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A common mechanism controls the life cycle and architecture of plants

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SUMMARY

The overall aerial architecture of flowering plants depends on a group of meristematic cells in the shoot apex. We demonstrate that the *Arabidopsis TERMINAL FLOWER 1* gene has a unified effect on the rate of progression of the shoot apex through different developmental phases. In transgenic *Arabidopsis* plants which ectopically express *TERMINAL FLOWER 1*, both the vegetative and reproductive phases are greatly extended. As a consequence, these plants exhibit dramatic changes in their overall morphology, producing an enlarged vegetative rosette of leaves, followed by a highly branched inflorescence which eventually forms normal flowers. Activity of the floral meristem identity genes *LEAFY* and *APETALA 1* is not directly inhibited by *TERMINAL FLOWER 1*, but their upregulation is markedly delayed compared to wild-type controls. These phenotypic and molecular effects complement those observed in the *tfl1* mutant, where all phases are shortened. The results suggest that *TERMINAL FLOWER 1* participates in a common mechanism underlying major shoot apical phase transitions, rather than there being unrelated mechanisms which regulate each specific transition during the life cycle.

Key words: Plant architecture, Phase change, *TERMINAL FLOWER 1* (*TFL1*), Inflorescence meristem, *LEAFY* (*LFY*), *APETALA 1* (*AP1*), Flower development

INTRODUCTION

The life cycle of most organisms can be divided into several developmental phases, from an early juvenile period when resources are built up, through to a reproductive phase (Poethig, 1990; Schultz and Haughn, 1993; Telfer et al., 1997). In the case of *Arabidopsis*, wild-type plants pass through three main phases, reflecting a sequence of identities in the shoot apical meristem (Figs 1, 2). Following germination the meristem has a vegetative (V) identity and produces leaf primordia on its flanks to form a rosette. The length of this V phase depends on the environmental conditions, and is regulated through a complex network of flowering-time genes (Martinez-Zapater et al., 1994). On receipt of favourable signals, reproductive development is initiated and the apical meristem acquires an inflorescence identity (I), which itself has 2 distinct phases. During the first-inflorescence phase (I_1), 2-3 cauline leaf primordia are produced which subtend axillary inflorescence meristems. This is followed by a second-inflorescence phase (I_2), during which determinate floral meristems are made on the periphery of the apex (Figs 1, 2). According to an alternative viewpoint, I_1 can be considered as part of the V phase, such that the apex switches directly to floral meristem production at the end of V (Hempel and Feldman, 1994). In this study, however, to allow a precise description of different plant phenotypes, it was essential to distinguish the period of cauline leaf initiation from other phases of growth, and for that reason we defined this period as I_1. In both descriptions, the fate of floral meristems depends on the action of meristem identity genes such as *LEAFY* (*LFY*) and *APETALA1* (*AP1*) (Mandel et al., 1992; Weigel et al., 1992; Bowman et al, 1993; Gustafson-Brown, 1994; Mandel and Yanofsky, 1995; Weigel and Nilsson, 1995). The inflorescence apex itself remains indeterminate and continues to form floral meristems on its periphery until senescence occurs.

TERMINAL FLOWER 1 (*TFL1*) is a key gene which affects the developmental phases and architecture of *Arabidopsis* (Shannon and Ry Meeks-Wagner, 1991; Schultz and Haughn, 1991; Alvarez et al, 1992; Ray et al., 1996). The most notable feature of the *tfl1* mutant is that a flower forms at the tip of the inflorescence, unlike the wild-type apex which grows indeterminately (Fig. 1). In accordance with this, *TFL1* expression is observed at high levels in the wild-type inflorescence apex (Bradley et al., 1997). Additionally, in *tfl1* mutants, *LFY* and *AP1* expression can be observed in the inflorescence apical meristem, correlating with its floral identity, whereas in wild type, expression of both genes is restricted to young floral meristems on the periphery of the apex (Weigel et al., 1992; Bowman et al., 1993; Gustafson-Brown et al., 1994; Bradley et al., 1997).

In addition to the terminal flower phenotype, the *tfl1* mutant also has a significantly shorter vegetative phase than wild type, due to an earlier commitment to reproductive growth (Schultz

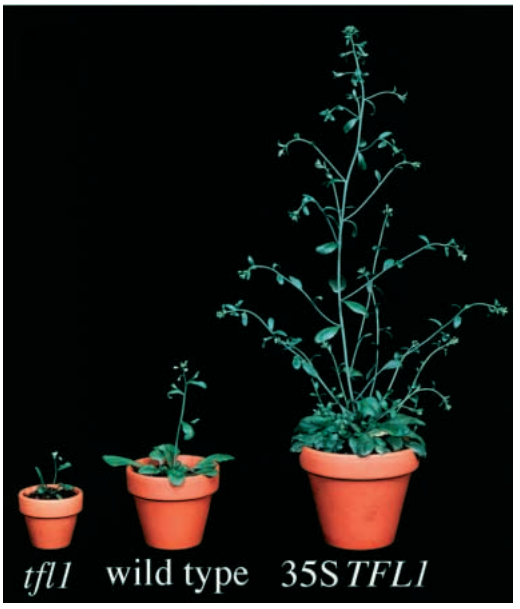

Fig. 1. Morphology of the *tfl1* mutant, wild-type and 35S*TFL1* *Arabidopsis* plants. The *tfl1* mutant is shown at 20 days, compared to wild type at 30 days and 35S*TFL1* at 60 days (all were grown under LD conditions of 16 hours light / 8 hours dark).

and Haughn, 1991; Shannon and Meeks-Wagner, 1991; Bradley et al., 1997). This correlates with *TFL1* expression in vegetative apices, although this is weaker than in inflorescence meristems. The *TFL1* gene product therefore has two effects: firstly it delays the switch to reproductive growth (flowering time) and secondly it prevents the formation of a terminal flower (Bradley et al., 1997).

The effects of *TFL1* could be explained in two ways. In one model, *TFL1* may have a dual function and act differently at separate times in the life cycle, affording a link between the regulation of flowering time and meristem identity. First it acts to control flowering time and secondly it acts by a separate mechanism to inhibit the activity of the floral meristem identity genes, *LFY* and *AP1*, in the apex (Shannon and Meeks-Wagner, 1991, 1993; Hicks et al., 1996; Bradley et al., 1997). An alternative view is that *TFL1* could have a single function influencing the rate of progression through all phases of the shoot apical meristem (Bowman et al., 1993; Schultz and Haughn, 1993). According to this second model, the *tfl1* apical meristem progresses more rapidly through each phase (V, I_1 and I_2) such that it then enters a fourth terminal phase with a floral (F) identity, which is not normally attained before senescence occurs in wild type.

To distinguish between these possibilities, we produced transgenic *Arabidopsis* plants in which *TFL1* was ectopically expressed at all stages of the life cycle. If the dual function model was applicable, we anticipated that these plants might show a considerable delay in flowering time and form an inflorescence comparable to that of a *lfy;ap1* double mutant, which never produces normal flowers (Weigel et al., 1992; Schultz and Haughn, 1993). By contrast, if *TFL1* fulfilled a single function influencing the rate of phase change, then there would be a consistent lengthening of all phases, but normal flowers would eventually be produced.

We show by a combination of molecular and physiological analyses that *TFL1* affects all phases of the *Arabidopsis* life cycle. Transgenic plants expressing *TFL1* throughout development display extended V and I_1 phases compared to wild-type controls but do ultimately produce a normal I_2 phase and fertile flowers. Consistent with this, we show by in situ hybridisation, that the upregulation of *LFY* and *AP1* is delayed in comparison to wild-type plants. We therefore conclude that *TFL1* does not function by directly restricting the activity of the meristem identity genes *LFY* and *AP1*, but rather modulates the mechanism of shoot phase transitions. These results are consistent with *TFL1* having a single function, over the entire life cycle, to maintain the duration of growth phases of the *Arabidopsis* shoot apex.

MATERIALS AND METHODS

Construction of transgenes

The *TFL1* gene (EST 129D7T7) was obtained from the *Arabidopsis* Biological Resource Centre at Ohio State University in pZL1. This was digested with *Sma*I and *Xba*I to release the *TFL1* cDNA. The *TFL1* cDNA fragment was ligated into plasmid SLJ4K1 (Jones et al., 1992) containing the CaMV 35S promoter, after this had been cut with *Cla*I, filled in, and then digested with *Xba*I. The product of the ligation was named pSR15. The 35S*TFL1* fusion was released from pSR15 by digestion with *Eco*RI and *Hin*dIII and ligated into the binary vector SLJ 44024A (Jones et al., 1992), which conferred kanamycin resistance, to give pJAM 2076.

Arabidopsis transformation

All transformation experiments were performed on *Arabidopsis* plants of the ecotype *Columbia*. *Agrobacterium* mediated transformation with pJAM 2076 was attempted by both root transformation (Valvekans et al., 1988) and vacuum infiltration of flowers (Bechtold et al., 1993). Approximately 30 primary transformants derived from root transformation were established on soil. These often displayed excessive vegetative growth and sometimes produced aerial rosettes. However, it was difficult to establish whether these were genuine features or artifacts of tissue culture. Three primary root transformants set seed and could be analyzed in future generations. A further 3 primary transformants were obtained from vacuum infiltration, all of which set seed. A total of 6 lines were therefore available and all displayed similar phenotypes in subsequent generations. Two representative independent lines, JI.At1 and JI.At2, each derived from a T_2 plant which contained the 35S*TFL1* insertion at a single locus, were selected for further study. All subsequent analysis described was performed on the T_3 population from these plants.

Plant growth conditions

For all experiments, seeds were imbided and stratified for 5 days at 4°C in the dark, then germinated and grown on soil in growth cabinets at 20°C In the photoperiod shift experiments to assess commitment, 35S*TFL1* plants of line JI.At1 were grown in long days (16 hours light/8 hours dark, 90-120 $\mu E\ m^{-2}\ s^{-1}$) and transferred to short days (8 hours light/16 hours dark) after 8, 12, or 19 days. Plants were also grown in continuous long days and short days as controls. Numbers of rosette leaves were counted for approximately 10 plants in each batch. Errors indicate standard error about the mean with 95% confidence limits attached.

Scanning electron microscopy (SEM)

Shoot apices were moulded as described by Green and Linstead (1990). Casts of the moulds were then sputter coated with gold and viewed and photographed using a scanning electron microscope.

In situ hybridisation experiments

Longitudinal sections of plant tissue were probed with digoxigenin-labelled *TFL1*, *LFY*, or *AP1* antisense RNA. These probes were made using plasmids pJAM 2045 (Bradley et al.,1997), pDW122 (kindly provided by Detlef Weigel; Weigel et al., 1992), and pKY89 (kindly provided by Martin Yanofsky; Mandel et al., 1992) respectively. RNA signal was detected as a dark blue/black colour on a light blue background when viewed under the light microscope. In situ hybridisation was carried out as described by Coen et al. (1990).

RESULTS

Phenotypes of the 35S*TFL1* lines

We established six independent lines in which *TFL1* was expressed from the 35S CaMV promoter (Odell et al., 1985), all of which displayed similar dominant and heritable phenotypes (Fig. 1). These plants had greatly extended V and I_1 phases: under long day (LD) conditions, the V phase was 2-3 times longer, and the I_1 phase 15-20 times longer than in wild type (Fig. 2). The latter two thirds of the I_1 phase displayed a novel region (I_1*), not observed in wild type, in which axillary shoots were not subtended by cauline leaves (Fig. 3A,C,D). These shoots became progressively more flower-like towards the apex; the uppermost typically comprising small clusters of flowers surrounded by leaf-like organs arranged in a whorled phyllotaxy (Fig. 3C). Sometimes such structures consisted of a flower with organ abnormalities and additional flowers buds in the axils of those organs (Fig. 3B). Eventually the 35S*TFL1* apex entered an I_2 phase during which it produced apparently normal fertile flowers (Fig. 3C). Occasionally, when the plants were very old, the shoot apex passed into a floral (F) phase, terminating in a flower (data not shown). The growth pattern of the primary apex was repeated by secondary shoots, (i.e. those growing from the axils of cauline and rosette leaves) giving the plant a highly branched architecture. This extensive branching resulted in the 35S*TFL1* plants becoming much larger and producing a much greater quantity of seed than wild type and *tfl1* mutants. Since they all grew at approximately the same rate, it took proportionately longer for the 35S*TFL1* lines to set seed: the *tfl1* mutant produced ripe seed by about 30 days, wild type by about 45 days, whilst the transgenics required 80-100 days.

In short day (SD) conditions, the 35S*TFL1* lines produced approximately twice as many rosette leaves as long day grown plants indicating that they were still sensitive to photoperiod. However, after bolting the inflorescence phenotypes were very variable between individuals of the same line. Some plants produced an inflorescence similar to those grown in long days but with a slightly increased number of nodes in the I_1 and I_1* phases before making normal flowers. One individual, however, remained in the I_1 phase, making over 50 cauline leaf nodes, and did not form flowers even after seven months. Other individuals also failed to produce flowers in a seven month period but produced large aerial rosettes during the I_1 phase. These aerial structures sometimes consisted of more that 20

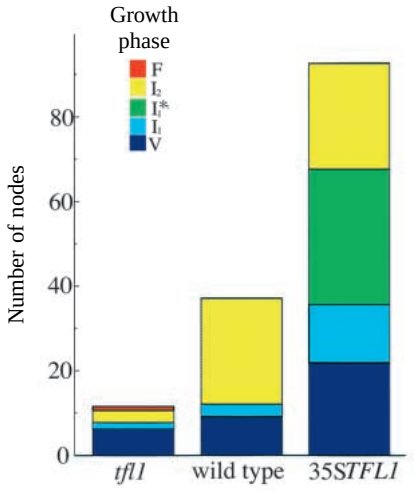

Fig. 2. Effect of *TFL1* activity on growth phase duration of the primary shoot apical meristem. Coloured bars indicate the number of nodes produced during each phase of the shoot apical meristem. V, vegetative phase where rosette leaves are produced; I_1, first-inflorescence phase where shoots with a subtending cauline leaf are produced; I_1*, phase where shoots lacking a subtending cauline leaf are produced; I_2, phase where normal flowers are produced; F, floral phase where apex itself becomes a flower. Under long day conditions, *tfl1* produced 6.3±0.5 rosette leaves, 1.5±0.3 cauline leaves, 2.8±0.5 flowers and 1 terminal flower. Wild type produced 9.2±0.7 rosette leaves, 2.9±0.3 cauline leaves, and approximately 20-30 flowers, before senescence. A representative line of 35S*TFL1* (JI.At1), produced 21.9±0.9 rosette leaves, 13.7±0.8 cauline leaves subtending shoots, 32±2 shoots lacking cauline leaves, and around 20-30 flowers prior to senescence. Approximately 10-20 plants were counted in each batch.

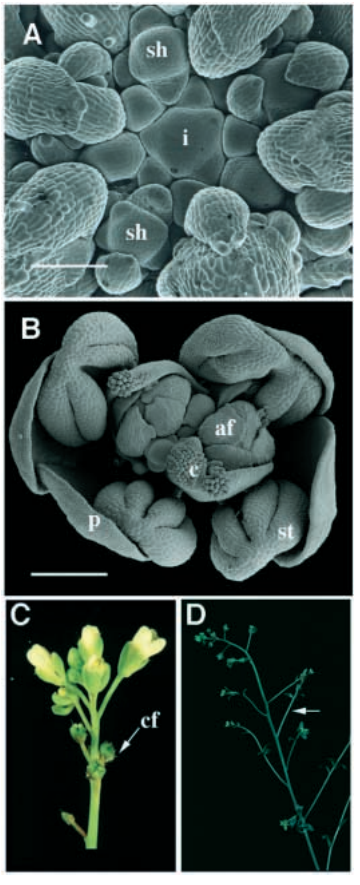

Fig. 3. Nodes produced during the I_1* and I_2 phases of 35S*TFL1* plants. (A) Scanning electron micrograph (SEM) showing the shoot apex during the I_1* phase, at 40 days. Note the shoots (sh) produced by the inflorescence meristem (i). (B) SEM, taken at 80 days, showing uppermost flower-like shoot (initiated by apex at end of I_1*). Note the petals (p), stamens (st), carpelloid tissue (c) and flowers formed in the axils of floral organs (af). (C) The shoot apex at 80 days. Note the normal flowers, which were initiated in the I_2 phase, and the small clusters of flowers (cf) which were initiated at the end of I_1*. (D) Shoots lacking cauline leaves (arrow) at 60-70 days, which were initiated during the I_1* phase. Scale bars, 90 μm in A, 160 μm in B.

leaves and developed at each axillary position instead of a single cauline leaf bearing a secondary inflorescence.

Timing of growth phases in the 35S*TFL1* lines

The eventual appearance of normal flowers in long day grown 35S*TFL1* plants indicated that *TFL1* most likely acted through a common mechanism to extend the duration of phases. The appearance of an extra phase, I_1*, intermediate between I_1 and I_2 could also be accounted for by a general retardation in the rate of phase change.

To test this model further, patterns of meristem identity gene expression were examined in relation to the various phases. The timing of phase transitions was determined by monitoring the total number of nodes made by the primary shoot apex of 35S*TFL1* plants over time, by dissection and scanning electron microscopy (Fig. 4). By comparison with the total numbers of different structures visible on the main axis of adult plants (Fig. 2), it was possible to deduce time-points at which the phase transitions had taken place.

This analysis showed that 35S*TFL1* plants switch from making V to I_1 nodes at approximately 13-15 days after sowing, a delay of about 7 days compared to wild type (Bradley et al., 1997). To check that this was due to a delay in commitment to reproductive growth, batches of plants were moved from inductive (long days, LD), to non-inductive (short days, SD) conditions, at various times during their development. In SD regimes, 35S*TFL1* plants made about twice as many rosette leaves (41±6) compared to similar plants in LD conditions (21.9±0.9 rosette leaves). Those batches that received 8 or 12 inductive days had not been committed and produced 44±7 and 38±4 rosette leaves respectively. This was not significantly fewer than for the non-induced controls. Eventually 35S*TFL1* plants did become committed to reproductive growth, as revealed by batches receiving 19 LD which produced 21±2 rosette leaves. Wild-type controls given the same treatment made about 10 rosette leaves for the 8 and 12 day time-points.

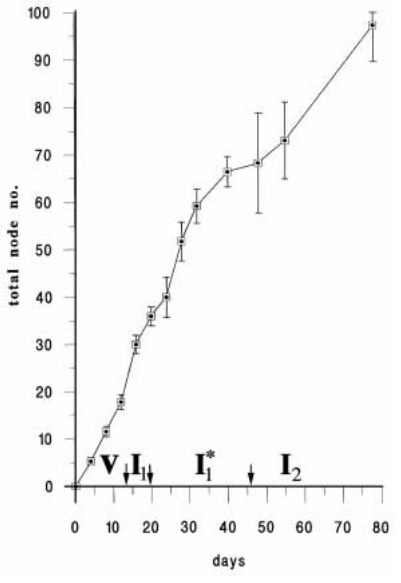

Fig. 4. Rate of node initiation by primary apices of 35S*TFL1* plants in long day conditions. Arrows indicate approximate times of growth phase transitions, based on total numbers of each different type of node visible on the primary axis of mature plants. The total number of nodes made by the primary shoot apex at each time-point was assessed by counting from the most recently initiated primordium (visible by SEM), to the lowermost rosette leaf (excluding cotyledons). Approximately 5 plants were counted at each time-point. Error bars indicate standard error of mean with 95% confidence limits attached. The rate of node initiation (plastochron) was similar to wild type, with approximately 1.5 nodes produced per day.

The 35S*TFL1* plant apex continued in the I_1 phase until about day 20, after which it entered the novel I_1* phase, during which it produced shoots lacking cauline leaves. Finally at around 45-50 days, it switched to I_2, and initiated normal floral meristems. The I_1*-I_2 transition was not an abrupt switch, but occurred gradually, as indicated by the increasingly flower-like nature of structures produced by the inflorescence meristem (Fig. 3). To examine how this related to the activity of floral meristem identity genes, expression of *LFY* and *AP1* was compared to the timing of the different phases in wild-type and transgenic plants.

TFL1 delays upregulation of *LFY* and *AP1*

In wild type, small quantities of *LFY* RNA were detected in rosette and cauline leaf primordia of the V and I_1 phases (data not shown; Bradley et al., 1997; Blazquez et al., 1997, Hempel et al., 1997). Strong *LFY* expression was first observed at 10-12 days from sowing when the plant apex was in early I_2 (Fig. 5). *AP1* was upregulated slightly later than *LFY,* at 12-14 days from sowing, and was not observed during wild-type vegetative growth.

In 35S*TFL1* plants, only weak *LFY* expression was noted during the V and I_1 phases, comparable to the levels observed in the V and I_1 phases of wild type. Control sections probed with *TFL1* showed that regions expressing *LFY* overlapped with ectopic *TFL1*, although expression from the 35S promoter was somewhat patchy (Fig. 5). This suggested that *TFL1* did not have a distinct role as an overall repressor of *LFY* expression. At 26 days, just after the start of the I_1* phase (Fig. 4), *AP1* expression was not detected and *LFY* expression was still weak. Later in the I_1* phase, at the 35 day time-point, strong *LFY* expression was observed but *AP1* RNA was still absent (Fig. 5). The gradient of structures observed in the I_1* phase was consistent with such a gradual increase in meristem identity gene activity. Eventually strong expression of both *LFY* and *AP1* was observed in young floral meristems during I_2, as seen at the 59 day time-point. Again these genes were co-expressed in many regions containing *TFL1* RNA. It appears, therefore, that *TFL1* delays the upregulation of *LFY* and *AP1* expression but cannot directly block their activity.

DISCUSSION

We have shown that *TFL1* regulates the duration of growth phases of the shoot apex, and as a consequence, the overall morphology of *Arabidopsis* plants. Previous models for the action of *TFL1* have suggested interactions with the flowering-time genes during the vegetative phase and a second role antagonising *LFY* and *AP1* in the shoot apical meristem. We propose that *TFL1* acts by influencing a central mechanism

controlling the identity of shoot apical meristem at all stages of development throughout the life cycle.

TFL1 action under inductive and non-inductive conditions

Our main analyses were conducted under inductive long day conditions. However, the phenotype of the 35S*TFL1* lines did become more severe in short days, confirming that other factors act to extend the *Arabidopsis* life cycle, independently of *TFL1*, in non-inductive conditions. Furthermore, the increase in rosette leaf number of 35S*TFL1* lines in SD compared to LD conditions, indicated that their extended vegetative phase and delayed flowering was not merely due to inhibition of flowering-time genes such as *CONSTANS* which mainly function to promote reproduction in long days (Koornneef et al., 1991; Putterill et al., 1995; Hicks et al., 1996; Simon et al., 1996).

Interactions between *TFL1* and the meristem identity genes *LFY* and *AP1*

LFY is expressed in young leaf primordia at low levels during the wild-type vegetative phase and is upregulated during reproductive development (Bradley at al, 1997; Blazquez et al., 1997; Hempel et al., 1997). In the 35S*TFL1* lines low level *LFY* expression persisted much longer, and upregulation was not observed until around 23 days after that of the wild type. *AP1* upregulation was even later and did not occur until about 40-45 days after that in wild type. Therefore, *TFL1* delays upregulation of floral meristem identity genes, rather than acting as a direct inhibitor throughout the life cycle.

It is also noteworthy that the upregulation of *AP1* was significantly delayed compared to *LFY*. These genes are induced via separate pathways with *AP1* closely following *LFY* induction (Simon et al., 1996). That *AP1* upregulation was uncoupled from *LFY* upregulation, suggests that *TFL1* impinges more stringently on the pathway governing *AP1* activation than on that controlling *LFY*. Recent data suggests that this pathway could involve the activation of *AP1* by genes such as *FT* and *FWA* (Ruiz-Garcia et al., 1997).

Newly initiated floral meristems arise from the periphery of the inflorescence meristem. It has been suggested that floral meristem identity genes such as *LFY* and *AP1* act as mutual antagonists with *TFL1*, to ensure that floral meristems pursue a separate determinate growth pattern from the indeterminate inflorescence meristem which produced them (Shannon and Meeks-Wagner, 1993; Schultz and Haughn, 1993). This stand-point is supported by the complementary phenotypes of the *tfl1* mutant (where flowers replace inflorescences) when compared to those of *lfy* and *ap1* mutants (where inflorescences replace flowers). Additionally, lines which ectopically express *LFY* or *AP1* from a 35S promoter, broadly phenocopy the *tfl1* mutant (Weigel and Nilsson, 1995; Mandel and Yanofsky, 1995). Although we have demonstrated that *TFL1* does not directly inhibit *LFY* or *AP1*, it is still not clear if *LFY* or *AP1* can directly inhibit *TFL1*. Answering this question awaits a study of *TFL1* expression in the 35S*LFY* and 35S*AP1* plants.

TFL1 retards phase change in the shoot apex

A key factor which has become apparent from this work is that *TFL1* does not have two separate functions, of first delaying reproduction and then inhibiting *LFY* and *AP1* in the apex.

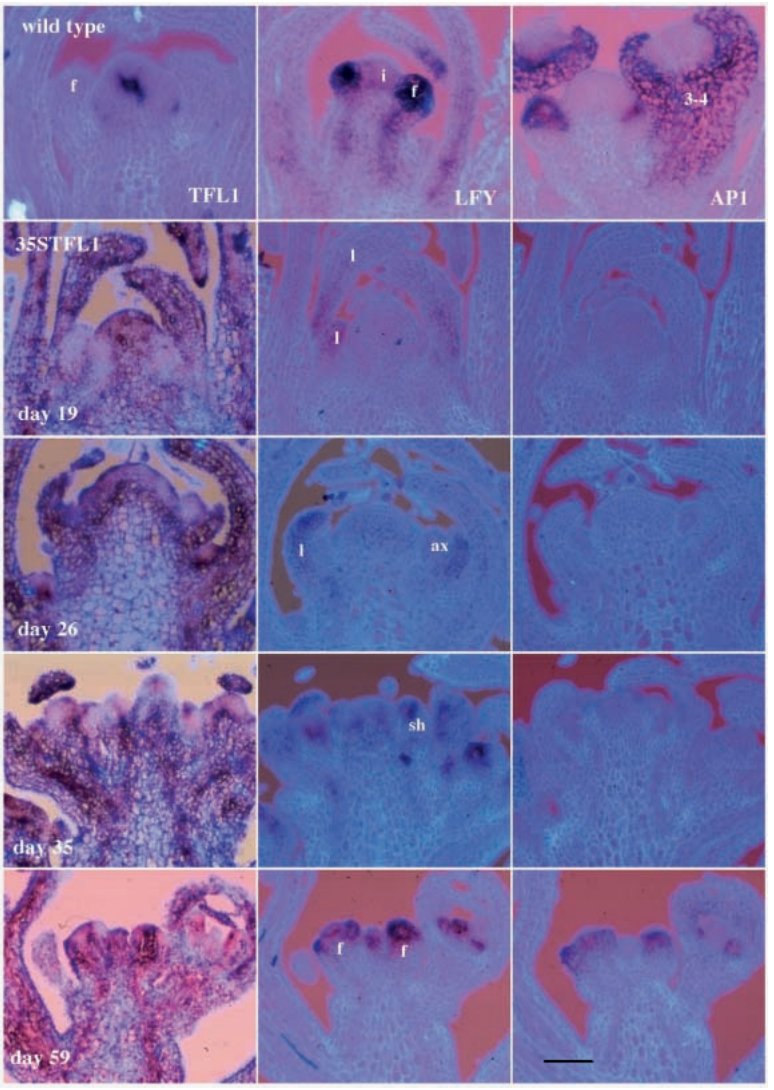

Fig. 5. Expression pattern of *TFL1*, *LFY* and *AP1* in wild type and 35S*TFL1* plants. The top row shows typical expression patterns in wild-type apices at 10-14 long days after sowing. Note *TFL1* expression in the inflorescence (i), *LFY* expression in young (approx stage 2; Smyth et al., 1990) floral meristems (f), and *AP1* expression in young floral meristems (f) and outer whorls of developing stage 3-4 flower (3-4). Bottom 4 rows compare expression of *TFL1*, *LFY* and *AP1* in adjacent sections from apices of 35S*TFL1* harvested at the time-points indicated. Note the low levels of *LFY* RNA in leaf primordia (l), overlapping regions containing *TFL1* RNA, and the absence of *LFY* in axillary shoot meristems (ax), at the 19 and 26 day time-points. Note, also, the absence of *AP1* RNA at those times. At the 35-day time-point, *LFY* was strongly expressed in shoots (sh), which lacked a subtending cauline leaf, and *AP1* RNA was still absent. At the 59 day time-point *LFY* and *AP1* RNA were present at high levels in young floral meristems (f), again overlapping regions of high *TFL1* expression. Scale bar, approximately 50 μm.

Rather, the results show that *TFL1* has a unified effect on the rate of progression through all the different phases of the shoot apex. This implies that there is a common mechanism underlying phase transitions during the life cycle, rather than there being unrelated mechanisms specific to each transition. In a similar way, common components, such as *cdc2*, are involved in progression through different phases of the yeast cell cycle (Forsburg et al., 1991). The complementary effects of accelerating phase transitions in the *tfl1* mutant, and of retarding transitions in the 35S*TFL1* lines, indicate that *TFL1* activity is both necessary and sufficient to influence this common process. By acting in this way, *TFL1* can have profound consequences for the life cycle and overall architecture of the plant, modulating its degree of branching and determinacy.

The TFL1 protein is similar to phosphatidylethanolamine binding proteins (PBPs) of animals which can bind to membrane protein complexes (Bradley et al., 1996, 1997; Ohshima et al., 1997). This suggests that *TFL1* might be involved in a signalling process, perhaps influencing the response to signals arriving at the plant apex. The effects of *TFL1* might eventually be overcome if such signals were to reach a sufficiently high level; possibly accounting for the transition to a floral identity that sporadically occurs in very old 35S*TFL1* apices. *Centroradialis* (*cen*), a homologue of *TFL1* which also has similarity to PBPs, is found in another flowering plant, *Antirrhinum majus* (Bradley et al., 1996). However, *cen* only appears to prevent the transition of the apex from an inflorescence to floral identity, and does not notably affect earlier developmental phases. It is possible that *cen* had an ancestral role similar to that proposed for *TFL1* but that its involvement in other phase transitions has become redundant.

Perspectives

The production of a terminal flower is thought to be an ancestral state from which the indeterminate condition evolved (Stebbins, 1974). By accelerating progression through growth phases, the *tfl1* mutant, in effect, recapitulates this aspect of the ancestral form. The evolution of an indeterminate inflorescence from an ancestor with a determinate inflorescence might be regarded as an example of neoteny (De Beer, 1940); a process in which juvenille traits persist into later periods of the life cycle. According to this view, the indeterminate growth of the wild-type *Arabidopsis* apex may have arisen from *TFL1* activity retarding its progression and ensuring that it never reaches the mature determinate floral phase, exhibited by the ancestor.

The *tfl1* mutant and the 35S*TFL1* plants illustrate different reproductive strategies. The *tfl1* mutant passes through its life cycle very rapidly but produces relatively small amounts of seed. By contrast 35S*TFL1* plants grow for much longer and eventually produce a larger quantity of seed. In the same way, some plants have a very short life-span, whereas others, such as trees, accumulate substantial reserves over many years before reproducing. It is possible that variation in levels of *TFL1*-like gene activity underlies some of these differences and it will therefore be interesting to examine how *TFL1*-like genes function in diverse species.

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