristem, after which a new secondary inflorescence meristem is formed on the flank of the apical dome, resulting in a zig-zag structure (Figure 3).

Determinate inflorescence: terminated by a flower; the inflorescence meristem is transformed into a flower and meristematic activity is lost.

Indeterminate inflorescence: not terminated by a flower; the inflorescence meristem maintains its meristematic activity.

Meristem identity genes: genes that establish the identity of the floral meristem; mutations in these genes lead to inflorescence structures in which flowers are replaced by inflorescences.

Raceme: inflorescence structure with an inflorescence meristem that remains indeterminate and produces floral meristems on its flanks (Figure 3).

Stem cells: undifferentiated cells defined by their abilities of self-renewal and for generating differentiated cells. Plant stem cells are located in meristems, structures where indeterminate growth and differentiation into organs take place.

Sympodial: sympodial branches are those formed where the main axis of growth is by a succession of meristems (as opposed to monopodial growth, in which the main axis of growth is from a single shoot apical meristem).

maintenance [1,2]. These circuits integrate cues from different cellular origins, which might be the meristem itself or the young lateral organ primordia. The undifferentiated condition is promoted mainly by the homeodomain proteins *SHOOTMERISTEMLESS* (*STM*) and *WUSCHEL* (*WUS*), whose loss-of-function mutants fail to maintain a population of stem cells. The *CLAVATA* genes (*CLV1–CLV3*) act antagonistically by inhibiting the proliferation of stem cells in a feedback loop with *WUS* [3,4]. The *CLV1–CLV3*, *STM* and *WUS* genes are all expressed in undifferentiated cells and are thus meristem intrinsic, but not necessarily in the stem cells themselves. Stem cells are specified by positional cues, not by intrinsic properties.

The study of *Petunia* has provided novel mutants with unusual loss-of-meristem phenotypes; their corresponding genes have revealed mechanisms of stem cell maintenance that were not previously anticipated [5,6] (Table 1). *NO APICAL MERISTEM* (*NAM*) [5] and its recently identified putative orthologues from *Arabidopsis CUP-SHAPED COTYLEDON1–CUP-SHAPED COTYLEDON3* (*CUC1–CUC3*) [7–9] are involved in the formation of the SAM and are required for establishing the boundary of the cotyledons. These *CUC* genes are essential for the expression of *STM*, suggesting that *STM* is active downstream in the initiation pathway of the SAM.

Another example of a *Petunia* gene active in the SAM is *HAIRY MERISTEM* (*HAM*); the identification of *HAM* led to the definition of a new clade of GRAS genes whose members might be involved in meristem maintenance [6,10]. The *HAM* gene is expressed in organ primordia and stem provasculature, which suggests the presence of a signalling system to enable differentiating tissues to keep control of meristem perpetuation.

One of the most striking features of *ham* meristems is their differentiation into layers of specialized cell types (epidermis, cortex, vasculature and pith) with a stem-like histology. This pattern of differentiation is distinctly different from that seen in *stm* or *wus* mutants. In *stm* mutants, the SAM differentiates to become incorporated into lateral organs [11]. Both *wus* (*Arabidopsis*) and its orthologous *terminator* (*ter*) mutants (*Petunia*) develop in a characteristic stop-and-go mode, with new defective meristems appearing on flat apices in mutants. If true differentiation was defined as histogenesis, *wus*/*ter* apices would not meet this definition. Ectopic meristems on *wus*/*ter* apices are likely to be intrinsic to this class of mutation, although their origin remains unclear.

In contrast to lateral organ formation, which is an active process that can be disturbed by mutations in auxin transport proteins [12], stem tissue develops by default. Mutant *ham* meristems therefore appear as simple continuations of cellular identity patterns pre-existing in mature tissue. This condition might arise in a manner analogous to the root meristem, in which *HAM* homologues such as *SCARECROW* (*SCR*) are required to prevent stem cells from adopting the fate of their differentiated neighbours [13]. Unlike *SCR*, *HAM* does not fail to specify certain cell types in addition to a loss of stem cell activity but appears to serve a specific role to prevent the default differentiation of apical cells.

Mutant *ham* meristems initially express other meristem genes (*STM* and *WUS*) in normal patterns and maintain this expression until a few days after termination, after which they disappear. These two genes had previously been suggested to provide meristematic potential when ectopically expressed [4,14] but require additional, unknown factors to provide full meristem function [15]. In *ham* meristems, these genes appear to be insufficient to prevent differentiation even at their normal sites of expression. It is possible that *HAM* is one of the factors that function in conjunction with *WUS* and *STM* to provide meristem function.

Based on actual data, two possible models for *HAM* function are given in Figure 2. The default path for the meristem to develop into stem should be actively inhibited by a special set of genes, of which *HAM* would be one. Figure 2a depicts the situation in *ham* meristems in which full differentiation has taken place. *HAM* might emit or relay a signal into the meristem that antagonizes differentiation (Figure 2b). Alternatively, *HAM* might inactivate a differentiation signal emanating from mature tissue (Figure 2c). *HAM* activity gives the apex an undifferentiated condition in which new meristem-specific cellular identities can be established, including stem cell identity (Figure 2d). This suggests that genes such as *WUS*/*TER* and *STM* work convergently rather than functioning downstream of *HAM* in a simple linear sequence [6]. However, it is clear that the cellular patterns of identity and behaviour instructed by *WUS*/*TER* and *STM* cannot materialize in the absence of *HAM*.

Further study of *HAM* in *Petunia* should reveal details of its functioning and test various aspects of the models in

Table 1. *Petunia* genes involved in meristem function and their putative orthologues from *Arabidopsis* and *Antirrhinum*

Petunia gene name	Gene family	Proposed function	*Arabidopsis* orthologue	*Antirrhinum* orthologue	Refs
TER	Homeobox TF	Stem cell maintenance	*WUS*	?	[6,55]
PhSTM	Homeobox TF	Preventing meristem differentiation	*STM*	?	[6,56]
HAM	GRAS TF	Meristem maintenance	?	?	[6]
NAM	NAC TF	Initiating (axillary) meristems and establishing boundaries	*CUC1, CUC2, CUC3*	?	[5,7–9]
DAD1	Carotenoid cleavage dioxygenase	Controls axillary branching	*MAX4*	?	[22,23]
ALF	Orphan TF	Meristem identity	*LFY*	*FLO*	[32,57,58]
PIE7	MADS TF	Meristem identity	*AP1*	*SQUA*	[59,60]
PMADS3	MADS TF	Specification stamen and carpel identity C-function gene	*AG*	*PLE*	[37,41,61]
FBP11	MADS TF	Specification ovule identity D-function gene	*STK*	*DEFH9*	[48,51,62]
FBP2	MADS TF	Specification floral organ identity E-function gene	*SEP*	*DEFH72, DEFH84, DEFH200*	[43,46,63]

Abbreviations: TF, transcription factor; ?, no clear orthologue known.

Figure 2. Production of *ham* phenocopies in *Arabidopsis* will be important for integrating *HAM* into a wider scheme of genes implicated in meristem function. Currently, four *Arabidopsis* GRAS genes have been assigned to the *HAM* clade [10] and combinations of mutations in these genes might be required to obtain a phenotype.

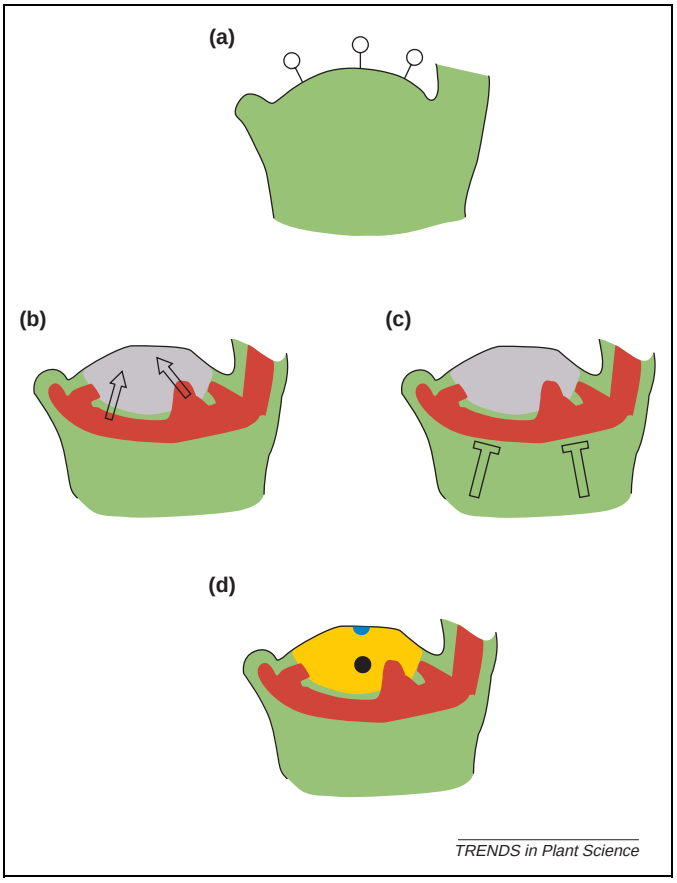

Figure 2. Models for meristem maintenance inferred from *ham* mutants. **(a)** In the absence of *HAM*, the shoot apex is converted into stem and displays full histological differentiation as depicted by, for example, trichome differentiation on the epidermal surface (stick-and-ball structures). **(b)** In the presence of *HAM* (red), a field of undifferentiated cells (grey) is maintained in the apex by a possible signal that emanates from *HAM* expressing cells. **(c)** Alternatively, maintenance of undifferentiated cells is the result of blocking an inductive differentiation signal emanating from mature tissues. **(d)** The *HAM*-induced field of undifferentiated cells can be patterned by factors such as *STM* (yellow), *WUS* (black) and *CLV3* (blue) to generate a functional meristem.

Axillary meristems and branching patterns

During the vegetative growth phase, the SAM generates leaf primordia in a well-defined pattern. In most plants, including *Arabidopsis* and *Petunia*, leaf phyllotaxy is spiral and, within the leaf axils, new (axillary) meristems arise that can grow out into side branches.

The patterns in which axillary meristems are generated and their subsequent outgrowth or dormancy result in the diverse range of branching architectures seen in plants. In *Petunia*, plant architecture is defined by three distinct branching patterns, which are controlled genetically and environmentally [16,17]. Branches formed from axillary meristems in the basal nodes are initiated in an acropetal direction and, after the transition to flowering, branches also form from the axillary meristems of the apical nodes in a basipetal direction. The main axis of *Petunia* growth is continued with a series of sympodial branches. *Arabidopsis* has similar basipetal and acropetal patterns of branch formation, although branching in the acropetal direction is usually only observed in mutants with delayed flowering time [18,19].

The *Petunia dad* (*decreased apical dominance*) mutants are being used to study the control of branching in the basal nodes during vegetative development [16]. Three *DAD* loci have been identified to date and mutations in each of these loci result in plants with an increase in basal branches, as well as a reduction in plant height [17,20,21].

The *DAD* genes are being cloned by using a combination of transposon tagging and a candidate gene approach. *DAD1* has been isolated and is orthologous to the *Arabidopsis* and pea branching genes *MAX4* and *RMS1*, respectively, and is a member of the carotenoid cleavage dioxygenase (CCD) gene family [22,23]. The *in vivo* substrate of the DAD1 or MAX4 enzymes is currently unidentified, but it is known that the *Arabidopsis* MAX4 enzyme (also known as AtCCD8) is capable of cleaving the carotenoid-derived cleavage product of the related AtCCD7 [24].

Grafting studies between wild-type and *dad1-1* plants have shown that a wild-type rootstock can revert *dad1-1* to a near-wild-type branching appearance. Grafting a small wild-type interstock between a *dad1-1* rootstock and scion is also sufficient to revert the plant [20]. These results indicate that a branching signal is produced by the roots of the plant and can be efficiently metabolized by stem tissue. In *Arabidopsis*, the *max4* branching mutant phenotype can also be rescued by grafting the *max4* mutant to a wild-type rootstock [23]. In *Petunia*, the *dad1-1* scion does not revert if mutant roots are allowed to form above the graft union [20], whereas this non-reversion of the mutant phenotype has not been reported for *Arabidopsis*. This result indicates that, although the product of DAD1 might inhibit branching, the substrate of this enzyme must promote branching (and both roles are not mutually exclusive). Although this might not be the case in other plant species, studies geared towards identifying a bioactive compound with an effect on branching should take into account the possible involvement of either the substrate or the product and their potential opposite biological effects. *Petunia* is an ideal system in which to search for new compounds in this process because of the different traits that can be used in bioassays, as well as the ease of isolating extracts from sources such as xylem sap.

Specification of distinct inflorescence structures

At some point during development, the SAM undergoes a transition into an IM, which now produces floral meristems (FMs) rather than leaf primordia. There is astonishing variation in inflorescence architecture between species because of differences in the behaviour of the apical IM [25] (Figure 3).

A major characteristic of racemes such as *Arabidopsis* and *Antirrhinum* is that the IM generates FMs on its flank. FMs are determinate structures that lose meristematic capacity with the formation of carpel primordia in the centre of the flower. However, the IM is truly indeterminate because it never transforms into an FM. Thus, the racemous inflorescence consists of a straight main axis, topped with an apical (indeterminate) IM with many flowers on its flanks.

The floral fate of FMs, and thereby their determinacy, is specified by meristem identity genes such as *LEAFY* (*LFY*) and *APETALA1* (*AP1*), paralogous genes in *Arabidopsis*, and the corresponding homologues *FLORICAULA* (*FLO*) and *SQUAMOSA* (*SQUA*) from *Antirrhinum*, which are expressed in the incipient FMs. In the absence of meristem identity function, FMs develop as IMs and form an (indeterminate) inflorescence shoot, which is the apparent default pathway.

Normally the meristem identity genes are expressed in the incipient FMs located on the flank of the IM but are inactive in the IM at the shoot apex [26]. However, if meristem identity genes are ectopically expressed in the apex, IM identity and indeterminacy are lost (or overruled) and the inflorescence concludes development with the formation of a terminal flower [26–28].

The homologous genes *CENTRORADIALIS* (*CEN*) of *Antirrhinum* and *TERMINAL FLOWER* (*TFL*) from *Arabidopsis* promote the indeterminacy of inflorescence by inhibiting meristem identity gene expression in the apical IM [27,29,30]. Consequently, *CEN* and *TFL* mutations result in loss of indeterminacy and formation of a terminal flower.

In cymose species such as tobacco, tomato and *Petunia*, the apical meristem is determinate and transforms into a flower. In *Petunia* and tomato, the specification of FM fate and determinacy of the apex requires homologues of *LFY*/*FLO*, which are encoded by *ABBERANT LEAF AND FLOWER* (*ALF*) and *FALSIFLORA* (*FAL*), respectively [31,32]. *ALF*, *FAL* and the tobacco homologue *NFL* are expressed in the apex of the inflorescence shoot, whereas LFY and FLO are expressed at the periphery in a raceme, which correlates with the floral fate and determinacy of the inflorescence apex in cymes [31–34]. This change in the expression domain of meristem identity genes seems to be accompanied by changes in the expression pattern and/or the function of *TFL*/*CEN* homologues. For example, analysis of tobacco homologues of *CEN* and *TFL* showed that none of the genes analysed is expressed in the inflorescence apex, and so they cannot inhibit the expression of FM identity genes at that position [35]. In tomato, the *SELF-PRUNING* locus (*SP*) contains a *CEN*/*TFL* homologue that is expressed in the inflorescence apex, but the phenotype of *sp* mutants does not imply misexpression of meristem identity genes in the shoot apex [36].

The key step that distinguishes a solitary flower from a cyme is that in the cyme the development of the shoot continues via the formation of a new ('sympodial') meristem after the apex terminates in a flower. However, whether this new sympodial meristem arises by redifferentiation of non-meristematic cells (in the ideal determinate situation) or from a small set of meristematic cells on the flank of the apex is difficult to distinguish. In *Petunia*, several mutants have been described in which sympodial branching is lost and a single solitary flower is formed. This includes the mutants *extrapetals* (*exp*) [32], *sympodial* (*sym*) [21] and *hermit* (*her*) (R. Koes and R. Castel, unpublished), representing at least two distinct loci. In a *Petunia* meristem identity mutant such as *alf*, FMs are transformed into IMs, resulting in a continuously bifurcating structure (consisting of many sympodial shoots) that carries bracts but no flowers. The introduction of *exp* in an *alf* background (as in *exp alf* double mutants) eliminates the bifurcations and results in a straight shoot that carries only bracts [32]. This indicates that EXP is required for initiating the sympodial meristem rather than for specifying its IM identity and inhibiting FM fate. *EXP* and *HER* have been cloned from tagged mutants and their role in specifying branching pattern is now being studied in further detail (A. Procissi, R. Castel, E. Souer and R. Koes, unpublished).

Overall, these findings show that mutations in single genes are sufficient to change indeterminate meristems into determinate ones, or a cymose into a solitary flower inflorescence, suggesting that the evolution of these distinct structures might have been caused by alterations in a limited number of genes. The ultimate test of this hypothesis would be to change a species with solitary flowers, such as tulip or poppy, into a cyme or raceme.

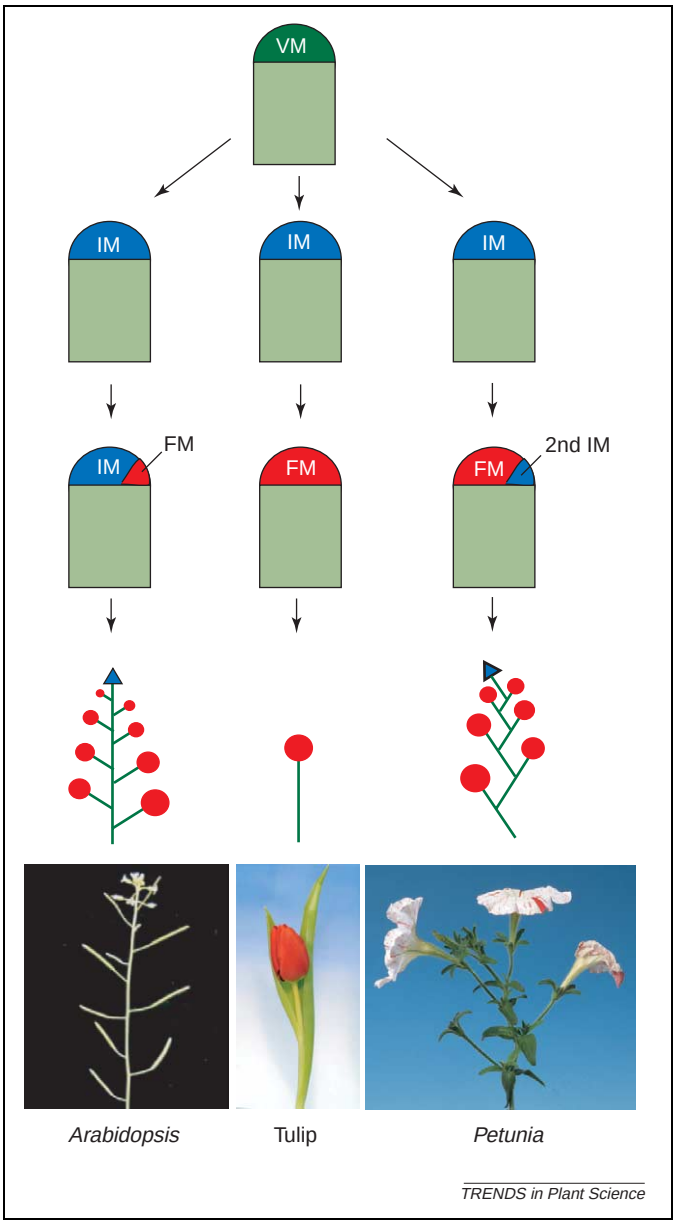

Figure 3. Development of distinct inflorescence architectures. After germination, the shoot apical meristem (SAM) initially has a vegetative nature (green; VM indicates the vegetative meristem) and, upon the switch to flowering, transforms into an inflorescence meristem (IM; blue). In racemes (left), this IM maintains its indeterminate character indefinitely and forms new floral meristems at the periphery, resulting in the formation of a main axis that is topped with the IM and flowers placed on the side, as exemplified by the *Arabidopsis* inflorescence (bottom left). In other species, the apical IM is determinate and undergoes a transition into a flower (middle). Because no meristematic cells remain, only a single flower is formed, as exemplified by the tulip inflorescence (bottom centre). Cymose inflorescences develop as solitary flowers, except that they can form a new (secondary) sympodial meristem that will again terminate with the formation of a flower (right). The reiteration of this program results in a zig-zag structure carrying multiple terminal flowers, as exemplified by the *Petunia* inflorescence (bottom right). Notice that the more vigorous growth of the sympodial shoot tends to push the flower to a more lateral position, weakening the zig-zag shape of the inflorescence.

When does it stop?

FMs are fundamentally different to most plant meristems in that they are determinate structures, producing a fixed number of whorls with floral organs. Although we know a few components of the regulatory circuit that controls FM determinacy, there are still many gaps in our understanding. Termination of the FM requires at least the floral organ identity MADS box gene *AGAMOUS* (*AG*), whose loss-of-function mutation causes indeterminacy in the centre of the flower [37]. *WUS* is likely to be the target of pathways that lead to this termination of the floral meristem. It has recently been proposed that a negative feedback loop involving *WUS*, the FM identity gene *LFY* and the floral organ identity gene *AG* takes place in the FM and is responsible for *WUS* suppression [38,39].

In *Petunia*, *pMADS3* is the most likely candidate to be the orthologue of *AG* because it initiates reproductive organs in gain-of-function mutants [40] and gives rise to altered stamen identity in suppression mutants [41]. Surprisingly, in the suppression mutants, indeterminacy was observed in the third whorl region, resulting in the formation of new floral buds alternating with the petaloid organs in whorl 3. This contrasts with the flowers of *ag* loss-of-function mutants, which display indeterminacy only in the centre of the flower. This suggests that *pMADS3* is responsible for specifying stamen identity and for terminating meristematic activity in the third whorl region of the floral meristem. Probably, *pMADS3* acts redundantly with another class C homeotic gene such as *FBP6* [42] in suppressing *TER* in the centre of the flower.

In addition to the class C homeotic genes, the *Arabidopsis SEPALLATA* genes (*SEP*) [43] and the orthologous *FBP2* and *FBP5* genes [44,45] in *Petunia* appear to be involved in abolishing floral meristem activity. Downregulation of the class E homeotic genes *FBP2* and *FBP5*, and possibly additional homologous genes in a cosuppression mutant [46], affects floral determinacy and leads to a reversion to an indeterminate inflorescence structure in the centre of the flower. A similar reversion was recently observed in quadruple mutants disrupted in all four *SEP* genes [47], demonstrating the conserved role of the *Arabidopsis SEP* genes and the *FBP2* clade in *Petunia* in regulating FM identity and suppression of *WUS*/*TER*.

However, there are still missing links that are responsible for the correct timing of repression of *WUS*/*TER*. Expression of *WUS* persists until stage 6 of flower development, whereas *AG* mRNA can be detected from stage 3 onwards and *SEP*, *FBP2* and *FBP5* genes are expressed even before the induction of the class C organ identity genes (*AG*, *pMADS3*). A candidate for this 'timing' gene is the *Petunia* MADS box ovule identity gene *FBP11* and its paralogue *FBP7* [48], the initiation of expression of which coincides with the downregulation of *TER* (*WUS*). *FBP11* expression appears in the centre of the flower in between the two emerging carpel primordia at the moment when FM identity is lost and placenta formation is initiated (Figure 4). Based on yeast two-and three-hybrid [44,49] and *in vivo* fluorescence resonance energy transfer (FRET) experiments [50], it has been shown that FBP11 can participate in a transcription factor complex together with the E and C class proteins FBP2 and pMADS3, respectively. Together, these data point to a role for the ovule identity gene *FBP11* and its orthologue in *Arabidopsis*, *SEEDSTICK* (*STK*) [51], in the loss of FM activity and the downregulation of *TER* and *WUS* in the flower. Obviously, this hypothesis needs further investigations to prove the relationship between the MADS box

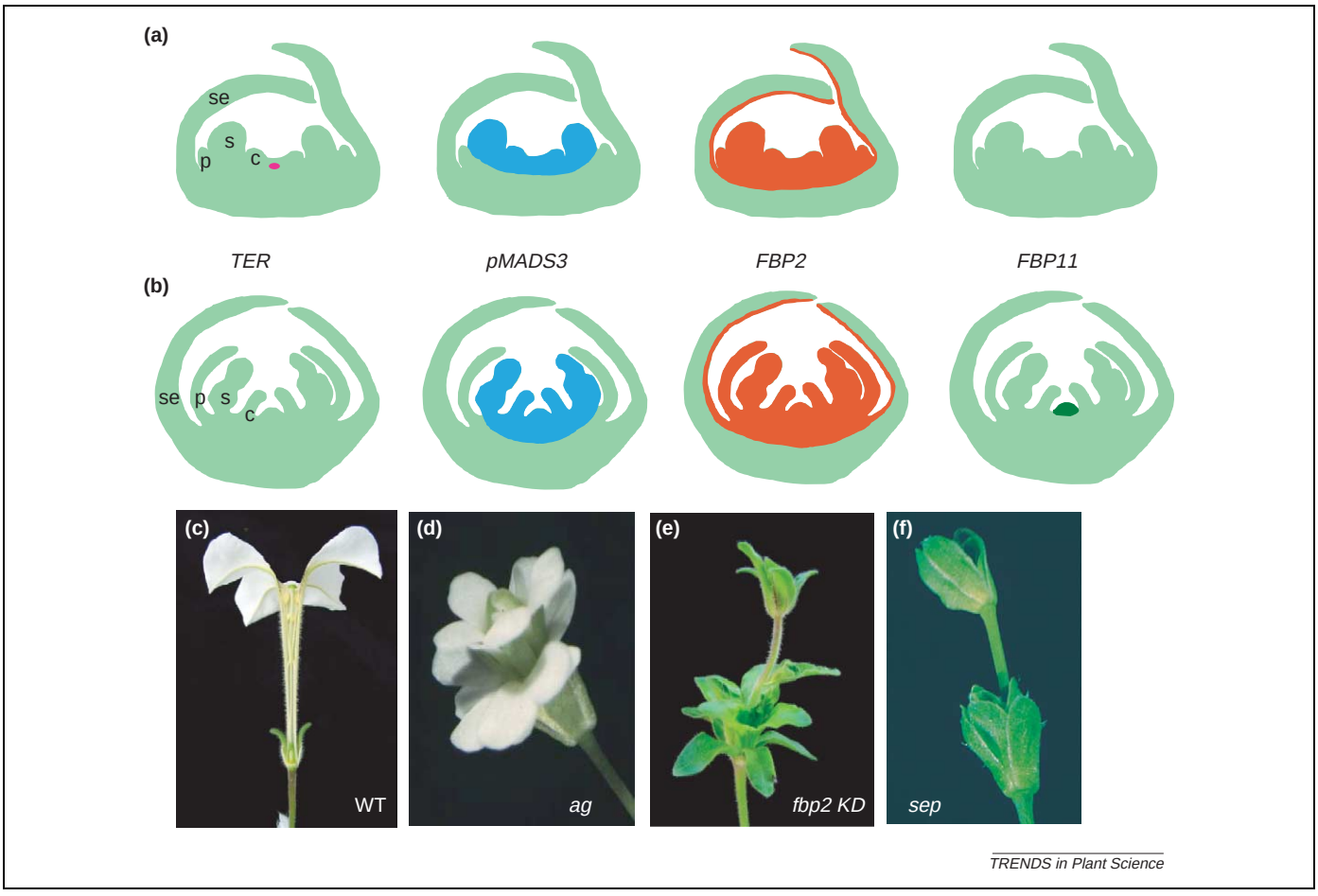

Figure 4. Determinacy in the *Petunia* floral meristem. **(a,b)** Expression patterns of *TER*, *pMADS3*, *FBP2* and *FBP11* (depicted in pink, blue, red and green, respectively) in two consecutive developmental stages [6,40,44,48]. (a) *TER* is still expressed in the centre of the floral meristem at the stage when carpel primordia just appear. (b) At a later developmental stage, the initiation of class D homeotic genes (*FBP11*) in the centre of the flower coincides with the suppression of *WUS*. **(c)** The wild-type (WT) *Petunia* flower is determined and terminates when the full set of organs is formed. Indeterminacy in flowers of **(d)** the *Arabidopsis agamous* mutant [37], **(e)** the *Petunia fbp2 KD* cosuppression mutant [46] and **(f)** the *Arabidopsis sepallata* triple mutant [43]. Abbreviations: c, carpels; s, stamens; se, sepals; p, petals.

transcription factors and the homeobox stem cell regulator.

Prospects

For continuous growth and production of differentiated organs, plants rely on a well-balanced programme of maintaining meristematic activity and cell identity determination. The many studies of this subject using multiple model species have increased our knowledge about the control of plant meristems. Although many of the molecular and genetic control mechanisms are conserved between distinct species, there are several reasons why plant scientists should continue this type of research with different model plants. First, the position and timing of differentiation in meristems from distinct species might differ, as we have seen for the racemous and cyme inflorescence structures for *Arabidopsis* and *Petunia*, respectively. The identity of the genes and the nature of gene products that control these differences are still a mystery. Second, there are many examples of redundancy in genes controlling meristem development. Some are unique in *Petunia* but highly duplicated in *Arabidopsis* and *vice versa*, which favours paralleled approaches in both model plants. The transposon system in *Petunia* with corresponding populations comprising hundreds of thousands of insertions will be a powerful tool for these studies. Screening them for defects in meristem function will almost certainly yield new surprises. Finally, in addition to genetic strategies, methods are becoming available that will enable plant meristems to be analysed using physical, (bio)chemical or hormonal manipulations. A nice example is the microsurgical manipulation of tomato meristems with growth regulators [52]. The relatively large meristems make *Petunia* an ideal system for these kinds of experiments.

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