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REVIEW

Floral Initiation and Inflorescence Architecture: A Comparative View

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• *Background* A huge variety of plant forms can be found in nature. This is particularly noticeable for inflorescences, the region of the plant that contains the flowers. The architecture of the inflorescence depends on its branching pattern and on the relative position where flowers are formed. In model species such as *Arabidopsis thaliana* or *Antirrhinum majus* the key genes that regulate the initiation of flowers have been studied in detail and much is known about how they work. Studies being carried out in other species of higher plants indicate that the homologues of these genes are also key regulators of the development of their reproductive structures. Further, changes in these gene expression patterns and/or function play a crucial role in the generation of different plant architectures.
• *Scope* In this review we aim to present a summarized view on what is known about floral initiation genes in different plants, particularly dicotyledonous species, and aim to emphasize their contribution to plant architecture.

Key words: Plant architecture, inflorescence development, compound inflorescence, floral meristem identity, *LEAFY*, *APETALA1*, *TERMINAL FLOWER1*, legume, *VEG1*, *DET*.

INTRODUCTION: THE ARCHITECTURE OF INFLORESCENCES

A striking feature of plants is the huge variety of forms that can be found in nature. This enormous diversity is due to variation in the shape and size of different plant organs, basically leaves, shoots and flowers (later fruits), and in the proportion of the different kinds of organs and the position where they appear in the plant. The number and arrangement of plant organs are the basis of plant architecture.

Flowers tend to appear clustered in a region of the plant called the inflorescence (Weberling, 1989*a*). Inflorescence form varies enormously among different species and seems to play a determinant role in reproductive success as it has a strong effect on pollination and fruit set (Wyatt, 1982). Whilst particular forms of inflorescences frequently typify some plant families, the same type of inflorescence architecture can also be found in unrelated families, suggesting that adaptive selection has probably played a role in the evolution of inflorescences (Tucker and Grimes, 1999)

All the aerial organs of the plant derive from the shoot apical meristem (SAM). This meristem generates leaves and shoots during the vegetative phase, and in the reproductive phase – after the floral transition – it becomes an inflorescence meristem and flowers are produced. The architecture of the inflorescence depends on its branching pattern and the position of the flowers: on when and where flowers are formed.

Inflorescence types have been classified following several criteria (Weberling, 1989*a*). A main parameter for the classification is whether the shoot apices end in terminal flowers or not. When they do not terminate, the inflorescences are classified as *indeterminate*. A typical example of an indeterminate inflorescence is the *raceme*, present in species such as *Arabidopsis thaliana* or *Antirrhinum majus*. In this type of inflorescence, the apical meristem is able to grow indefinitely, generating a continuous main axis that laterally produces floral meristems (Fig. 1A–C). On the other hand, inflorescences that form terminal flowers are called *determinate*. A classical type of determinate inflorescence is the *cyme*. Cymose inflorescences lack a main axis: the main shoot terminates in a flower, while growth continues through lateral axes produced below the terminal flower (Fig. 1D–F). These lateral axes again form terminal flowers and this process is reiterated several times. Data on the developmental control of cymose inflorescences is available for several species such as *Silene latifolia* or tobacco (*Nicotiana tabacum*; Fig. 1D, E). A variation of the cymose pattern is found, for example, in tomato (*Solanum lycopersicum*; Fig. 1F); the inflorescence of this species is also a cyme but, in this case, after the main axis generates the terminal inflorescence, a new axis of growth develops from an axillary meristem that produces a certain number of leaves before again terminating in an inflorescence. This process repeats indefinitely, generating a plant with an apparently continuous growing axis in which the production of leaves and 'lateral inflorescences' alternates. This kind of plant architecture is called a *sympodium*. Finally, as pointed out in an elegant modelling analysis of inflorescence development (Prusinkiewicz *et al.*, 2007), a third main kind of inflorescence architecture, also determinate, is the *panicle* (Fig. 1G). In contrast to the cyme, in this type of inflorescence a clear main shoot axis exists but this is terminated by a flower, as also occurs in the series of lateral branches produced by the main shoot.

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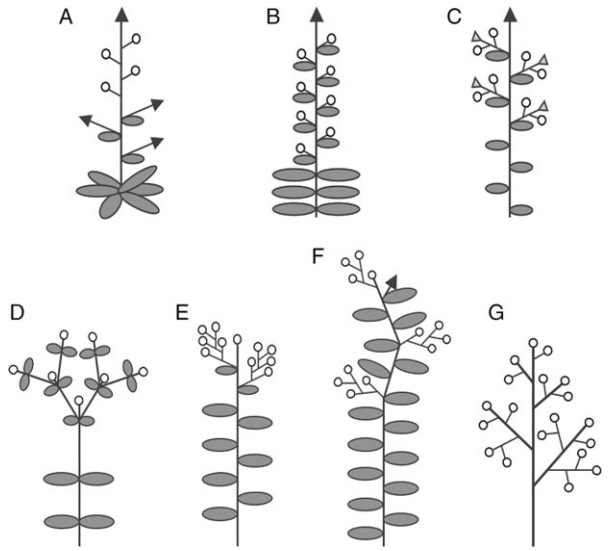

FIG. 1. Diagrams of different types of inflorescences. (A–C) Indeterminate inflorescences: (A) the simple raceme of *Arabidopsis thaliana* and (B) *Antirrhinum majus*, and (C) the compound raceme of pea. (D–G) Determinate inflorescences: (D) the dichasium of *Silene latifolia*; (E) the tobacco cyme; (F) the sympodium of tomato; and (G) a panicle. Open circles represent flowers and arrows represent indeterminate shoots. Grey triangles in (C) represent stubs.

Inflorescences are also classified according to the complexity of their branching. Those inflorescences where flowers are directly formed from the main axis are called *simple* inflorescences, while *compound* inflorescences are those where flowers are formed from secondary or higher-order branches. An example of a compound inflorescence is the *compound* or *double raceme* present in many Leguminosae species, such as pea (*Pisum sativum*), *Medicago truncatula* or *Lotus japonicus* (Fig. 1C). The inflorescences of arabidopsis and antirrhinum are *simple racemes* (Fig. 1A, B).

Although the evolution of inflorescences is poorly understood, it is generally accepted that the most primitive inflorescences would have had terminal flowers. This, in part, derives from the idea that the flower is a specialized shoot, and the transition of a vegetative apex to a flower would be direct in a primitive inflorescence. As discussed by Tucker and Grimes (1999), the first authors speculating about inflorescence evolution favoured the idea that a solitary terminal flower would be the ancestral inflorescence form (Parkin, 1914); this supported the idea of woody trees, such as those of Magnoliaceae, being among the most primitive families. However, the primitiveness of the Magnolia type of flower has been challenged by several authors, such as Stebbins (1974), based on questions such as the high complexity of its vasculature, and a more recent view is that the ancestral angiosperms would have had simple cymose inflorescences.

As explained above, the architecture of the inflorescence depends on which meristems give rise to shoots and which to flowers (Coen and Nugent, 1994). The genetic control of the specification of floral meristems has largely been studied in model species, mainly in antirrhinum and arabidopsis, and the main factors have been identified and a lot of information about how they work is available.

In recent years, the homologues of these and other genes with related functions have been identified and studied in many other plant species. These studies suggest that the functioning of the genetic network controlling the initiation of flowers is largely conserved among flowering plants, with key differences often relating to the different inflorescence architecture of each species. In this review we aim to present a summarized view on what is known about floral initiation genes in different species, and we try to emphasize their role in directing plant architecture.

CONTROL OF FLORAL INITIATION: HOW IT WORKS IN ARABIDOPSIS

As for many genetic processes in plants, the genetic control of floral initiation is best known in the model plant arabidopsis. However, the aim of this article is not to describe in detail how the specification of floral meristems is controlled in arabidopsis, a question that has been treated in several excellent reviews (Jack, 2004; Vijayraghavan *et al.*, 2005; Blázquez *et al.*, 2006), but to try to describe and compare what is known about the genes controlling this process in other species. Therefore, we will briefly introduce the key elements of the genetic network in arabidopsis as a basis for the comparison.

In arabidopsis, during the vegetative phase the SAM produces on its flanks vegetative primordia that will form leaves with shoot meristems in their axils. Upon transition to the reproductive phase, the SAM becomes an inflorescence meristem (IM) and the new lateral primordia produced after that point develop as floral meristems (FM). Therefore, with the floral transition the fate of these lateral primordia has to be reprogrammed so that they acquire the identity of floral meristems.

In arabidopsis, the acquisition of floral meristem identity (FMI) by these primordia is controlled by the interaction of positive and negative regulators. Although several other genes have also been shown to play important roles in the regulation of floral meristem identity in arabidopsis, we will concentrate on *LEAFY* (*LFY*), *APETALA1* (*AP1*) and *TERMINAL FLOWER1* (*TFL1*). These genes seem to form the backbone of the network and, consequently, they are the ones whose role in the process has been best analysed in arabidopsis and whose homologues have been studied most in many other species.

LEAFY

The *LFY* gene is required for the specification of FMI in arabidopsis. This is clearly deduced from the phenotype of *lfy* mutant plants, where the flowers are replaced by structures with shoot characteristics (Fig. 2A; Schultz and Haughn, 1991; Huala and Sussex, 1992; Weigel *et al.*, 1992). The shoot character of the *lfy* 'flowers' is more marked in the first positions in the inflorescence, while structures formed in more apical positions progressively acquire an increasing degree of floral identity due to independent activation of other floral meristem identity genes

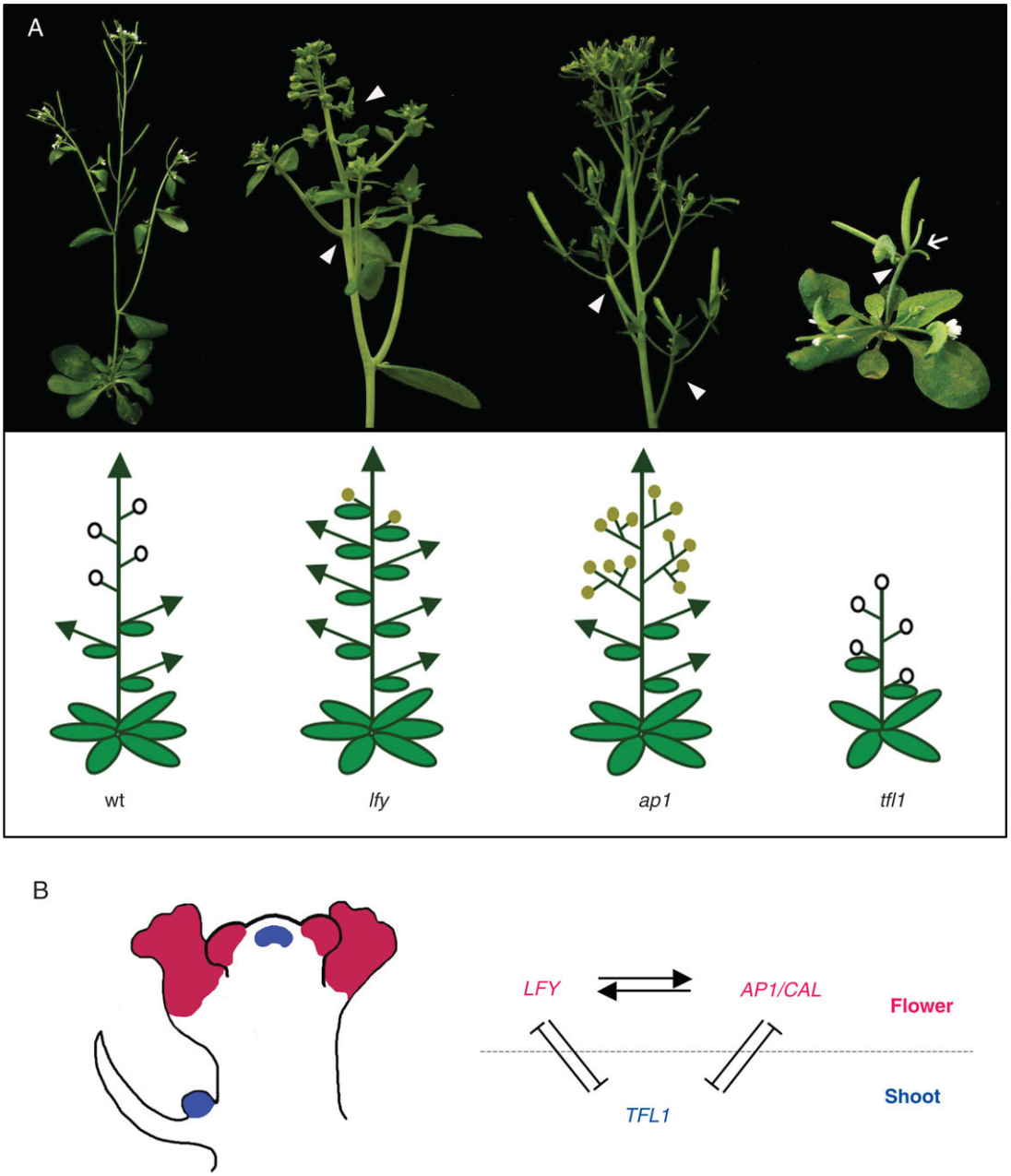

FIG. 2. Meristem identity genes in arabidopsis. (A) Inflorescence of the wild type (wt) and of *lfy*, *ap1* and *tfl1* mutants. In the inflorescences of *lfy* and *ap1*, flowers (open circles) are replaced by structures with shoot characteristics (indicated by arrowheads in the photographs), while in the *tfl1* mutant solitary flowers replace shoots in the axils of cauline leaves (arrowheads). The inflorescences of the wild type, *lfy* and *ap1* show indeterminate growth but the inflorescence of *tfl1* is determinate and forms a terminal flower (arrow in the photograph). Filled circles in the diagrams represent abnormal flowers with shoot traits. (B) Complementary expression of *TFL1* (blue) and *LFY/AP1/CAL* (red) genes in the arabidopsis inflorescence shoot apex. While *LFY* and *AP1/CAL* specify floral identity, *TFL1* is required to maintain the inflorescence identity of all shoot meristems.

such as *AP1* (Huala and Sussex, 1992; Bowman *et al.*, 1993). Another aspect of the *lfy* phenotype is that while in wild-type the flowers are bractless (no subtending leaf; Fig. 2A) many of the *lfy* transformed 'flowers' have bracts, indicating an additional role for *LFY* in bract suppression during the inflorescence phase (Schultz and Haughn, 1991).

LFY encodes a transcription factor that so far has been only found in the plant kingdom (Maizel *et al.*, 2005). In contrast to most other types of transcription factors, *LFY* does not belong to a multigene family. Arabidopsis and most angiosperms contain only one *LFY* gene. Consistent with the phenotype of the mutant, *LFY* is strongly expressed throughout the young floral meristems from the earliest stages of development (Fig. 2B; Weigel *et al.*, 1992). In fact, upregulation of *LFY* in these meristems is crucial for them to acquire floral identity, as it activates the expression of *AP1* and the floral meristem identity genes (Parcy *et al.*, 1998).

LFY expression is not absolutely confined to floral tissues. Expression can also be detected at low levels in leaf primordia during the vegetative phase, and gradually increases until the floral transition (Blázquez *et al.*, 1997; Hempel *et al.*, 1997). The actual level of *LFY* expression in the apex is considered to be a critical parameter that determines the time point at which the floral transition takes place (Blázquez *et al.*, 1997). *LFY* seems to act as an integrator of the pathways controlling flowering time and the initiation of floral meristems (Blázquez and Weigel, 2000; Parcy, 2005). In fact, *lfy* mutants are slightly delayed in the vegetative-to-inflorescence transition (Blázquez *et al.*, 1997).

In agreement with its proposed roles in floral initiation, constitutive expression of *LFY* in arabidopsis causes early flowering and the transformation of all shoots into flowers, indicating that *LFY* is not only necessary, but also sufficient to confer floral identity to emerging shoot meristems (Weigel and Nilsson, 1995).

APETALA 1

AP1 is the other main promoter of floral meristem identity. The *ap1* mutants show defects in FMI and defects in the identity of the floral organs of whorls 1 and 2. The flowers of *ap1* mutants do not have petals and produce bract-like organs instead of sepals. In the axils of those first-whorl organs, new floral meristems are produced that reiterate this pattern, generating 'branched flowers' (Fig. 2A; Irish and Sussex, 1990; Bowman *et al.*, 1993).

AP1 also encodes a transcription factor but, in contrast to *LFY*, it belongs to a large multigene family, the MADS-box gene family (Mandel *et al.*, 1992). Similarly to *LFY*, it is expressed throughout young floral meristems, shortly after the onset of *LFY* expression in these meristems (Fig 2; Mandel *et al.*, 1992). In fact, *AP1* (as well as *CAL*; see below) is directly activated by *LFY* (Wagner *et al.*, 1999). The phenotype of plants constitutively expressing *AP1* is also consistent with its role in floral meristem identity: 35S::AP1 plants are early flowering and show shoot-to-flower conversions, a phenotype similar to that of *tfl1* mutants and 35S::LFY transgenics (Mandel and Yanofsky, 1995).

CAULIFLOWER (*CAL*), another MADS-box gene highly related in its sequence to *AP1* and with a similar expression pattern, is partially redundant to *AP1* in FMI specification in arabidopsis. Single *cal* mutants show a wild-type phenotype, but simultaneous loss of *AP1* and *CAL* causes a complete transformation of floral meristems into inflorescence-like meristems, which give rise to new inflorescence-like meristems; this pattern reiterates an indefinite number of times to form structures similar to cauliflower heads (Bowman *et al.*, 1993; Mandel and Yanofsky, 1995). As expected, constitutive expression of *CAL* causes a similar, though weaker, phenotype to that of 35S::AP1 plants (Savidge, 1996; Liljegren *et al.*, 1999). The redundancy of *AP1*/*CAL* in specifying floral meristem identity has only been documented in arabidopsis and species from the Brassicacea family. This is consistent with the results of phylogenetic studies showing that *AP1* and *CAL* derive from a recent duplication event, found only within the Brassicaea (Lawton-Rauh *et al.*, 1999; Lowman and Purugganan, 1999).

TERMINAL FLOWER 1

The role played by *TFL1* in floral initiation is opposite to that of *LFY* and *AP1*. In *tfl1* mutants the shoot meristems are converted into floral meristems: cauline leaves subtend solitary flowers, rather than shoots, and inflorescence shoots are converted into terminal flowers (Fig. 2A; Shannon and Meeks-Wagner, 1991; Alvarez *et al.*, 1992; Schultz and Haughn, 1993). Therefore, while *LFY* and *AP1* specify floral meristem identity, *TFL1* would specify shoot identity. Mutations in *TFL1* also cause early flowering, indicating that *TFL1* also acts as a repressor of flowering (Shannon and Meeks-Wagner, 1991; Schultz and Haughn, 1993).

Constitutive expression of *TFL1* driven by the 35S promoter causes a great extension of all developmental phases (Ratcliffe *et al.*, 1998). The 35S::TFL1 plants produce an enlarged vegetative rosette with a high number of leaves and a long inflorescence stem, with many lateral branches, which eventually forms normal flowers. The phenotype of 35S::TFL1 plants led to the proposal that *TFL1* acts by retarding the phase transitions at the shoot apex. According to this view, the production of axillary and terminal flowers in *tfl1* would be the consequence of the mutant shoot meristems progressing through the phases much faster than the wild type. In this situation, these meristems would make the transition from inflorescence to floral, a phase that would not be reached by the wild-type shoot meristems under normal conditions.

TFL1 is strongly expressed in the centre of the main and lateral shoot inflorescence meristems, not in the floral meristems. This expression pattern is complementary to that of *LFY* and *AP1*, which are present in floral but not in inflorescence meristems (Fig 2B). Action of *TFL1* in the inflorescence apex is pivotal to its function, as a main role of *TFL1* is to prevent these meristems from assuming the floral identity by inhibiting the expression of FMI genes. Thus, in *tfl1* mutants *LFY* and *AP1* expression invades the inflorescence meristems, which are then converted into flowers (Weigel *et al.*, 1992; Bradley *et al.*, 1997).

Conversely, several pieces of evidence suggest that *LFY* and *AP1* prevent *TFL1* expression in floral meristems (Liljegren *et al.*, 1999; Ratcliffe *et al.*, 1999; Ferrándiz *et al.*, 2000), although it is not clear whether LFY or AP1 act as direct repressors of *TFL1* (Parcy *et al.*, 2002). Correlating with its function in repressing flowering, *TFL1* is also expressed, although at a lower level, in the shoot vegetative meristem. Upregulation of *TFL1* expression in the shoot apical meristem temporally coincides with commitment to flowering, representing a clear early marker for the floral transition (Bradley *et al.*, 1997).

In contrast to *LFY* and *AP1*, *TFL1* does not encode a transcription factor. TFL1 is homologous to phosphatidylethanolamine binding proteins (PEBPs; Bradley *et al.*, 1997; Ohshima *et al.*, 1997), a wide group of proteins also found in animals, yeast and bacteria, that play diverse roles related to signalling pathways controlling growth

and differentiation (Yeung *et al.*, 1999; Hengst *et al.*, 2001; Chautard *et al.*, 2004). *TFL1* belongs to a small gene family (Mimida *et al.*, 2001), one of whose members, *FLOWERING LOCUS T* (*FT*), is also a regulator of flowering time. Opposite to *TFL1*, mutations in *FT* cause late flowering and 35S::FT plants show a phenotype similar to that of *tfl1* mutants (Kardailsky *et al.*, 1999; Kobayashi *et al.*, 1999). The mechanism of action of TFL1 has not been elucidated yet, but recent studies indicate that its homologue FT promotes flowering by acting at the nucleus, as part of a complex with the bZIP transcription factor FD (Abe *et al.*, 2005; Wigge *et al.*, 2005). TFL1 also can bind to bZIP factors. The structure of the TFL1 and FT proteins were recently resolved and are very similar (Ahn *et al.*, 2006). This is highlighted by swapping discrete domains among these proteins, as TFL1 can be converted into FT and vice versa, suggesting that the biochemical function of both proteins is very similar and that differences in their functions could be due to differential binding to interactors (Hanzawa *et al.*, 2005; Ahn *et al.*, 2006).

Other FMI genes, other species

Although *LFY*, *AP1* and *TFL1* are considered to be major regulators of floral initiation, the picture is, of course, not quite so simple and several other genes have also been shown to play important roles in the control of this process in arabidopsis. Among them are, for example, the MADS-box gene *FRUITFULL* (*FUL*), highly related in sequence to *AP1*, which is required for the initiation of the flowers that are eventually formed by the proliferating inflorescence meristems of the double *ap1 cal* mutant (Gu *et al.*, 1998; Ferrándiz *et al.*, 2000), and *AGL24*, which has been implicated in the upregulation of *LFY* expression (Yu *et al.*, 2002). Other examples are the genes *APETALA2* (*AP2*) and *UNUSUAL FLORAL ORGANS* (*UFO*) whose mutations enhance the meristem defects of *ap1* or *lfy* mutants, respectively (Ingram *et al.*, 1995; Okamuro *et al.*, 1997).

Comparative studies carried out on FMI genes in other species, however, have been mostly focused on homologues of *LFY*, *AP1* and *TFL1*. In the following sections we will try to summarize what is known about the homologues of these and other related genes in different species. We will emphasize what changes in function and/or expression have occurred and the possible effects of these changes in the generation of different plant architectures. Although occasionally monocotyledonous species will also be mentioned, for simplicity we will focus on eudicot species. Excellent reviews on the genetics of monocotyledonous inflorescence development have recently been published elsewhere (Bommert *et al.*, 2005; Kellogg, 2007).

FMI PROMOTERS: HOMOLOGUES OF *LFY* AND *AP1*

LFY and *AP1* are the main activators of the cascade of genes initiating floral development. In the last decade, important efforts have been made in order to understand the function and evolution of both factors. *LFY* is present in all land plants analysed, which have evolved for at least 400 million years. There is no doubting its key role in flower meristem identity acquisition in angiosperms. However, the ancestral function of *LFY* and its evolution is far from being clear. *LFY* homologues have been isolated from distant species, such as the moss *Physcomitrella patens* and different species of ferns and gymnosperms. The LFY proteins have low rates of amino acid substitutions and have been used in the phylogenetic analysis of seed-plant relationships (Frolich and Parker, 2000). In a different study, Maizel *et al.* (2005) investigated the functionality of different *LFY* homologues, representing the different taxa from the mosses to angiosperms, by testing their ability to complement the arabidopsis *lfy* mutant. The degree of complementation of the *lfy* mutant phenotype correlated with the taxonomic distance from arabidopsis. *PpLFY* (from *Physcomitrella patens*) was unable to complement the *lfy* mutant, while the homologues of ferns and gymnosperms partially complemented the mutation, and the angiosperm homologues fully complemented it. The authors also studied the ability to activate known LFY targets by transcriptional profiling. A major conclusion of these analyses was that the ability of *LFY* homologues to activate *AP1* is restricted to flowering plants.

The results of these experiments agree with a progressive functional divergence of *LFY* from moss to angiosperms. For example, the moss *P. patens* contains two *LFY* homologues, *PpLFY1/2*, and these are expressed in the main and lateral apices, in the developing archegonium, but not in the antheridium (Tanahashi *et al.*, 2005). The disruption of both *PpFLY* genes affects the first zygotic division, suggesting an important role of *PpLFY* in this process, a function that widely diverges from that described in angiosperm species. Gymnosperm species also have two *LFY* homologues, and both are involved in the development of reproductive tissues. In *Pinus radiata*, for example, one of the *LFY* homologues, *NEEDLY*, is expressed at high levels in female reproductive meristems (Mouradov *et al.*, 1998), while the expression of the second homologue, *PRFLL*, is detected in buds and male cones (Mellerowicz *et al.*, 1998). The presence of these two paralogous genes, with expression in different reproductive tissues, together with the analysis of *LFY* homologues from different taxa has led to the proposal of the 'mostly male' theory for the origin of the flower (Frolich and Parker, 2000). This theory proposes that a duplication occurred before the separation of flowering plants that gave rise to the *LFY* and *NEEDLY* clades. Angiosperm species lost the *NEEDLY* gene and the theory proposes that *LFY* would have been recruited to specify female reproductive organs in addition to male reproductive tissues. In this way, the flower would have arisen by the development of ectopic female structures in a *LFY*-expressing male reproductive shoot.

LFY could have evolved from a different, broader, function in more distant species before recruitment in flowering plants for the acquisition of the floral fate. Expression of *LFY* in tissues other than reproductive meristems such as leaves, tendrils or vegetative meristems (as will be discussed below) could be a remnant of this broader function that has been retained in certain cases.

In contrast with the presence of *LFY* orthologues in all land plants, *AP1* orthologues have only been found in angiosperm species. Arabidopsis has two additional genes, *FUL* and *CAL*, which have high sequence homology with *AP1* and share functions in floral meristem identity specification. *AP1* and *FUL* belong to different gene clades, which were generated as the result of a duplication event at the base of the core eudicots (Litt and Irish, 2003). The fact that both genes belong to the MADS-box family of transcription factors and that they present high sequence homology frequently makes it difficult to clearly ascribe the homologues isolated from other species to one of the two clades. The study of phylogenetic relationships between *AP1* and *FUL* homologues from a variety of angiosperm species has led to the identification of specific C-terminal motifs characteristic of each clade. It has been suggested that this duplication together with the appearance of a new C-terminal motif in the AP1 clade contributed to fix the floral structure observed in core eudicots (Litt and Irish, 2003).

LFY and *AP1* are key regulators of flower and inflorescence development. For that reason, many groups have become interested in the comparative study of their function in different species. Such studies are helping us to understand how diversity in plant architecture has been generated. Below, we discuss significant examples of homologues that have been studied in some detail in different dicot species. Relevant data from these species, and from others that we have been not able to describe due to space limitations, are summarized in Table 1.

LFY/AP1 *in herbaceous species with indeterminate inflorescences*

Antirrhinum majus. Antirrhinum majus is a euasterid from the order Lamiales (Fig. 3) and, with arabidopsis, was a key model species for the initial studies on the genetic control of inflorescence and flower development. In fact, the first member of the *LFY* gene family to be isolated and characterised was *FLORICAULA* (*FLO*) from antirrhinum (Coen *et al.*, 1990) and the isolation of *AP1* and its antirrrhinum homologue *SQUAMOSA* (*SQUA*) was reported almost simultaneously (Huijser *et al.*, 1992; Mandel *et al.*, 1992). The architecture of both species is very similar, both having inflorescences that are simple racemes. However, in antirrhinum all the stem internodes elongate during the vegetative phase, whilst in arabidopsis these internodes remain compressed, forming a rosette. In addition, in antirrhinum all the flowers are subtended by a bract, while the arabidopsis flowers are bractless (Fig. 1).

Correlating with similar inflorescence architectures, *FLO* and *SQUA* seem to work in a very similar way in antirrhinum as do their arabidopsis counterparts. *FLO* and *SQUA* essentially exhibit the same expression patterns as *LFY* and *AP1*, respectively. In addition, the phenotype of the *flo* and *squa* mutants also indicates that the functions of the anthirrhinum genes are similar to their arabidopsis homologues (Coen *et al.*, 1990; Huijser *et al.*, 1992). As in *lfy* mutants, *flo* mutants exhibit conversion of flowers into inflorescences, confirming the role of *FLO* in floral meristem identity. One notable difference is that *LFY* is also involved in bract suppression while *FLO* does not have this function.

Mutations in *SQUA* also cause conversion of flowers into shoots. However, while in the *ap1* mutant these shoots consist of branched flowers, lacking petals but bearing normal stamens and carpels, the FMs of the *squa* mutant are replaced by vegetative shoots that only rarely produce flowers (Huijser *et al.*, 1992). The weaker inflorescence phenotype of the *ap1* mutant in comparison to *squa* can probably be explained by the redundant activity of the *AP1* paralogue *CAL*, a gene possibly only present in Brassicaceae (Lawton-Rauh *et al.*, 1999; Lowman and Purugganan, 1999).

Fabaceae. Many species from the large Fabaceae family, also known as Leguminosae (Fig. 3), have compound double racemes (Weberling, 1989*a*, *b*). After floral transition, the SAM of these legume species becomes a primary inflorescence meristem (I1) that rather than producing flowers, generates second-order inflorescence meristems (I2) that produce the flowers. These I2 usually produce a certain number of flowers, depending on the species, before they are consumed in forming a rudimentary stub (Fig. 1C; Singer *et al.*, 1999). This generates a compound raceme architecture where the main axis, rather than subtending individual flowers, subtends small racemes. The lateral secondary inflorescences of these legume species share morphological features with the simple racemes of arabidopsis or antirrhinum, in the sense that both consist of a main axis that laterally produces flowers (from 1–2 flowers in the case of most pea cultivars, to many more as, for example, in some *Trifolium* species) and do not differentiate into a terminal flower. As we will see, the analysis of the legume *LFY* and *AP1* homologues confirms that they work as functionally equivalent structures.

Homologues of *LFY* and *AP1* have been isolated and characterized from several model legume species (Hecht *et al.*, 2005; Domoney *et al.*, 2006) and a general conclusion is that, in spite of the differences between the inflorescence of legumes and arabidopsis, these genes play similar functions in the legume lateral secondary inflorescences as do *LFY* and *AP1* in the arabidopsis inflorescence. The pea *LFY* homologue, *UNIFOLIATA* (*UNI*), although with a wider expression pattern than its arabidopsis homologue, is expressed in floral meristems and its mutations cause flower-to-inflorescence conversions (Fig. 4; Hofer *et al.*, 1997). A similar expression pattern and mutant phenotype have also been described for the *Lotus japonicus LFY* homologue, *LjLFY* (Dong *et al.*, 2005). On the other hand, the pea *AP1* homologue, *PROLIFERATING INFLORESCENCE MERISTEM* (*PIM*, also known as *PEAM4*; Berbel *et al*, 2001; Taylor *et al.*, 2002), is expressed in floral meristems with a pattern essentially identical to that of *AP1* and *SQUA*. Mutations in *PIM* also cause flower-to-shoot conversions and its functional homology with *AP1* is also supported by the phenotypes of *PIM* over-expression in arabidopsis. As in the case of the antirrhinum *squa* mutant, the mutations in *PIM*, as well as in *MtPIM*, the *M. truncatula* homologue, cause a

TABLE 1. LEAFY, APETALA1 *and* TERMINAL FLOWER1 *homologues in diverse eudicot species*

Species	Subclass; Order	Name	Homologous gene	Expression	Mutant phenotype	Over-expression in *Arabidopsis*	Over-expression in other species	References
Herbaceus species								
Arabidopsis thaliana	Rosids; Brassicales	*LEAFY* (*LFY*)	–	L (weak), FM	F → I conversions	Early flowering; shoot-to-flower conversions; terminal flower	–	Weigel *et al.*, 1992
		APETALA1 (*AP1*)	–	Young FM, whorls 1 + 2	F → I conversions; organ identity defects	Early flowering shoot-to-flower conversions; terminal flower	–	Mandel and Yanofsky, 1995
		TERMINAL FLOWER1 (*TFL1*)	–	VM, IM (main and lateral)	Shoot-to-flower conversions; early flowering	Late flowering	Tobacco: no phenotype	Shannon and Meeks-Wagner, 1991
Laevenworthia crassa	Rosids; Brassicales	*LcrLFY*	*LFY*	–	–	*pLcrLFY*:: *LcrLFY*: over-expression of *TFL1*; partly complements *lfy-6*	–	Baum *et al.*, 2005; Sliwinski *et al.*, 2006
Pisum sativum	Rosids; Fabales	*UNIFOLIATA* (*UNI*)	*LFY*	FM, VM, LP, L	F → I conversions; simpler leaves	–	–	Hofer *et al.*, 1997
		PEAM4/PIM	*AP1*	Young FM, whorls 1 + 2	F → I conversions; organ identity defects	35S::*PEAM4*: Early flowering, terminal flower, cauline leaves curling; partially rescues *ap1-1*	Tobacco: early flowering	Berbel *et al.*, 2001; Taylor *et al.*, 2002
		PsTFL1a/ *DETERMINATE* (*DET*); *PsTFL1c*/*LATE FLOWERING* (*LF*)	*TFL1*	*DET*: roots, IM, FM; *LF*: roots, L, VM, IM, FM, F	*det*: determination of main apex; *lf*: early flowering	–	–	Foucher *et al.*, 2003
Lotus japonicus	Rosids; Fabales	*LjLFY*	*LFY*	LP, FM	*pfm:* F → I conversions; simpler leaves	–	–	Dong *et al.*, 2005
		LjAP1a; *LjAP1b*	*AP1*	FM	–	–	–	Dong *et al.*, 2005
		LjCEN	*TFL1*	IM (main and lateral)	–	–	–	Guo *et al.*, 2006
Medicago truncatula	Rosids; Fabales	*MTPIM*	*AP1*	FM	F → I conversions; organ identity defects	–	–	Benlloch *et al.*, 2006
Antirrhinum majus	Asterids; Lamiales	*FLORICAULA* (*FLO*)	*LFY*	FM and subtending bract	F→ I conversions	–	–	Coen *et al.*, 1990
		SQUAMOSA (*SQUA*)	*AP1*	FM, bracts	F→ I conversions	–	–	Huijser *et al.*, 1992
		CENTRORADIALIS (*CEN*)	*TFL1*	IM	Shoot-to-flower conversions	–	Tobacco: late flowering; delayed downregulation of CET2/4	Bradley *et al.*, 1996; Amaya *et al.*, 1999
Petunia hybrida	Asterids; Solanales	*FLOP*	*LFY*	LP, L, IM	F → I conversions	–	–	Souer *et al.*, 1998

Continued

TABLE 1. *Continued*

Species	Subclass; Order	Name	Homologous gene	Expression	Mutant phenotype	Over-expression in *Arabidopsis*	Over-expression in other species	References
Nicotiana tabacum	Asterids; Solanales	*NFL1; NFL2*	*LFY*	VM (peripheral zone), AM, FM (peripheral)	Co-suppression: unregulated initiation of lateral meristems	35S::*NFL1*: prevents inflorescence branching; complements *lfy-16*	Tobacco: prevents I branching; promotes terminal flower formation	Kelly *et al.*, 1995; Ahearn *et al.*, 2001
		CET2; CET4	*CEN*	AM (only during vegetative phase)	–	–	–	Amaya *et al.*, 1999
Solanum lycopersicum	Asterids; Solanales	*FALSIFLORA* (*FA*)	*LFY*	VM, AM, L, FM and sympodial M	F → I conversions; reduced number of leaflets	–	–	Molinero-Rosales *et al.*, 1999
		SELF-PRUNING (*SP*)	*CEN*	VM, AM, L, IM, FM, SM, vasculature, primordia of floral organs	Determinate growth. Gradual reduction in the number of vegetative nodes per sympodial unit	35S::*SP*: extends vegetative phase	Tobacco: extends vegetative phase	Pnueli *et al.*, 1998
Impatiens balsamina	Asterids; Ericales	*IbLFY*	*LFY*	LP (peripheral), AM, FM	–	Early flowering; shoot-to-flower conversions; terminal flower	–	Pouteau *et al.*, 1998; Ordidge *et al.*, 2005
		IMP-SQUA	*AP1*	Petal primordia	–	–	–	Pouteau *et al.*, 1998
		IbTFL1	*TFL1*	AM that will produce inflorescences.	–	Similar to 35S::*TFL1*.	–	Ordidge *et al.*, 2005
Gerbera hybrida	Asterids; Asterales	*GSSQUA1*	*AP1*	Primordia of vascular tissues (receptacle and flower)	–	–	–	Yu *et al.*, 1999
Silene latifolia	Caryophyllales	*SLM4/5*	*AP1*	IM, FM	–	–	–	Hardenack *et al.*,1994
Perennial species								
Citrus sinensis	Rosids; Sapindales	*CsLFY*	*LFY*	Adult tissues after floral induction	–	–		Pillitteri *et al.*, 2004
		CsAP1	*AP1*	Adult tissues after floral induction	–	–	–	Pillitteri *et al.*, 2004
		CsTFL1	*CEN*	Juvenile stem tissue, F	–	Late flowering	–	Pillitteri *et al.*, 2004
Populus	Rosids; Malpighiales	*PTLF*	*LFY*	FM, F (male and female), bracts, VM	–	Early flowering (variable phenotype)	*Populus*: no phenotype	Rottmann *et al.*, 2000
Hevea brasiliensis	Rosids; Malphigiales	*HbLFY*	*LFY*	FM (male and female)	–	*pLFY*::*HbLFY*: complements *lfy-26*	–	Dornelas and Rodríguez, 2005
Eucalyptus	Rosids; Myrtales	*ELF1; ELF2* (pseudogene)	*LFY*	FM, LP	–	Early flowering; shoot-to-flower conversions; terminal flower	–	Southerton *et al.*, 1998
Betula pendula	Rosids; Fagales	*BpMADS3*	*AP1*	Male/female I, seed	–	35S::*BpMADS3* early flowering	Tobacco: early flowering	Elo *et al.*, 2001
Metrosideros excelsa	Rosids; Myrtales	*MEL*	*LFY*	IM and AM, FM, F; bimodal pattern	–	–	–	Sreekantan *et al.*, 2004
		MESAP1	*AP1*	AM, FM; bimodal pattern	–	–	–	Sreekantan *et al.*, 2004

		MeTFL1	*TFL1*	Bracts, IM; bimodal pattern, latent buds (1st season) and developing buds (2nd season)	–	–	–	Sreekantan *et al.*, 2004
Malus domestica	Rosids; Rosales	*ALF1*; *ALF2*	*LFY*	*AFL1*: FM; *ALF2*: constitutive	–	Early flowering; shoot-to-flower conversions	–	Wada *et al.*, 2002
		MdTFL1	*TFL1*	Developing buds, shoots, roots	–	35S::*MdTFL1* phenotype similar to 35S::*TFL1*, late flowering, flower to shoot conversions	–	Kotoda and Wada, 2005
Actinidia deliciosa	Asterids; Ericales	*ALF*	*LFY*	Developing buds; bimodal pattern	–	–	–	Walton *et al.*, 2001
		AAP1	*AP1*	Developing buds; bimodal pattern	–	–	–	Walton *et al.*, 2001
Vitis vinifera	Vitales	*VFL*	*LFY*	LM, young FM, LP, L, tendrils	–	–	–	Carmona *et al.*, 2002
		VAP1	*AP1*	IM, FM, tendrils	–	–	–	Calonje *et al*, 2004
		VvTFL1	*CEN*	Bimodal pattern, latent buds (1st season) and developing bud (2nd season)	–	Reduced apical dominance in arabidopsis	–	Carmona *et al.*, 2007

LP: leaf primordium; L: leaf; F: flower; I: Inflorescence; FM: floral meristem; IM: inflorescence meristem; VM: vegetative meristem; AM: axillary meristemos; SM: sympodial meristem.

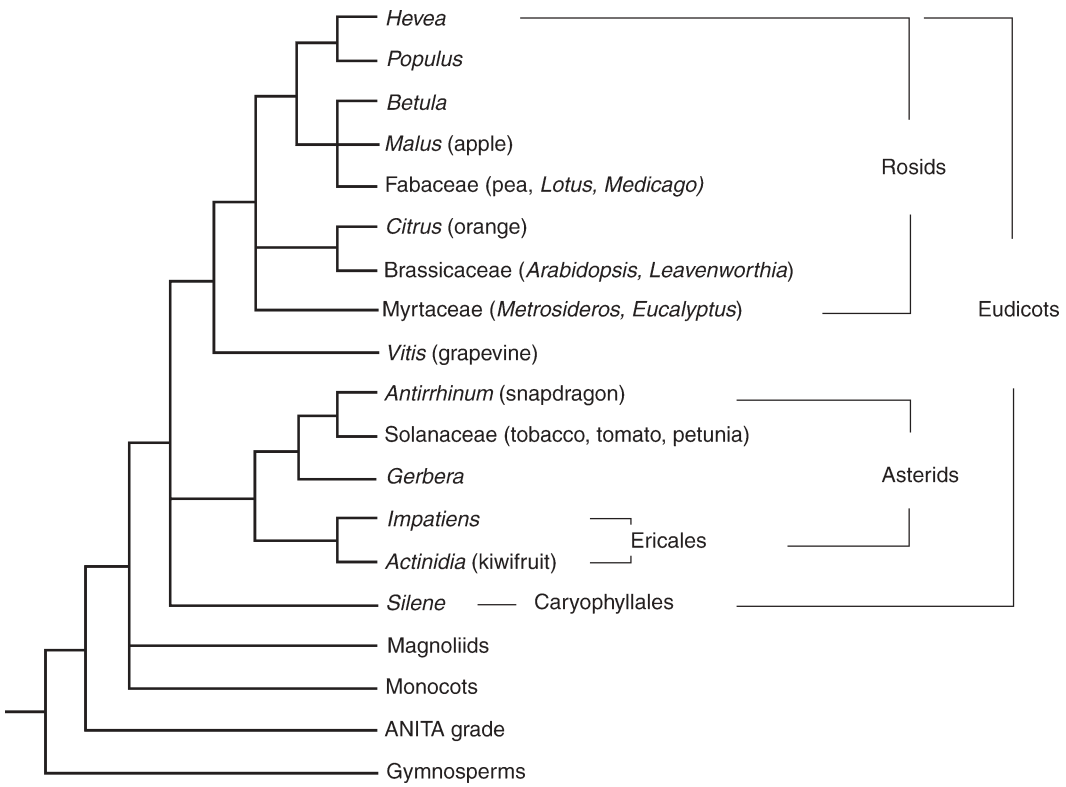

FIG. 3. Tree of the phylogenetic relationships among the species cited in this review. The tree is based on Soltis and Soltis (2003), Leebens-Mack *et al.* (2005) and the 'Tree of life web project' (http://www.tolweb.org/tree/).

phenotype more severe than that of the arabidopsis *ap1* mutant. SEM analysis has shown true conversion of the *mtpim* floral meristems into proliferating secondary inflorescence meristems, which generate structures that resemble the proliferating inflorescence meristems produced by the double *ap1 cal* mutants (Benlloch *et al.*, 2006).

A particular feature of the *uni* mutant is that it is also affected in its leaf morphology (Hofer *et al*, 1997). While the pea wild-type leaves are compound odd-pinnate, with a rachis supporting several pairs of leaflets, the leaves of the *uni* mutant are much simpler, having a shorter petiole bearing only one-to-three leaflets. Accordingly, *UNI* is also expressed in developing leaves. It has been suggested that *UNI* could have a function controlling the indeterminacy during leaf or flower development, reminiscent of an ancestral broader function of *LFY* genes in meristem control. The expression of *UNI* during pea leaf development would temporarily inhibit leaf determination, allowing the development of a complex leaf (Hofer and Ellis, 2002). The role of *LFY* homologues in leaf complexity also extends to other legume species, as mutations in *LjLFY* transform the *Lotus* trifoliate leaf into unifoliate (Dong *et al.*, 2005).

Brassicaceae. Most species from this family of rosids (Fig. 3) have a simple raceme type of inflorescence, similar to arabidopsis. However, a few species, such as *Ionopsidium acaule* (violet cress), *Idahoa scapigera* and *Laevenworthia crassa*, exhibit 'rosette flowering'. In such plants the main stem does not elongate and flowers are produced on long pedicels that emerge from the axils of rosette leaves, positions that in arabidopsis would produce cofloresccences. Evidence suggests that changes in the *LFY* homologues of these plants might have played an important role in the evolution of this different inflorescence architecture (Yoon and Baum, 2004).

These *LFY* homologues show expression patterns that differ to that of arabidopsis *LFY*. The *LFY* homologue of violet cress is strongly expressed in its SAM (Shu *et al.*, 2000) and the promoter of the *L. crassa LFY* directs expression to the SAM in transgenic arabidopsis plants (Yoon and Baum, 2004). Moreover, *lfy* mutant plants transformed with a genomic construct of *LcrLFY* exhibit morphological features that are reminiscent of rosette flowering. Finally, expression induced by AtLFY and LcrLFY has been compared by microarray analysis (Sliwinski *et al.*, 2006). Analysis of genes up- or down-regulated, showed that the *TFL1* gene was over-expressed in plants containing an *LcrLFY* transgene in comparison with those carrying an arabidopsis *LFY* transgene. Therefore, the nature of the interaction between *LFY* and *TFL1* could have changed between these species, generating differences in the architecture of the inflorescence. This study suggests that changes in *cis*-regulatory elements, leading to ectopic expression in axillary meristems and also in the protein coding region of *LcrLFY*, could be at the origin of rosette flowering (Sliwinski *et al.*, 2006). The idea that morphological evolution involves changes in the regulation of

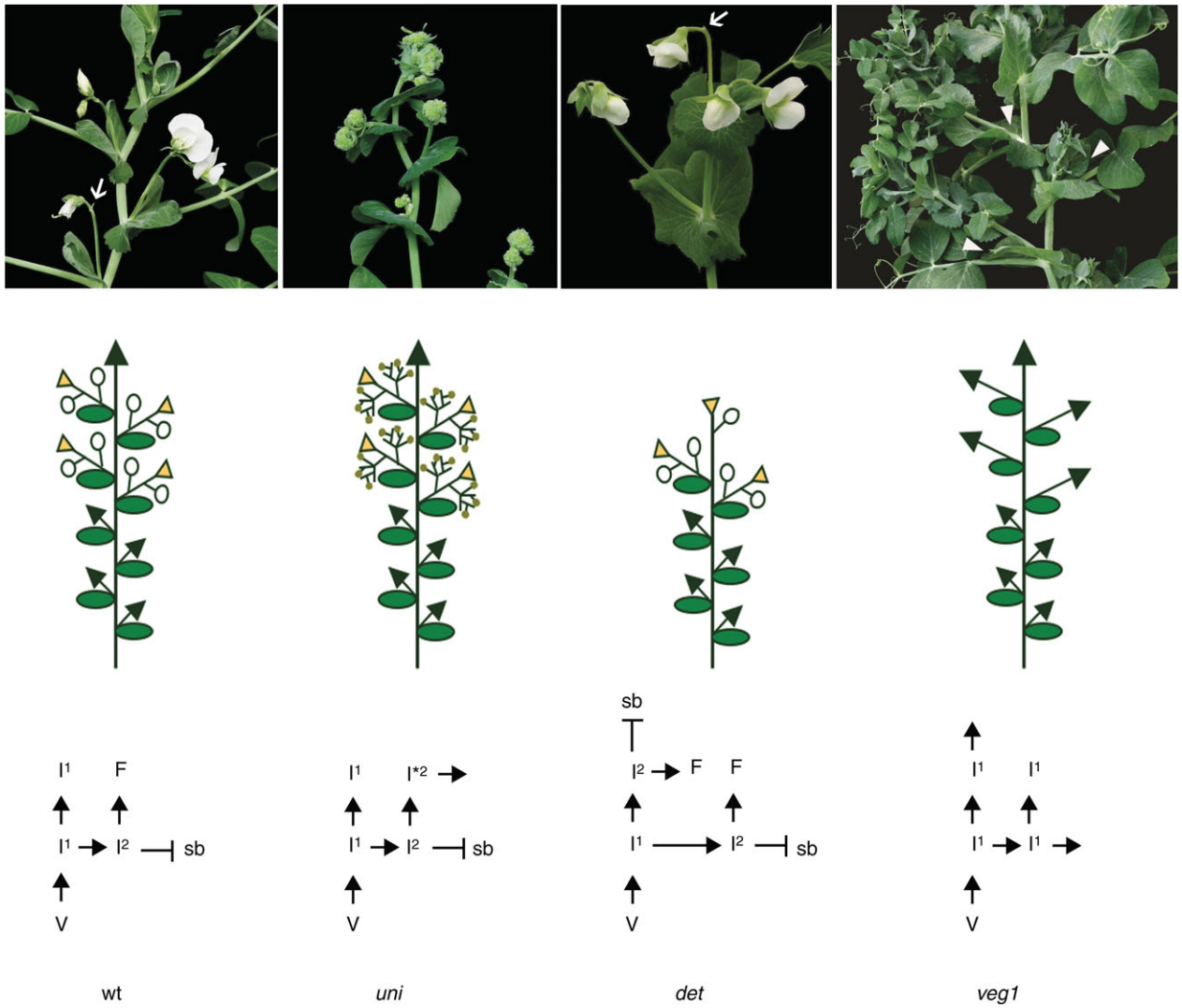

FIG. 4. Inflorescence and floral meristem mutants in pea, an example of a compound raceme. Inflorescences of the wild-type (wt) pea plant and of *uni*, *det* and *veg1* mutants. In a wt plant, the vegetative meristem (V) after the floral transition becomes a primary inflorescence meristem (I1) that generates secondary inflorescence meristems (I2) that produce the flowers (F). The secondary inflorescences are formed in the axil of leaves and produce 1–2 flowers before generating a rudimentary stub (indicated by arrows in the photographs). In the *uni* mutant the I2, instead of flowers, generate other I2s that keep proliferating indefinitely (I*2). In the *det* mutant, the I1 is prematurely transformed into an I2 that produces one or two flowers before terminating into a stub (arrow). In the *veg1* mutant, the I2s are transformed into vegetative I1 meristems (indicated by arrowheads in the photograph), generating a plant that never flowers.

developmental genes has already been suggested (Doebley and Lukens, 1998). This example and others discussed in this review place *LFY*, *AP1* and *TFL1* as master developmental genes whose changes in expression pattern could play a key role in determining plant and inflorescence architecture.

LFY/AP1 *in herbaceous species, determinate inflorescences*

Solanaceae. Another dicot family where *LFY* homologues have been studied in detail is the Solanaceae (Fig. 3). Many species from this family have determinate cymose inflorescences, including tobacco (Fig. 1E). Tomato and *Petunia hybrida* plants, in addition to cymose inflorescences, also exhibit a sympodial growth habit (Fig. 1F). Again, the behaviour of the *LFY* homologues in these plants show particular features that relate to the architecture of their inflorescences.

The function of the *LFY* homologues in tomato and petunia in FMI seems to be similar to that in arabidopsis. Mutations in *FALSIFLORA* (*FA*) in tomato, or in *ABERRANT LEAF AND FLOWER* (*ALF*) in petunia, cause the conversion of the floral meristem into an inflorescence meristem similar to *lfy* (Souer *et al.*, 1998; Molinero-Rosales *et al.*, 1999). However, the expression of *FA* and *ALF* significantly differ from that of *LFY* in arabidopsis. In addition to being expressed in floral meristems, in petunia *ALF* is expressed in the inflorescence meristem and in tomato *FA* is also expressed in the sympodial meristem. The expression of *LFY* homologues in the shoot meristems of these plants is very likely related to the formation of

terminal flowers by their inflorescences. In both species *LFY* transcripts can be also found, as in pea, in leaf primordia. In agreement with this, tomato *fa* mutants show certain reductions in leaf complexity; however, no leaf phenotype can be observed in petunia *alf* mutants.

Tobacco contains two *LFY* homologues, *NFL1* and *2*, possibly due to the allotetraploid origin of this species (Kelly *et al.*, 1995). Expression of *NFL* genes is similar to that of their petunia and tomato homologues, with their transcripts also being found in vegetative and axillary meristems. However, the function of the tobacco homologues could be different. *NFL1* over-expression in tobacco promotes terminal flower formation and inhibits inflorescence branching but does not cause early flowering, such as 35S::LFY causes in tobacco. On the other hand, the co-suppression of *NFL* genes produces unregulated initiation of lateral meristems. All these data suggest that *NFL1* has an additional role compared to *LFY* in the allocation and placement of meristematic cells (Ahearn *et al.*, 2001).

Silene latifolia. This species (from the order Caryophyllales) also has a determinate inflorescence, of a type named a dichasium, where the apical meristem forms a terminal flower and two inflorescences meristems are formed on its flanks (Fig. 1D). In *S. latifolia*, two *AP1* homologues, *SLM4* and *SLM5*, have been characterized (Hardenack *et al.*, 1994). Only *SLM4* is a likely *AP1* orthologue, while *SLM5* is closer to *FUL* (Litt and Irish, 2003). *SLM4* is expressed in flowers with a similar pattern to that of *AP1* or *SQUA* but, as with the *LFY* homologues in the Solanaceae, the *S. latifolia AP1* homologue is also expressed in the inflorescence meristem. Again, this is another example where the generation of a determinate inflorescence seems to be related to expression of a FMI gene in the shoot apical meristem.

Impatiens balsamina. In this species, indeterminate and determinate inflorescence varieties are found. For determinate varieties, the first nodes produce leaves subtending axillary vegetative shoots, the next nodes produce axillary inflorescences also subtended by leaves, and the last nodes before the terminal flower produce flowers with subtending bracts. Indeterminate varieties continue producing axillary inflorescences indefinitely (Pouteau *et al.*, 1998; Ordidge *et al.*, 2005). Flowering in *I. balsamina* is dependent on short-day (SD) conditions and the plants remain vegetative under long days (LD). A specific feature of *I. balsamina* is that the terminal flower of determinate varieties reverts to vegetative growth after transfer to LD non-inductive conditions, a phenomenon that is termed floral reversion.

The expression of *LFY* and *AP1* homologues, *IbLFY* and *IMP-SQUA*, have been analysed in the determinate variety (Pouteau *et al.*, 1997). As occurs in other species such as arabidopsis, *IMP-SQUA* expression is only expressed after floral induction. However, the situation with *IbLFY* is somewhat unusual, as it is expressed in the vegetative and in the flowering terminal meristem of *I. balsamina.* This is in contrast to arabidopsis or antirrhinum but similar to what occurs in tobacco, and could be related to the determination of the shoot meristem into a terminal flower. However, expression of *IbLFY* is also detected in the shoot meristem after floral reversion and in the meristem of non-induced LD-growing plants, suggesting that *IbLFY* expression in the meristem is not sufficient to specify floral identity. Nevertheless, the phenotype of *IbLFY* over-expression in arabidopsis indicates functional homology between *IbLFY* and *LFY* (Ordidge *et al.*, 2005). Therefore, a possibility is that the interaction of *LFY* with other regulators of FMI could be different in *I. balsamina* (see *IbTFL1*, below).

LFY/AP1 *in perennial species*

Woody species have two important developmental characteristics that make them different from annual herbaceous plants. First, they have long juvenile phases (from several years to decades) during which they produce only vegetative organs. Second, the flowering process often extends to two consecutive seasons – during the first season buds are formed that during the second season will develop and produce flowers or inflorescences. Genetic analysis in perennials is a complicated task and, consequently, our understanding of the function of the *LFY* and *AP1* homologues from this type of plants is not as precise as in herbaceous species. Nevertheless, homologues have been analysed in several perennial species and the available data indicate that these genes affect both these characteristics of woody plant development.

As expected, expression of *LFY* and *AP1* homologues in perennials is also associated with floral and inflorescence buds (see Table 1). Expression of these genes appears to follow a bimodal pattern related to the two seasons that are needed to flower. This has been studied in detail in the case of grapevine (*Vitis vinifera*). During the first season, the SAM produces lateral meristems that will generate inflorescence meristems. These inflorescence meristems form inflorescence branch meristems before the buds enter dormancy. In the second growing season, these buds form additional inflorescence branch meristems before dividing into 3–4 floral meristems. *VFL*, the *LFY* homologue, is expressed in lateral meristems independent of their fate, although *VFL* expression increases in young floral meristems. The expression level of *VFL* reaches two peaks, one at the time of flowering induction during the first growing season, and a second peak at the time of bud reactivation and flower initiation during the second growing season (Carmona *et al.*, 2002). The grapevine *AP1* homologue, *VAP1*, is expressed in early stages of inflorescence development during the first season, and later on in inflorescence branch meristems. During the second season, *VAP1* expression is detected in floral meristems and it is maintained during flower development. Expression patterns of the grapevine *LFY/AP1* homologues suggest that both genes are also involved in other processes in addition to flower development. Thus, *VFL* is also expressed in leaf primordia and in the growing margins of developing leaves, where it has been suggested that it maintains the cell proliferation needed for the typical palmate morphology of the grapevine leaves (Carmona *et al.*, 2002). On the other hand, *VAP1* seems to be involved in

tendril development as it is expressed during the development of these organs, independent of the flowering process, even in very young plants that have not undergone the floral transition (Calonje *et al.*, 2004).

Another example of expression of FMI genes associated with the two growing seasons in perennials is that of *BpMADS3*, the likely *AP1* orthologue of birch (*Betula pendula*; Elo *et al.*, 2001). *BpMADS3* also exhibits a bimodal expression pattern during inflorescence development. Birch has separate male and female inflorescences and *BpMADS3* shows different expression patterns in each of them, according to their different timing of development. A peculiarity of the *B. pendula AP1* homologue is that expression also continues at a high level during late flower development and even during seed development.

TFL1 HOMOLOGUES, REPRESSORS OF PHASE CHANGES

TFL1 has an opposite function to LFY and AP1 and belongs to the group of PEBP proteins. PEBP genes have been found in many angiosperm species, dicots and monocots, and constitute gene families whose number varies in different species – from six members in arabidopsis or tomato to 19 in rice. Plant PEBP proteins can be grouped into three main clades: the MFT-, FT- and TFL1-like subfamilies (Mimida *et al.*, 2001; Carmel-Goren *et al.*, 2003; Chardon and Damerval, 2005). Those *TFL1*-like genes for which a function has been found have roles in the control of plant development, usually in flowering. As we will see in the examples that follow, many *TFL1*-like genes are key controllers of flowering time and inflorescence architecture.

TFL1 *in herbaceous species with indeterminate inflorescences*

Antirrhinum majus. As was the case with *LFY* and *FLO*, *CENTRORADIALIS* (*CEN*) from *Antirrhinum majus* was the first member of the plant PEBP gene family that was characterized (Bradley *et al.*, 1996); thereafter *TFL1* was isolated as an arabidopsis *CEN* homologue. In agreement with the similarities between arabidopsis and antirrhinum inflorescences, mutations in the antirrhinum homologue also cause the conversion of the SAM into a terminal flower, changing the inflorescence from indeterminate to determinate. As with *TFL1* in arabidopsis, *CEN* is expressed in the subapical region of the shoot meristem, somehow inhibiting the expression of the *LFY* homologue in this meristem. However, while *TFL1* is expressed both in vegetative and inflorescence shoot meristems of arabidopsis, *CEN* is only expressed in the inflorescence meristem. The absence of *CEN* expression in the apex before floral transition has been used to explain the fact that, in contrast to *tfl1*, *cen* mutations do not affect flowering time (Bradley *et al.*, 1996; Cremer *et al.*, 2001). Nevertheless, although the expression patterns could explain the different mutant phenotypes, differences in the function of the two proteins can not be discarded. In fact, while 35S::CEN causes an extreme delay of flowering in tobacco, 35S::TFL1 did not show any effect in this species (Amaya *et al.*, 1999).

Interestingly, the most likely arabidopsis orthologue of *CEN* is not *TFL1* but the *ATC* gene (*Arabidopsis thaliana CEN* homologue; Mimida *et al.*, 2001), another member of the *TFL1* clade. However, while *ATC* can functionally substitute for *TFL1*, as suggested by its ability to complement the *tfl1* mutant phenotype when constitutively expressed, *ATC* loss-of-function mutations do not cause any obvious phenotype, indicating that *ATC* could be involved in a function different to IM identity. *ATC* expression is very low and restricted to the tissues around the vasculature of the hypocotyl. The striking differences between the expression patterns of *ATC* and *TFL1* have been considered to be the basis of their different functions (Mimida *et al.*, 2001).

Fabaceae. Among legume species, *TFL1* homologues have been described for pea and *Lotus japonicus*. Pea contains at least three *TFL1*/*CEN* homologues. No function has been assigned to *TFL1b*, a likely orthologue of *CEN*. *TFL1a* and *TFLc* are most closely related to *TFL1* and play *TFL1*-related functions. Mutations in *PsTFL1a*, also known as *DETERMINATE* (*DET*), cause the determination of the main apex without affecting flowering time, in a similar manner to what occurs in *cen* mutants of antirrhinum. On the other hand, mutations in *PsTFL1c*, also known as *LATE FLOWERING* (*LF*), cause early flowering without affecting determination (Foucher *et al.*, 2003). Therefore, it seems that in pea the 'two functions' of the arabidopsis *TFL1* gene, flowering time and apex determinacy, are controlled by two different genes. Whether the different functions of *DET* and *LF* are due to differences in their encoded proteins or to differential expression patterns is unclear. Expression patterns might at least partly explain the differences; thus, similar to *CEN*, *DET* is expressed in the shoot apex only after the floral transition, while *LF* expression is observed also in the vegetative apex. Expression of pea *TFL1* homologues have only been analysed by RT-PCR, but *in situ* hybridization analysis has indicated that the *L. japonicus DET* homologue, *LjCEN1*, is expressed in the inflorescence meristems (Guo *et al.*, 2006).

It is interesting to point out that the phenotype of the pea *det* mutant underlines the differences between the arabidopsis simple raceme and the pea compound raceme. The shoot apex of *det* mutants does not form a terminal flower, but instead it is transformed into an I2 and produces 1–2 flowers before terminating in a stub (Fig. 4); thus, in *det* mutants the I1 is transformed into an I2 (Singer *et al.*, 1990). Conversely, pea plants mutated in either the *VEGETATIVE1* (*VEG1*) or *VEGETATIVE2* (*VEG2*) genes show very similar phenotypes, where I2s are replaced by I1s and thus are opposite to *det* (Reid and Murfet, 1984; Murfet, 1992; Singer *et al.*, 1994; Reid *et al.*, 1996). During the vegetative phase, *veg1* mutant plants look identical to wild-type plants, and both produce several vegetative nodes, each with a leaf bearing an axillary vegetative meristem that stays dormant. In the wild-type plant, the meristems in the nodes produced after the floral transition

develop as secondary inflorescences. In contrast, in the *veg1* and *veg2* mutants, the meristems of nodes in equivalent apical positions also start growing but, rather than producing secondary inflorescences, they generate indeterminate vegetative shoots identical to I1s. This generates plants with a unique non-flowering phenotype, never observed in any other species as the result of a single recessive mutation. We can explain the phenotypes of *det* and *veg* mutants by considering that *VEG1/2* and *DET* genes control the identity of the primary and secondary inflorescence meristems. *DET* would specify I1 identity, whilst I2 identity would be specified by *VEG1* and *VEG2*. Such phenotypes do not occur in arabidopsis as compound racemes have additional levels of regulation of meristem identity.

In summary, we can easily establish a parallelism between the repression network of *TFL1-LFY/AP1*, which maintains the indeterminacy of the shoot meristem, and the *DET-VEG1/2* network, which prevents the conversion of the shoot apical meristem into an I2. The molecular characterization of *VEG1* and *VEG2* will represent a major advance in understanding the molecular mechanism underlying the development of compound inflorescences.

TFL1 *in herbaceous species, determinate inflorescences*

Solanaceae. TFL1/CEN homologues have been studied from two Solanaceae, tobacco and tomato. From tobacco, seven *CEN*-like genes (*CET* genes) have been isolated (Amaya *et al.*, 1999). Sequence analysis indicates that *CET2/4*, *CET5/6* and *CET1/7* are pairs of genes probably representing single copy genes in the diploid progenitors of this allotetraploid species. *CET2/4* are the most closely related to *CEN* and, like *TFL1*, are expressed in axillary shoot meristems. However, expression of *CET2/4* was not detected in the apex of the main shoot, which expresses *NFL* (the *LFY* orthologue) and forms a terminal flower (Ahearn *et al.*, 2001). In fact, *CET2/4* expression is restricted to vegetative axillary meristems and is not detected in those axillary meristems just below the terminal flower that will give rise to terminal cymes, and which express *NFL*. As the vegetative axillary meristems develop into flowering shoots, expression of *CET2/4* decreases and *NFL* is upregulated, suggesting that the *CET* genes act to maintain the vegetative character of these meristems, delaying their transition to an inflorescence phase. These observations also suggest that the antagonism between *TFL1* and FMI genes observed in arabidopsis also occurs in tobacco and again supports the view that in some species with determinate inflorescences the formation of terminal flowers depends on the balance between *TFL1* and FMI genes in the shoot meristems.

Interestingly, the *CET2/4* genes from tobacco are most closely related to *CEN* and *ATC*. Actually, the expression of *CET1* or *CET6* (the most similar to *TFL1*) was not detectable by *in situ* hybridization. This might indicate that in tobacco, as in antirrhinum (also from the asterids clade), the *CEN*-related genes have a more prevalent function than the *TFL1*-related ones, as also suggested by the different effects of *CEN* and *TFL1* over-expression in tobacco (Amaya *et al.*, 1999).

Tomato has a sympodial growth habit and the tomato *TFL1*-like gene *SELF PRUNING* (*SP*) acts specifically on the sympodial meristem (Pnueli *et al.*, 1998). Mutations in *SP* do not affects the floral transition of the vegetative shoot meristem, which produces the same number of nodes as the wild type before forming the primary inflorescence. However, in *sp* mutants, the number of leaves per sympodial unit progressively decreases with age until the last unit generates only an inflorescence, so that the shoot is terminated by two consecutive inflorescences. The *SP* expression pattern is somewhat unusual. While expression of the arabidopsis, antirrhinum and tobacco homologues (*TFL1*, *CEN* and *CET2/4*) is localized to specific shoot meristems, *SP* expression occurs in all meristems (vegetative, inflorescence floral and axillary), and in leaf and floral organ primordia (Pnueli *et al.*, 1998). It is intriguing how *SP* only affects vegetative-to-reproductive transitions of sympodial meristems.

As in tobacco, *SP* is more closely related to *CEN* than to *TFL1*, and 35S:CEN (as 35S:SP) rescues the indeterminate phenotype in *sp* mutants (Pnueli *et al.*, 1998). In tomato again, therefore, a *CEN* homologue would be playing a '*TFL1*-related' function, although affecting only the lateral sympodial meristem. *SP9D*, the tomato homologue most closely related to *TFL1*, is expressed in roots and in the shoot apex (Carmel-Goren *et al.*, 2003). It would be interesting to see whether mutations in this gene cause a phenotype similar to *tfl1*, and accelerated flowering.

Impatiens balsamina. In *I. balsamina*, the expression of *IbTFL1*, a functional homologue of *TFL1*, has been analysed in both determinate and indeterminate varieties (Ordidge *et al.*, 2005). Expression of *IbTFL1* in determinate plants resembles that of the tobacco *TFL1*-homologues *CET2/4*, being found in axillary meristems that produce inflorescences, but not in the shoot apical meristem nor in axillary meristems of the upper nodes of the inflorescence, which develop as flowers. This has been used as a basis to suggest that *IbTFL1* may be involved in maintaining the inflorescence state of axillary shoot meristems and axillary inflorescences (Ordidge *et al.*, 2005). However, the regulation of the terminal inflorescence seems to be different. While, in principle, the absence of expression in the apex of determinate plants would appear to be linked to terminal flower formation, *IbTFL1* is not expressed either in the apex of indeterminate plant varieties or in the apex of plants of the determinate line grown under non-inductive conditions. The meristems of these indeterminate apices express *IbLFY*, which is therefore insufficient to specify them as floral, even in the absence of *IbTFL1* (Pouteau *et al.*, 1997). Consequently, it has been proposed that in *I. balsamina* the fate of the terminal inflorescence is controlled by an integration system not depending on *LFY* and *TFL1*. The *AP1* pathway has been suggested as a more likely candidate for this control (Ordidge *et al.*, 2005).

TFL1 *in woody perennials*

TFL1 homologues have been studied in some detail in a few perennial dicots; species such as orange tree

(*Citrus sinensis*; Pillitteri *et al.*, 2004), apple (*Malus domestica*; Kotoda and Wada, 2005), *Metrosideros excelsa* (Sreekantan *et al.*, 2004) and grapevine (Carmona *et al.*, 2007) are good examples (Table 1). As for *LFY* homologues, no mutants have been described for any of the *TFL1* perennial homologues and conclusions on their functions are based on expression studies and analysis of transgenic plants.

In general, expression of the *TFL1* homologues in these woody perennials is detected in vegetative tissues, such as apical buds and stems. *In situ* hybridization has also shown that *MeTFL1* is expressed in subapical regions of the inflorescence meristems of *M. excelsa*, in a pattern very similar to that of *TFL1* in arabidopsis. There seems to be a relationship between the two seasons required for flowering in woody plants and the expression of their *TFL1* homologues. Thus, the *TFL1*-like genes from *M. excelsa* and grapevine have been shown to follow a bimodal expression pattern associated with reproductive development; expression is high in latent buds of the first season, then disappears during the dormancy period, and is then observed again in the second season when bud development is resumed (Sreekantan *et al.*, 2004; Carmona *et al.*, 2007). In general, expression of the *TFL1* homologues in perennials does not coincide in space and/or time with that of the *LFY* or *AP1* homologues; instead they are largely complementary. Thus, in grapevine and apple expression of the *TFL1* homologues in developing buds is high during the initial stages of inflorescence development, but is absent later during flower development, when expression of the *LFY* and *AP1* homologues becomes high.

For the *TFL1*-like genes of apple and citrus, constitutive expression in arabidopsis has been shown to cause a late-flowering phenotype, similar to that of plants over-expressing the arabidopsis *TFL1* gene. This, and their expression patterns, suggests a role for the *TFL1*-like genes of these perennials in maintaining indeterminacy of the shoot meristems within the developing bud.

Regarding the juvenile phase, the *TFL1* homologues from citrus and apple have been suggested also to be involved in the regulation of its duration. For citrus, this is based on the high levels of *CsTFL* transcripts found in juvenile tissues (Pillitteri *et al.*, 2004), and in apple on the strong reduction of the juvenile phase in antisense *MdTFL1* apple trees (Kotoda *et al.*, 2003). Whether this is also a likely function for the other perennial *TFL1* homologues remains to be investigated.

SUMMARY AND CONCLUSIONS

A major conclusion of this review is that *LFY*, *AP1* and *TFL1* genes are major factors controlling the behaviour of reproductive meristems, not only in arabidopsis but in most plant species. The emerging picture from the comparative studies described here is that differences in their expression patterns or in the activity of their proteins could explain a great part of the variation in the basic inflorescence architecture found among species.

Something that seems generally conserved is the antagonism between the function of *LFY* and *TFL1* homologues, clearly shown by their generally mutual complementary expression patterns. Although they are often considered regulators of floral identity specification, both genes seem to play a relatively general role in the control of meristem fate. *TFL1* homologues apparently act by 'repressing' transitions between developmental phases: all phase transitions at the arabiodpsis SAM, floral transition of the sympodial meristem of tomato or, in legumes, I1 to I2 transition. In addition to floral meristem identity, *LFY* genes also appear to be involved in phase transitions, though with a promoting role. For example, in arabidopsis *LFY* integrates genetic pathways inducing the floral transition.

LFY genes encode a type of transcription factor, unique to the plant kingdom, which is found from mosses to eudicots. Remarkably, the *LFY* genes have dupicated very rarely, usually being represented by only one or two homologues in the different plant species. Nevertheles, during its long evolutionary history, the *LFY* genes appear to have been recruited for other functions not related to flowering, such as control of leaf development in legumes and tomato.

AP1 is a MADS-box type transcriptional regulator. The MADS-box gene family has greatly diversified in the plant kingdom, leading to the appearance of a high number of genes with very diverse roles in the regulation of plant development. The *AP1* genes are only found in the core eudicot clade, probably linked to the origin of the eudicot flower. Accordingly, the expression and function of this group of genes are generally related to floral meristems.

Finally, knowledge on the evolution of *TFL1* homologues is much more limited than that of *LFY*- or *AP1*-like genes. In the different eudicot species analysed, the *TFL1* gene family has no more than six members and the available data suggest that its function as a phase-change repressor could have been adopted by two different members of the *TFL1* clade, *TFL1*-homologues in rosids and *CEN*-homologues in asterids, possibly as consequence of evolution of their expression patterns.

Our knowledge of the genetic control of inflorescence development in different plants is still limited. It seems clear that more studies, systematically comparing the function of the relevant genes in species representing the major clades of the plant phylogenetic tree, need to be done in order to have a more comprehensive view. Nevertheless, it is remarkable that, despite the available information still being fragmentary, the analysis in different species of a few genetic functions initially identified in model plants is showing that such a very simple regulatory network could be the basis of the generation of a huge variety of inflorescence architectures. This is a clear example of how comparative studies of genetic regulatory functions are helping us to understand the evolution of plant development.

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LITERATURE CITED

Abe M, Kobayashi Y, Yamamoto S, Daimon Y, Yamaguchi A, Ikeda Y, *et al*. 2005. FD, a bZIP protein mediating signals from the floral pathway integrator FT at the shoot apex. *Science* **309**: 1052–1056.

Ahearn KP, Johnson HA, Weigel D, Wagner DR. 2001. *NFL1*, a *Nicotiana tabacum LEAFY*-like gene, controls meristem initiation and floral structure. *Plant Cell Physiology* **42**: 1130–1139.

Ahn JH, Miller D, Winter VJ, Banfield MJ, Lee JH, Yoo SY, *et al*. 2006. A divergent external loop confers antagonistic activity on floral regulators FT and TFL1. *EMBO Journal* **25**: 605–614.

Alvarez J, Guli CL, Yu X-H, Smyth DR. 1992. *TERMINAL FLOWER*: a gene affecting inflorescence development in *Arabidopsis thaliana*. *Plant Journal* **2**: 103–116.

Amaya I, Ratcliffe OJ, Bradley DJ. 1999. Expression of *CENTRORADIALIS* (*CEN*) and *CEN*-like genes in tobacco reveals a conserved mechanism controlling phase change in diverse species. *The Plant Cell* **11**: 1405–1418.

Baum DA, Yoon H-S, Oldham RL. 2005. Molecular evolution of the transcription factor *LEAFY* in *Brassicaceae*. *Molecular Phylogenetics and Evolution* **37**: 1–14.

Benlloch R, d'Erfurth I, Ferrándiz C, Cosson V, Beltrán JP, Canas LA, *et al*. 2006. Isolation of *mtpim* proves *Tnt1* a useful reverse genetics tool in *Medicago truncatula* and uncovers new aspects of *AP1*-like functions in legumes. *Plant Physiology* **142**: 972–983.

Berbel A, Navarro C, Ferrándiz C, Canas LA, Madueño F, Beltrán JP. 2001. Analysis of *PEAM4*, the pea *AP1* functional homologue, supports a model for *AP1*-like genes controlling both floral meristem and floral organ identity in different plant species. *Plant Journal* **25**: 441–451.

Blázquez MA, Weigel D. 2000. Integration of floral inductive signals in *Arabidopsis*. *Nature* **404**: 889–892.

Blázquez MA, Soowal LN, Lee I, Weigel D. 1997. *LEAFY* expression and flower initiation in *Arabidopsis*. *Development* **124**: 3835–3844.

Blázquez MA, Ferrándiz C, Madueño F, Parcy F. 2006. How floral meristems are built. *Plant Molecular Biology* **60**: 855–870.

Bommert P, Satoh-Nagasawa N, Jackson D, Hirano H-Y. 2005. Genetics and evolution of inflorescence and flower development in Grasses. *Plant Cell Physiology* **46**: 69–78.

Bowman JL, Alvarez J, Weigel D, Meyerowitz EM, Smyth DR. 1993. Control of flower development in *Arabidopsis thaliana* by *APETALA*1 and interacting genes. *Development* **119**: 721–743.

Bradley D, Carpenter R, Copsey L, Vincent C, Rothstein S, Coen E. 1996. Control of inflorescence architecture in *Antirrhinum*. *Nature* **379**: 791–797.

Bradley D, Ratcliffe O, Vincent C, Carpenter R, Coen E. 1997. Inflorescence commitment and architecture in *Arabidopsis*. *Science* **275**: 80–83.

Calonje M, Cubas P, Martínez-Zapater JM, Carmona MJ. 2004. Floral meristem identity genes are expressed during tendril development in grapevine. *Plant Physiology* **135**: 1491–1501.

Carmel-Goren L, Liu YS, Lifschitz E, Zamir D. 2003. The *SELF-PRUNING* gene family in tomato. *Plant Molecular Biology* **52**: 1215–1222.

Carmona MJ, Cubas P, Martínez-Zapater JM. 2002. *VFL*, the grapevine *FLORICAULA*/*LEAFY* ortholog, is expressed in meristematic regions independently of their fate. *Plant Physiology* **130**: 68–77.

Carmona MJ, Calonje M, Martínez-Zapater JM. 2007. The *FT*/*TFL1* gene family in grapevine. *Plant Molecular Biology* **63**: 637–650.

Chardon F, Damerval C. 2005. Phylogenomic analysis of the PEBP gene family in cereals. *Journal of Molecular Evolution* **61**: 579–590.

Chautard H, Jacquet M, Schoentgen F, Bureaud N, Benedetti H. 2004. Tfs1p, a member of the PEBP family, inhibits the Ira2p but not the Ira1p Ras GTPase-activating protein in *Saccharomyces cerevisiae*. *Eukaryotic Cell* **3**: 459–470.

Coen ES, Nugent JM. 1994. Evolution of flowers and inflorescences. *Development Supplement*: 107–116.

Coen ES, Romero JM, Doyle S, Elliott R, Murphy G, Carpenter R. 1990. *Floricaula*: a homeotic gene required for flower development in *Antirrhinum majus*. *Cell* **63**: 1311–1322.

Cremer F, Lonnig WE, Saedler H, Huijser P. 2001. The delayed terminal flower phenotype is caused by a conditional mutation in the *CENTRORADIALIS* gene of snapdragon. *Plant Physiology* **126**: 1031–1041.

Doebley J, Lukens L. 1998. Transcriptional regulators and the evolution of plant form. *The Plant Cell* **10**: 1075–1082.

Domoney C, Duc G, Ellis THN, Ferrándiz C, Firnhaber C, Gallardo K, *et al*. 2006. Genetic and genomic analysis of legume flowers and seeds. *Current Opinion in Plant Biology* **9**: 133–141.

Dong ZC, Zhao Z, Liu CW, Luo JH, Yang J, Huang WH, *et al*. 2005. Floral patterning in *Lotus japonicus*. *Plant Physiology* **137**: 1272–1282.

Dornelas MC, Rodríguez APM. 2005. The rubber tree (*Hevea brasiliensis* Muell. Arg.) homologue of the *LEAFY*/*FLORICAULA* gene is preferentially expressed in both male and female floral meristems. *Journal of Experimental Botany* **56**: 1965–1974.

Elo A, Lemmetyinen J, Turunen M-L, Tikka L, Sopanen T. 2001. Three MADS-box genes similar to *APETALA* 1 and *FRUITFULL* from silver birch (*Betula pendula*). *Physiologia Plantarum* **112**: 95–103.

Ferrándiz C, Gu Q, Martienssen R, Yanofsky MF. 2000. Redundant regulation of meristem identity and plant architecture by *FRUITFULL*, *APETALA1* and *CAULIFLOWER*. *Development* **127**: 725–734.

Foucher F, Morin J, Courtiade J, Cadioux S, Ellis N, Banfield MJ, Rameau C. 2003. *DETERMINATE* and *LATE FLOWERING* are two *TERMINAL FLOWER1*/*CENTRORADIALIS* homologs that control two distinct phases of flowering initiation and development in pea. *The Plant Cell* **15**: 2742–2754.

Frolich MW, Parker DS. 2000. The mostly male theory of flower evolutionary origins: from genes to fossils. *Systematic Botany* **25**: 155–170.

Gu Q, Ferrándiz C, Yanofsky MF, Martienssen R. 1998. The *FRUITFULL* MADS-box gene mediates cell differentiation during *Arabidopsis* fruit development. *Development* **125**: 1509–1517.

Guo X, Zhao Z, Chena J, Hua X, Luo D. 2006. A putative *CENTRORADIALIS*/*TERMINAL FLOWER1*-like gene, *Ljcen1*, plays a role in phase transition in *Lotus japonicus*. *Journal of Plant Physiology* **163**: 436–444.

Hanzawa Y, Money T, Bradley D. 2005. A single amino acid converts a repressor to an activator of flowering. *Proceedings of the National Academy of Sciences of the USA* **102**: 7748–7753.

Hardenack S, Ye D, Saedler H, Grant S. 1994. Comparison of MADS box gene expression in developing male and female flowers of the dioecious plant white campion. *The Plant Cell* **6**: 1775–1787.

Hecht V, Foucher F, Ferrándiz C, Macknight R, Navarro C, Morin J, *et al*. 2005. Conservation of *Arabidopsis* flowering genes in model legumes. *Plant Physiology* **137**: 1420–1434.

Hempel FD, Weigel D, Mandel MA, Ditta G, Zambryski PC, Feldman LJ, Yanofsky MF. 1997. Floral determination and expression of floral regulatory genes in *Arabidopsis*. *Development* **124**: 3845–3853.

Hengst U, Albrecht H, Hess D, Monard D. 2001. The phosphatidylethanolamine-binding protein is the prototype of a novel family of serine protease inhibitors. *Journal of Biological Chemistry* **276**: 535–540.

Hofer J, Ellis N. 2002. Conservation and diversification of gene function in plant development. *Current Opinion in Plant Biology* **5**: 56–61.

Hofer J, Turner L, Hellens R, Ambrose M, Matthews P, Michael A, Ellis N. 1997. *UNIFOLIATA* regulates leaf and flower morphogenesis in pea. *Current Biology* **7**: 581–587.

Huala E, Sussex IM. 1992. *LEAFY* interacts with floral homeotic genes to regulate *Arabidopsis* floral development. *Plant Cell* **4**: 901–903.

Huijser P, Klein J, Lonnig WE, Meijer H, Saedler H, Sommer H. 1992. Bracteomania, an inflorescence anomaly, is caused by the loss of

function of the MADS-box gene *squamosa* in *Antirrhinum majus*. *EMBO Journal* **11**: 1239–1249.

Ingram GC, Goodrich J, Wilkinson MD, Simon R, Haughn GW, Coen ES. 1995. Parallels between *UNUSUAL FLORAL ORGANS* and *FIMBRIATA*, genes controlling flower development in *Arabidopsis* and *Antirrhinum*. *The Plant Cell* **7**: 1501–1510.

Irish VF, Sussex lM. 1990. Function of the *apetala-1* gene during *Arabidopsis* floral development. *The Plant Cell* **2**: 741–753.

Jack T. 2004. Molecular and genetic mechanisms of floral control. *The Plant Cell* **16**: S1–S17.

Kardailsky I, Shukla VK, Ahn JH, Dagenais N, Christensen SK, Nguyen JT, *et al*. 1999. Activation tagging of the floral inducer *FT*. *Science* **286**: 1962–1965.

Kellogg EA. 2007. Floral displays: genetic control of grass inflorescences. *Current Opinion in Plant Biology* **10**: 26–31.

Kelly AJ, Bonnlander MB, Meeks-Wagner DR. 1995. *NFL*, the tobacco homolog of *FLORICAULA* and *LEAFY*, is transcriptionally expressed in both vegetative and floral meristems. *The Plant Cell* **7**: 225–234.

Kobayashi Y, Kaya H, Goto K, Iwabuchi M, Araki T. 1999. A pair of related genes with antagonistic roles in mediating flowering signals. *Science* **286**: 1960–1962.

Kotoda N, Wada M. 2005. *MdTFL1*, a *TFL1*-like gene of apple, retards the transition from the vegetative to reproductive phase in transgenic *Arabidopsis*. *Plant Science* **168**: 95–104.

Kotoda N, Wada M, Masuda T, Soejima J. 2003. The break-through in the reduction of juvenile phase in apple using transgenic approaches. *Acta Horticulturae (ISHS)* **625**: 337–343.

Lawton-Rauh AL, Buckler IV ES, Purugganan MD. 1999. Patterns of molecular evolution among paralogous floral homeotic genes. *Molecular Biology and Evolution* **16**: 1037–1045.

Leebens-Mack J, Soltis DE, Soltis PS. 2005. Plant reproductive genomics at the plant and animal genome conference. *Comparative and functional genomics* **6**: 159–169.

Liljegren SJ, Gustafson-Brown C, Pinyopich A, Ditta GS, Yanofsky MF. 1999. Interactions among *APETALA1*, *LEAFY*, and *TERMINAL FLOWER1* specify meristem fate. *The Plant Cell* **11**: 1007–1018.

Litt A, Irish VF. 2003. Duplication and diversification in the *APETALA1/FRUITFULL* floral homeotic gene lineage: implications for the evolution of floral development. *Genetics* **165**: 821–833.

Lowman AC, Purugganan MD. 1999. Duplication of the *Brassica oleracea APETALA1* floral homeotic gene and the evolution of domesticated cauliflower. *The American Genetic Association* **90**: 514–520.

Maizel A, Busch MA, Tanahashi T, Perkovic J, Kato M, Hasebe M, Weigel D. 2005. The floral regulator *LEAFY* evolves by substitutions in the DNA binding domain. *Science* **308**: 260–263.

Mandel AM, Yanofsky MF. 1995. A gene triggering flower formation in *Arabidopsis*. *Nature* **377**: 522–524.

Mandel MA, Gustafson-Brown C, Savidge B, Yanofsky MF. 1992. Molecular characterization of the *Arabidopsis* floral homeotic gene *APETALA1*. *Nature* **360**: 273–277.

Mellerowicz EJ, Horgan K, Walden A, Coker A, Walter C. 1998. *PRFLL*, a *Pinus radiata* homologue of *FLORICAULA* and *LEAFY* is expressed in buds containing vegetative shoot and undifferentiated male cone primordia. *Planta* **206**: 619–629.

Mimida N, Goto K, Kobayashi Y, Araki T, Ahn JH, Weigel D, *et al*. 2001. Functional divergence of the *TFL1*-like gene family in *Arabidopsis* revealed by characterization of a novel homologue. *Genes to Cells* **6**: 327–336.

Molinero-Rosales N, Jamilena M, Zurita S, Gomez P, Capel J, Lozano R. 1999. *FALSIFLORA*, the tomato orthologue of *FLORICAULA* and *LEAFY*, controls flowering time and floral meristem identity. *Plant Journal* **20**: 685–693.

Mouradov A, Glassick T, Hamdorf B, Murphy L, Fowler B, Marla S, Teasdale RD. 1998. *NEEDLY*, a *Pinus radiata* ortholog of *FLORICAULA/LEAFY* genes, expressed in both reproductive and vegetative meristems. *Proceedings of the National Academy of Sciences of the USA* **95**: 6537–6542.

Murfet IC. 1992. Garden pea and allies—an update from Hobart. *Flowering Newsletter* **13**: 10–20.

Ohshima S, Murata M, Sakamoto W, Ogura Y, Motoyoshi F. 1997. Cloning and molecular analysis of the *Arabidopsis* gene *TERMINAL FLOWER 1*. *Molecular General Genetics* **254**: 186–194.

Okamuro JK, Caster B, Villarroel R, Van Montagu M, Jofuku KD. 1997. The *AP2* domain of *APETALA2* defines a large new family of DNA binding proteins in *Arabidopsis*. *Proceedings of the National Academy of Sciences of the USA* **94**: 7076–7081.

Ordidge M, Chiurugwi T, Tooke F, Battey NH. 2005. *LEAFY*, *TERMINAL FLOWER1* and *AGAMOUS* are functionally conserved but do not regulate terminal flowering and floral determinacy in *Impatiens balsamina*. *Plant Journal* **44**: 985–1000.

Parcy F. 2005. Flowering: a time for integration. *The International Journal of Developmental Biology* **49**: 585–593.

Parcy F, Nilsson O, Busch MA, Lee I, Weigel D. 1998. A genetic framework for floral patterning. *Nature* **395**: 561–566.

Parcy F, Bomblies K, Weigel D. 2002. Interaction of *LEAFY*, *AGAMOUS* and *TERMINAL FLOWER1* in maintaining floral meristem identity in *Arabidopsis*. *Development* **129**: 2519–2527.

Parkin J. 1914. The evolution of the inflorescence. *Linnean Journal of Botany* **42**: 511–562.

Pillitteri LJ, Lovatt CJ, Walling LL. 2004. Isolation and characterization of a *TERMINAL FLOWER* homolog and its correlation with juvenility in citrus. *Plant Physiology* **135**: 1540–1551.

Pnueli L, Carmel-Goren L, Hareven D, Gutfinger T, Álvarez J, Ganal M, Zamir D, Lifschitz E. 1998. The *SELFPRUNING* gene of tomato regulates vegetative to reproductive switching of sympodial meristems and is the ortholog of *CEN* and *TFL1*. *Development* **125**: 1979–1989.

Pouteau S, Nicholls D, Tooke F, Coen E, Battey N. 1997. The induction and maintenance of flowering in *Impatiens*. *Development* **124**: 3343–3351.

Pouteau S, Tooke F, Battey N. 1998. Quantitative control of inflorescence formation in *Impatiens balsamina*. *Plant Physiology* **118**: 1191–1201.

Prusinkiewicz P, Erasmus Y, Lane B, Harder LD, Coen E. 2007. Evolution and development of inflorescence architectures. *Science* **136**: 1452–1456.

Ratcliffe OJ, Amaya I, Vincent CA, Rothstein S, Carpenter R, Coen ES, Bradley DJ. 1998. A common mechanism controls the life cycle and architecture of plants. *Development* **125**: 1609–1615.

Ratcliffe OJ, Bradley DJ, Coen ES. 1999. Separation of shoot and floral identity in *Arabidopsis*. *Development* **126**: 1109–1120.

Reid JB, Murfet IC. 1984. Flowering in *Pisum*: a fifth locus, *Veg. Annals of Botany* **53**: 369–382.

Reid JB, Murfet IC, Singer SR, Weller JL, Taylor SA. 1996. Physiological-genetics of flowering in *Pisum*. *Cell and Developmental Biology* **7**: 455–463.

Rottmann WH, Meilan R, Sheppard LA, Brunner AM, Skinner JS, Ma C, *et al*. 2000. Diverse effects of overexpression of *LEAFY* and *PTLF*, a poplar (*Populus*) homolog of *LEAFY/FLORICAULA*, in transgenic poplar and *Arabidopsis*. *Plant Journal* **22**: 235–245.

Savidge B. 1996. *Floral meristem specification and floral organ development in Arabidopsis*. PhD dissertation, University of California at San Diego, La Jolla, California, La Jolla, USA.

Schultz EA, Haughn GW. 1991. *LEAFY*, a homeotic gene that regulates inflorescence development in *Arabidopsis*. *Plant Cell* **3**: 771–781.

Schultz EA, Haughn GW. 1993. Genetic analysis of the floral initiation process (FLIP) in *Arabidopsis*. *Development* **119**: 745–765.

Shannon S, Meeks-Wagner DR. 1991. A mutation in the *Arabidopsis TFL1* gene affects inflorescence meristem development. *The Plant Cell* **3**: 877–892.

Shu G, Amaral W, Hileman LC, Baum DA. 2000. *Leafy* and the evolution of rosette flowering in violet cress (*Jonopsidium acaule*, *Brassicaceae*). *American Journal of Botany* **87**: 634–641.

Singer SR, Hsuing LP, Huber SC. 1990. Determinate (*det*) mutant of *Pisum sativum* L. (Leguminosae: Papilionoideae) exhibits an indeterminate growth pattern. *American Journal of Botany* **77**: 1330–1335.

Singer SR, Maki SL, Mullen HJ. 1994. Specification of meristem identity in *Pisum sativum* inflorescence development. *Flowering Newsletter* **18**: 26–32.

Singer S, Sollinger J, Maki S, Fishbach J, Short B, Reinke C, Fick J, Cox L. 1999. Inflorescence architecture: a developmental genetics approach. *The Botanical review* **65**: 385–410.

Sliwinski MK, White MA, Maizel A, Weigel D, Baum DA. 2006. Evolutionary divergence of *LFY* function in the mustards *Arabidopsis thaliana* and *Leavenworthia crassa*. *Plant Molecular Biology* **62**: 279–289.

Soltis DE, Soltis PS. 2003. The role of phylogenetics in comparative genetics. *Plant Physiology* **132**: 1790–1800.

Souer E, van der Krol A, Kloos D, Spelt C, Bliek M, Mol J, Koes R. 1998. Genetic control of branching pattern and floral identity during *Petunia* inflorescence development. *Development* **125**: 733–742.

Southerton SG, Strauss SH, Olive MR, Harcourt RL, Decroocq V Zhu X, *et al*. 1998. Eucalyptus has a functional equivalent of the *Arabidopsis* floral meristem identity gene *LEAFY. Plant Molecular Biology* **37**: 897–910.

Sreekantan L, Clemens J, McKenzie MJ, Lenton JR, Croker SJ, Jameson PE. 2004. Flowering genes in *Metrosideros* fit a broad herbaceous model encompassing *Arabidopsis* and *Antirrhinum. Physiologia Plantarum* **121**: 163–173.

Stebbins GL. 1974. *Flowering plants: evolution above the species sevel.* Cambridge, MA: Harvard University Press.

Tanahashi T, Sumikawa N, Kato M, Hasebe M. 2005. Diversification of gene function: homologs of the floral regulator *FLO/LFY* control the first zygotic cell division in the moss *Physcomitrella patens. Development* **132**: 1727–1736.

Taylor SA, Hofer JM, Murfet IC, Sollinger JD, Singer SR, Knox MR, Ellis TH. 2002. *PROLIFERATING INFLORESCENCE MERISTEM*, a MADS-box gene that regulates floral meristem identity in pea. *Plant Physiology* **129**: 1150–1159.

Tucker SC, Grimes J. 1999. The inflorescence: introduction. *The Botanical Review* **65**: 303–316.

Vijayraghavan U, Prasad K, Meyerowitz E. 2005. Specification and maintenance of the floral meristem: interactions between positively acting promoters of flowering and negative regulators. *Current Science* **89**: 1835–1843.

Wada M, Cao QF, Kotoda N, Soejima J, Masuda T. 2002. Apple has two orthologues of *FLORICAULA/LEAFY* involved in flowering. *Plant Molecular Biology* **49**: 567–577.

Wagner D, Sablowski RWM, Eyerowitz EM. 1999. Transcriptional activation of *APETALA1* by *LEAFY. Science* **285**: 582–584.

Walton EF, Podivinsky E, Wu RM. 2001. Bimodal patterns of floral gene expression over the two seasons that kiwifruit flowers develop. *Physiologia Plantarum* **111**: 396–404.

Weberling F. 1989*a*. *Morphology of flowers and inflorescences*, 1st edn. Cambridge, UK: Cambridge University Press.

Weberling F. 1989*b*. Structure and evolutionary tendencies of inflorescences in the *Leguminosae*. In: Stirton CH, Zarucchi JL, eds. *Advances in legume biology. Monographs in Systematic Botany.* Missouri: Missouri Botanical Garden, **29**: 35–58.

Weigel D, Nilsson O. 1995. A developmental switch sufficient for flower initiation in diverse plants. *Nature* **377**: 495–500.

Weigel D, Alvarez J, Smyth DR, Yanofsky MF, Meyerowitz EM. 1992. *LEAFY* controls floral meristem identity in *Arabidopsis. Cell* **69**: 843–859.

Wigge PA, Kim MC, Jaeger KE, Busch W, Schmid M, Lohmann JU, Weigel D. 2005. Integration of spatial and temporal information during floral induction in *Arabidopsis. Science* **309**: 1056–1059.

Wyatt R. 1982. Inflorescence architecture: how flower number, arrangement, and phenology affect pollination and fruit-set. *American Journal of Botany* **69**: 585–594.

Yeung K, Seitz T, Li S, Janosch P, McFerran B, Kaiser C, *et al* 1999. Suppression of Raf-1 kinase activity and MAP kinase signalling by RKIP. *Nature* **401**: 173–177.

Yoon H-S, Baum DA. 2004. Transgenic study of parallelism in plant morphological evolution. *Proceedings of the National Academy of Sciences of the USA* **101**: 6524–6529.

Yu D, Kotilainen M, Pollanen E, Mehto M, Elomaa P, Helariutta Y, Albert VA, Teeri TH. 1999. Organ identity genes and modified patterns of flower development in *Gerbera hybrida* (Asteraceae). *Plant Journal* **17**: 51–62.

Yu H, Xu Y, Tan EL, Kumar PP. 2002. *AGAMOUS-LIKE 24*, a dosage-dependent mediator of the flowering signals. *Proceedings of the National Academy of Sciences of the USA* **99**: 16336–16341.