25 Schubert, H. *et al.* (1995) *In situ* measurement of state transition in cyanobacterial blooms: kinetics and extent of the state change in relation to underwater light and vertical mixing. *Mar. Ecol. Prog. Ser.* 128, 99–108
26 Arsalane, W. *et al.* (1994) Influence of the pool size of the xanthophyll cycle on the effects of light stress in a diatom: competition between photoprotection and photoinhibition. *Biochem. Photobiol.* 60, 237–243
27 Olaizola, M. *et al.* (1994) Non-photochemical fluorescence quenching and the diadinoxanthin cycle in a marine diatom. *Photosynth. Res.* 41, 357–370
28 Casper-Lindley, C. and Björkman, O. (1998) Fluorescence quenching in four unicellular algae with different light-harvesting and xanthophyll-cycle pigments. *Photosynth. Res.* 56, 277–289
29 Kana, T.M. (1992) Relationship between photosynthetic oxygen cycling and carbon assimilation in *Synechococcus* WH7803 (Cyanophyta). *J. Phycol.* 28, 304–308
30 Lomas, M.W. and Glibert, P.M. (1999) Temperature regulation of nitrate uptake: a novel hypothesis about nitrate uptake and reduction in cool-water diatoms. *Limnol. Oceanogr.* 44, 556–572
31 Neidhardt, J. *et al.* (1998) Photosystem-II repair and chloroplast recovery from irradiance stress: relationship between chronic photoinhibition, light-harvesting chlorophyll antenna size and photosynthetic productivity in *Dunaliella salina* (green algae). *Photosynth. Res.* 56, 175–184
32 Anderson, J.A. *et al.* (1998) Unifying model for the photoinactivation of photosystem II *in vivo* under steady-state conditions. *Photosynth. Res.* 56, 1–13
33 Barkmann, W. and Woods, J.D. (1996) On using a Lagrangian model to calibrate primary production determined from *in vitro* incubation measurements. *J. Plankt. Res.* 18, 767–788
34 Franks, P.J.S. and Marra, J. (1994) A simple new formulation for phytoplankton photoresponse and an application in a wind-driven mixed-layer model. *Mar. Ecol. Prog. Ser.* 111, 143–153
35 Neale, J.P. *et al.* (1998) Inhibition of marine photosynthesis by ultraviolet radiation: variable sensitivity of phytoplankton in the Weddell–Scotia confluence during the austral spring. *Limnol. Oceanogr.* 43, 433–448
36 Taylor, A.H. *et al.* (1997) Seasonal and latitudinal dependencies of phytoplankton carbon-to-chlorophyll *a* ratios: results of a modelling study. *Mar. Ecol. Prog. Ser.* 152, 51–66

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Floral induction and determination: where is flowering controlled?

Frederick D. Hempel, David R. Welch and Lewis J. Feldman

Flowering is controlled by a variety of interrelated mechanisms. In many plants, the environment controls the production of a floral stimulus, which moves from the leaves to the shoot apex. Apices can become committed to the continuous production of flowers after the receipt of sufficient amounts of floral stimulus. However, in some plants, the commitment to continued flower production is evidently caused by a plant's commitment to perpetually produce floral stimulus in the leaves. Ultimately, the induction of flowering leads to the specification of flowers at the shoot apex. In *Arabidopsis*, floral specification and inflorescence patterning are regulated largely by the interactions between the genes *TERMINAL FLOWER*, *LEAFY* and *APETALA1/CAULIFLOWER*.

Floral induction is the process by which stimuli originating outside the shoot apex induce the formation of flower primordia (Fig. 1). The photoperiodic induction of flowering was discovered 86 years ago by Julien Tornois in hops[1]. Shortly afterwards, additional experiments suggested that the photoperiodic control of flowering was a general phenomenon, which controlled flowering in most plants[2]. Later, focused-light experiments showed that leaves perceive photoperiodic signals[3]. These studies, and numerous grafting experiments, indicate that the production of the photoperiod-induced floral stimulus[4] occurs in the leaves of a wide variety of flowering plants[5–7].

In contrast with floral induction, floral determination can be defined as the assignment of flower(ing) fate, which is persistent even when the flower-inducing conditions no longer exist[8,9]. Assays for floral determination include:

- Changing environmental conditions (from inductive to non-inductive).
- Microsurgical removal of shoot apices, and the placement of those apices into neutral environments[8,10].

However, both types of determination assay have limitations, and it is important to note that different determination assays might yield alternative conclusions for the same primordia (the caveats associated with determination experiments are discussed in Ref. 11). A third type of assay has been used to test leaf commitment to the continued production of floral stimulus: in this assay, photoinduced leaves are removed from the plant following an inductive treatment[12].

In this review we discuss firstly a variety of experiments that indicate the site(s) that control flowering. Secondly, we review recent studies that indicate how a few key molecular players regulate the specification of flower primordia in *Arabidopsis*.

Floral determination assays

Photoperiodic assays for floral determination

The simplest type of determination assay is one in which plants are moved to non-inductive conditions after various lengths of time under inductive conditions. Using this method, the duration of photoinduction treatment required to produce flowers can be

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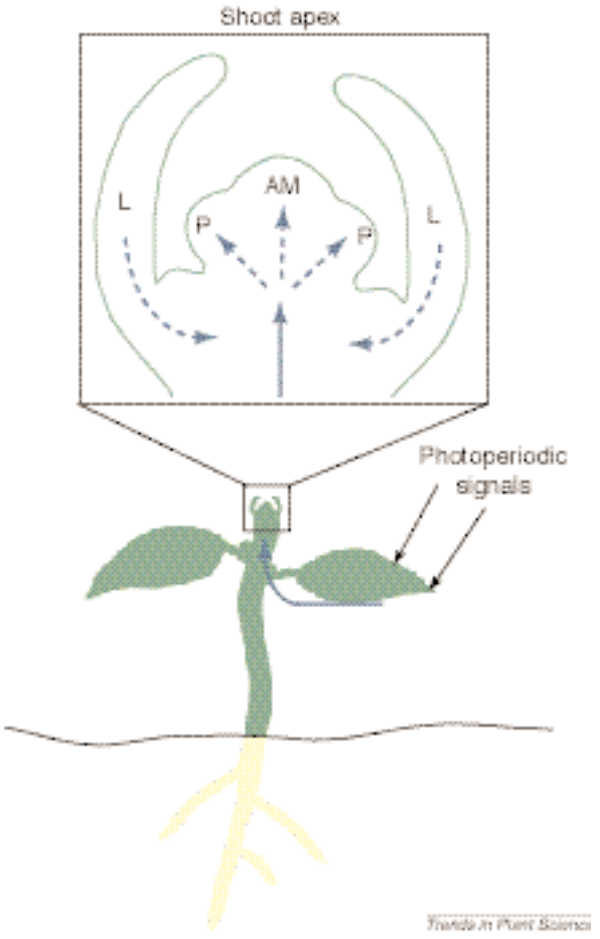

Fig. 1. During the induction of flowering a floral stimulus (unbroken, blue arrows) moves from the leaves to the shoot apex. Photoperiodic flower-inducing signals are perceived in the leaves, which produce the floral stimulus. Once this stimulus reaches the shoot apex it might act on the apical meristem (AM), or it might act directly on developing primordia (P). If the determination of flowering is controlled by the apical meristem, this implies that the meristem responds directly to the floral stimulus. If the leaves control the commitment to flowering, it is possible that the floral stimulus acts directly on developing primordia and/or on the shoot meristem (blue, broken arrows indicate the potential pathway for floral stimulus). Young leaves (L) are also a potential site for floral stimulus production, although they might have to develop further before they are able to do so. In apex-removal determination assays these young leaves would be transplanted as part of the shoot apex.

deduced[13–15]. A major limitation of this type of determination assay is that 'determination' is measured at the level of the whole plant. Therefore, it is not possible to draw strict conclusions about when individual primordia become irreversibly committed to develop as flowers. Thus, it is also not possible to deduce whether an irreversible commitment to flowering is because of a commitment to flowering within the shoot meristem, or if it is because of a commitment to the production of floral stimulus by the leaves (Fig. 1). However, the two types of assays discussed next are designed to help make this distinction.

Microsurgical assays for florally determined shoot apices

In the past two decades, microsurgery experiments have been used to assay for floral determination in shoot apices[16,17]. Using a grafting assay (Fig. 2)[16], axillary shoot apices from various regions of the plant are removed and grafted to rootstocks. Grafted apices that have not been determined to flower produce approximately the same number of vegetative nodes as a normal plant. However, grafted apices that have been determined to flower, produce flowers much more rapidly, indicating that they were committed to flower before they were removed from the original plant.

Because meristems cannot be removed and grafted without the inclusion of a few young leaves or leaf primordia, one potential limitation of a grafting experiment is that it does not conclusively indicate whether meristems have been determined. Interestingly, the expression of two genes involved in the control of flowering, *indeterminate* in maize and *CONSTANS* in *Arabidopsis*, occurs in young leaves[18,19]. The expression of these flower-promoting genes in young leaves suggests that leaves that are removed along with the shoot meristem might be able to affect the determination status of the shoot apex. It is possible that, in some cases, these leaves can be induced to produce sufficient floral stimulus to affect the determination status of the shoot apex (Fig. 1).

Assays for leaves that are committed to the persistent production of floral stimulus

Leaf removal experiments can be used to test for a commitment to persistent floral stimulus production in the leaves. In red-flowered *Impatiens balsamina*, it has been shown that a continuous supply of a leaf-derived floral stimulus is needed to maintain floral identity in developing flower primordia[20]. Without a persistent supply of floral stimulus, the inner whorls of red-flowered *Impatiens* flowers revert to vegetative growth[15,21]. Although red-flowered *Impatiens* flowers revert when returned to non-inductive conditions, other lines of *Impatiens* do not revert (i.e. once flowers begin to form, the plant is irreversibly committed to produce complete flowers[12]).

Recent leaf-removal experiments on a non-reverting purple-flowered *Impatiens* indicate that the irreversible commitment to flowering is because of the continued production of floral stimulus by leaves that unfolded during the photoperiodic induction treatment (Fig. 3). In these experiments, when the leaves that unfolded during the photoperiodic induction treatment were removed, floral shoots reverted to vegetative growth. If the same leaves were not removed, the plants did not revert after they were placed in non-inductive conditions[12]. This indicates that commitment to flowering did not occur because the meristem became determined to produce flowers, but because the leaves became committed to continually produce floral stimulus. Additionally, the observation that commitment and reversion are often incomplete in *Impatiens* suggests that above-threshold levels of floral stimulus are required for the full specification of flower primordia[12].

This recent work on *Impatiens* fits well with the initial concept of photoperiodic induction put forward by V.N. Lubimenko and O.A. Sceglova[22]. They proposed that photoperiodic induction treatments cause stable changes that affect the whole plant, and not just the ephemeral production and movement of a 'florigen' that acts at the shoot apex. Of course, in the case of purple-flowered *Impatiens*, changes at the shoot apex are stable only if the communication with the leaves is maintained. The evidence for a leaf-based commitment to flowering is also consistent with classical grafting experiments in which leaves detached from photoperiodically induced plants were able to induce flowering when grafted onto non-induced plants growing in non-inductive photoperiods[7].

Floral induction, determination and specification in *Arabidopsis*

Determination experiments

The types of leaf-removal experiments described here have not been reported for *Arabidopsis*. Indeed, the growth habit and architecture of *Arabidopsis* would make such experiments difficult. However,

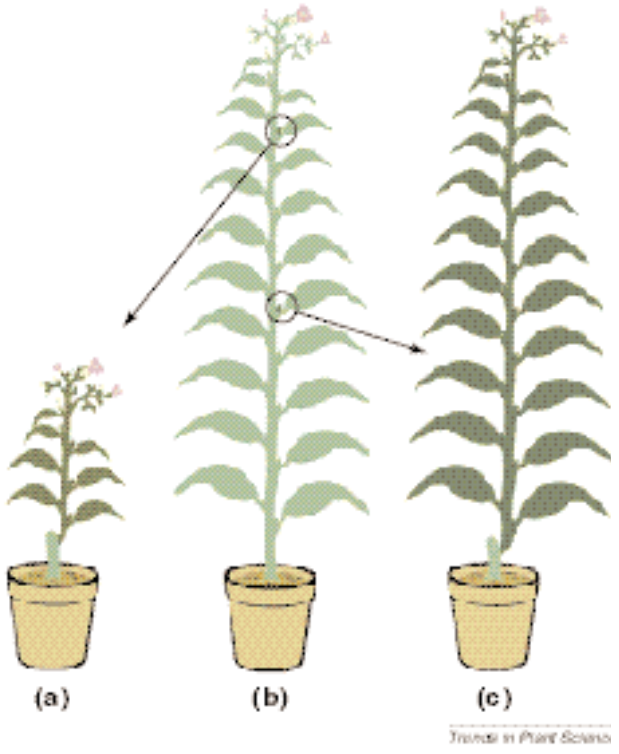

Fig. 2. Grafting assay for floral determination in *Nicotiana tabacum*. When grown under uniform environmental conditions, tobacco produces a certain number of nodes before forming a terminal flower. Axillary shoot apices below the inflorescence are relatively inactive, but will develop rapidly after rooting or grafting, or after decapitation of the plant. When excised from a donor plant (b) and grafted to the base of a second decapitated plant (a and c), apices either produce approximately the same number of nodes as a normal plant before forming a terminal flower (c), or produce significantly fewer nodes (a). The grafted shoot apex in (a) is judged to have been determined to flower before grafting, whereas the apex in (c) is judged to have been undetermined. Adapted from Ref. 16.

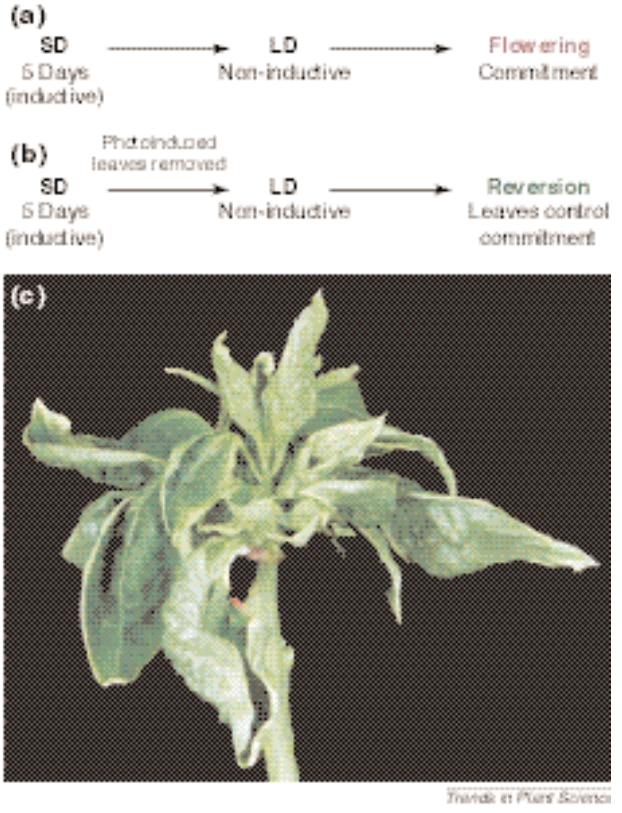

Fig. 3. Leaf removal experiments in the purple-flowered *Impatiens balsamina*. (a) Plants from a purple-flowered line of *Impatiens* are grown for five days in inductive conditions. When transferred to non-inductive conditions, the plants do not revert and continue to flower. Abbreviations: SD, short day (8 h light and 16 h dark); LD, long day (16 h light and 8 h dark). (b) Plants receive the same inductive treatment, but, at the time of transfer to non-inductive conditions, all unfolded leaves are removed. These plants revert and, after the production of several intermediate organs, leaf production resumes from the terminal flower. This data, coupled with the fact that red-flowered *Impatiens* plants revert to vegetative growth after transfer from inductive to noninductive conditions[8,20], indicates that meristem commitment does not occur in *Impatiens* and that primordium fate is controlled by the amount of floral stimulus coming from the leaves. (c) A reverted shoot that was formed under the experimental conditions described in (b). Note that the purple petal tissue is evidence of the initiation of a flowering before the reversion of this shoot. Photograph courtesy of Fiona Tooke and Nick Battey.

the results of simple photoperiodic determination assays have been reported[13]. It is evident from these studies that the early-flowering ecotypes of *Arabidopsis* can be irreversibly committed to flower within one day of the start of photoinduction, and that low red:far-red light ratios strongly promote the commitment to flowering[13,23]. This is not surprising, as high red:far red ratios, acting through phytochromes (particularly *PHY B*), are known to prolong vegetative growth in *Arabidopsis*[23,24]. Additional determination assays indicate that plants grown in long-day photoperiods are determined to flower after approximately seven days[14], when the first two leaves are approximately the same size as the cotyledons[25].

Photoinduction experiments also indicate that flowers can be formed from undetermined primordia that are present already at the shoot apex at the start of strong photoinduction treatments[26]. These experiments, and additional experiments in which a partial reversion of flowering occurred[27], suggest that the determination of primordium identity in *Arabidopsis* is not instantaneous. Thus, developing primordia evidently respond to floral induction signals over a period of time.

It is not known whether *Arabidopsis* meristems are florally determined, or if the leaves are committed to a perpetual production of floral stimulus. Although it might be tempting to assume that the *Arabidopsis* shoot meristem is determined to flower because *Arabidopsis* apical meristems cannot be reverted to vegetative growth, there is no evidence to indicate that this is the case. Furthermore, data on the commitment to flowering in *Arabidopsis*[13,14,23,28] is consistent with the hypothesis that the irreversible commitment to flowering is controlled outside the shoot meristem.

The mechanism(s) by which *Arabidopsis* primordia are committed to flower should, in part, be elucidated by the molecular and biochemical analyses of flowering-time genes and mutants over the next few years. Within the catalog of genes that correspond to early and late-flowering genes (reviewed in Refs 29,30) we should find genes involved in floral stimulus production, floral stimulus transport and floral stimulus perception within the shoot apex. It will be interesting to see if we also find genes for meristem determination, and/or genes for the irreversible commitment to floral stimulus production in the leaves.

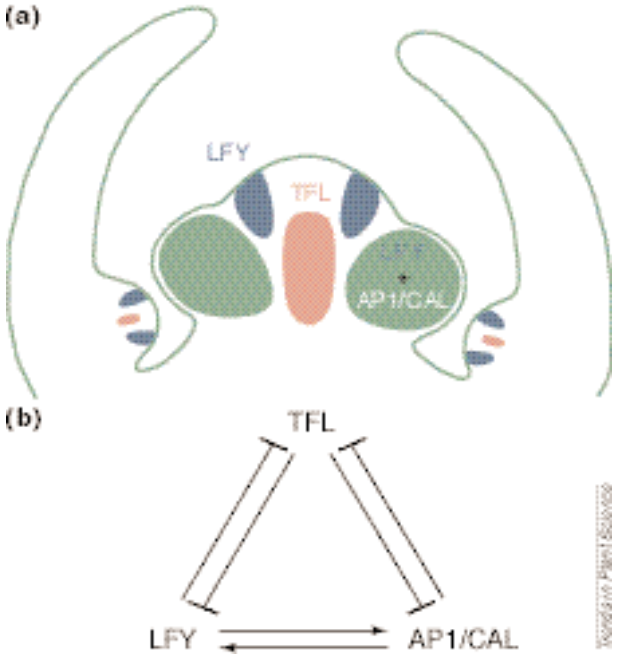

Fig. 4. Genes controlling flower primordium specification and inflorescence patterning in *Arabidopsis*. (a) The expression patterns of *LEAFY* (*LFY*), *APETALA1/CAULIFLOWER* (*AP1/CAL*) and *TERMINAL FLOWER 1* (*TFL1*) in the shoot apex. *LFY* and *AP1/CAL* are expressed in young flower primordia. Only relatively high levels of *LFY* are depicted here. Low levels of *LFY* expression occur in leaf primordia before, and after, flowering begins. High levels of *LFY* or *AP1/CAL* induce flower development. *LFY* expression precedes *AP1/CAL* expression. *TFL1* is expressed in the center of indeterminate meristems in *Arabidopsis*. (b) Interactions between *TFL1*, *LFY* and *AP1/CAL* during flower primordium specification. *TFL1* suppresses both the expression and the activity of *LFY* and *AP1*. Conversely, *LFY* and *AP1* suppress the expression of *TFL1*. *LFY* and *AP1* positively regulate each other. *LFY* promotes *AP1* transcription directly. Note: *AP1* and *CAL* are MADS-box genes with high similarity[31]. They are largely redundant, particularly with regard to their floral-meristem-identity function, as indicated by comparing the *ap1* mutant with the *ap1 cal* double mutant. In the *ap1* mutant, *CAL* expression is sufficient for floral meristem identity (although it is not sufficient for the supression of axillary flowers from the base of the sepals). However, in the *ap1 cal* double mutant, flowers are converted to indeterminate shoots indicating that either *CAL* or *AP1* (or both) is necessary for floral meristem identity.

Control of flower primordium specification by LEAFY and APETALA1/CAULIFLOWER

Although we know little about the mechanisms involved in the production of floral stimulus in the leaves of *Arabidopsis*, we have a much better idea of the mechanisms by which flowers are specified on the flanks of the shoot apex. For example, the replacement of flowers with indeterminate shoots in *lfy* and in *ap1 cal* double mutant indicates that *LFY* and *AP1/CAL* are critical for flower primordium specification[31,32]. The conversion of shoots to flowers in plants that ectopically express *LFY* and *AP1* (*35S::LFY* and *35S::AP1* plants) indicates that these genes are sufficient to specify flowers when expressed in shoot primordia[33,34]. Of course many other genes are also involved in the specification of flowers, along with *LFY* and *AP1/CAL*. But, because the genetics of floral meristem specification was reviewed only recently[35], we will limit our discussion primarily to the molecular interactions between *LFY* and the MADS-Box genes *AP1/CAL* and *AGAMOUS* (*AG*).

In wild-type plants, *LFY* is expressed throughout flower primordia early in their ontogeny[36,37]. *AP1* and *CAL* expression also occurs throughout flower primordia, although the expression of these two highly similar and largely redundant genes occurs in primordia only after they have become distinct from the meristem[38,39] (Fig. 4). Furthermore, the upregulation of *AP1* during floral induction treatments does not occur until many hours after *LFY* has been upregulated[13,19]. In *lfy* mutants, *AP1* expression is both weak and delayed[40], whereas the ectopic expression of *LFY* induces the ectopic expression of *AP1* in leaf primordia and in axillary flower primordia[36]. These results clearly indicate that *LFY* is a formal regulator of *AP1*.

New investigations into the regulation of *AP1* by *LFY* have shown that a steroid-inducible LFY::rat-glucocorticoid-receptor-binding-domain gene can transcriptionally activate *AP1* in the absence of protein synthesis. This indicates that the *AP1* promoter is a direct target of LFY (Ref. 41). The *AG* promoter is also evidently a direct target of LFY, as it has a LFY-responsive enhancer that is required for its activity[42]. Thus, *LFY* has direct and distinct roles in both the specification of flowers and in the patterning of floral organs[36,41,42]. Furthermore, the direct regulation of *AP1* by *LFY* and the temporal lag in *AP1* expression (Fig. 4) indicates that *LFY* is upstream of AP1 in the flower meristem specification process. However, recent analyses suggest that *AP1* also positively regulates *LFY*. For example, in *35S::AP1* plants, the upregulation of LFY occurs earlier, suggesting that *AP1* regulates *LFY* via a feedback loop, or through an interaction with another factor[40].

Interactions between LFY, AP1 and TERMINAL FLOWER1 (TFL1) during the transition to flowering

Although the initial specification of flowers is controlled largely by the floral-meristem-identity genes *LFY* and *AP1/CAL*, the patterning of the *Arabidopsis* inflorescence is regulated by interactions between these genes and *TFL1* (Fig. 4). *TFL1* prevents the expression of floral meristem identity genes in the shoot meristem and promotes indeterminate growth[14,43], although the mechanism by which it does this is unknown. Because TFL1 has similarity to animal proteins that associate with membrane protein complexes, it is unlikely that it directly regulates *LFY* and *AP1/CAL* transcription[14,44]. More likely, TFL1 affects the movement of signals or is the source of a signal that affects the expression of genes such as *LFY* and *AP1*. *TFL1* is also able to suppress the activity of *AP1* and *LFY* in the shoot primordia of plants that are expressing ectopically both *TFL1* and *LFY* or *AP1* (Ref. 44), which indicates that *TFL1* can also prevent shoot meristems from responding to *LFY* and *AP1*.

Conversely, *LFY* and *AP1* are both able to suppress the activity of *TFL1*. In *lfy* plants, ectopic *TFL1* expression is evident in the secondary meristems that normally would produce flowers[44]. In *35S::LFY* plants, there is no detectable TFL1 expression at any time during development[44]. Similarly, *TFL1* is ectopically expressed in the proliferating meristems of the *ap1 cal* double mutant[44], and *TFL1* expression is suppressed in *35S::AP1* plants[40,44]. Whether *LFY* and *AP1* suppress *TFL1* expression directly or indirectly is unknown.

Future prospects

In recent years, there has been a concerted effort to use model systems to unravel the secrets of floral induction, but many unanswered questions remain. With regard to floral commitment, we do not know whether it is generally controlled by the leaves or by the shoot meristem. However, it is now clear that in one plant, purple-flowered *Impatiens*, the commitment to flowering is controlled by the leaves[12], although the specification of flowers must also include additional levels of regulation within the flowering shoot apex. In *Arabidopsis*, we have uncovered much about the

initial specification of flowers, and many key genes involved in floral meristem identity have been identified. One of these genes, *LFY*, promises to be critically important for the further unraveling of floral induction mechanisms. *LFY* is of particular interest because the transition to flowering in *Arabidopsis* is modulated by levels of *LFY* activity in the meristem[28]. Thus, analyses of *LFY* promoter activation might well identify molecules that make up the floral stimulus[45]. The makeup of the floral stimulus remains one of the great mysteries of plant science. It potentially includes proteins or peptides, sugars, plant hormones and/or other small diffusible molecules[4]. Will these molecules, or their second messengers, interact with the emerging primordia (at, for example, the LFY promoter) or with the shoot meristem? Will we find molecular evidence for irreversible changes that lead to the perpetual production of floral stimulus within the leaves, and if so, within which leaves? Time will tell.

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References

1 Tournois, J. (1914) Etudes sur la sexualite du houblon. *Ann. Sci. Nat. Bot.* (*9th Series*) 19, 49–189

2 Garner, W.W. and Allard, H.A. (1920) Effect of the relative length of day and night and other factors of the environment on growth and reproduction in plants. *J. Agric. Res.* 18, 553–606

3 Knott, J.E. (1934) Effect of a localized photoperiod on spinach. *Proc. Am. Soc. Hortic. Sci.* 31, 152–154

4 Bernier, G. *et al*. (1993) Physiological signals that induce flowering. *Plant Cell* 5, 1147–1155

5 Chailakhyan, M.K. (1968) Internal factors of plant flowering. *Annu. Rev. Plant Physiol.* 19, 1–36

6 Evans, L.T. (1969) The nature of floral induction. In *The Induction of Flowering: Some Case Histories* (Evans, L.T., ed.), pp. 457–480, MacMillan

7 Lang, A. (1965) The physiology of flower initiation. In *Handbuch d. Pflanzenphysiologie (Encyclopedia of Plant Physiology)* (Vol. XV/1) (Ruhland, W., ed.), pp. 1379–1536, Springer-Verlag

8 Battey, N.H. and Lyndon, R.F. (1990) Reversion of flowering. *Bot. Rev.* 56, 162–189

9 Huala, E. and Sussex, I.M. (1993) Determination and cell interactions in reproductive meristems. *Plant Cell* 5, 1157–1165

10 McDaniel, C.N. *et al*. (1992) Developmental states associated with the floral transition. *Dev. Biol.* 153, 59–69

11 Harrison, R.G. (1933) Some difficulties of the determination problem. *Am. Nat.* 67, 306–321

12 Tooke, F. *et al*. (1998) Non-reversion of *Impatiens* in the absence of meristem commitment. *J. Exp. Bot.* 49, 1681–1688

13 Hempel, F.D. *et al*. (1997) Floral determination and expression of floral regulatory genes in *Arabidosis*. *Development* 124, 3845–3853

14 Bradley, D. *et al*. (1997) Inflorescence commitment and architecture in *Arabidopsis*. *Nature* 275, 80–83

15 Battey, N.H. and Lyndon, R.F. (1986) Apical growth and modification of the development of primordia during re-flowering of reverted plants of *Impatiens balsamina* L. *Ann. Bot.* 58, 333–341

16 Singer, S.R. and McDaniel, C.N. (1986) Floral determination in the terminal and axillary buds of *Nicotiana tabacum* L. *Dev. Biol.* 118, 587–592

17 Larkin, J.C. *et al*. (1990) Floral determination in the terminal bud of the short-day plant *Pharbitis nil*. *Dev. Biol.* 137, 434–443

18 Colasanti, J. *et al*. (1998) The *indeterminate* gene encodes a zinc finger protein and regulates a leaf-generated signal required for the transition to flowering in maize. *Cell* 93, 593–603

19 Simon, R. *et al*. (1996) Activation of floral meristem identity genes in *Arabidopsis*. *Nature* 384, 59–62

20 Pouteau, S. *et al*. (1997) The induction and maintenance of flowering in *Impatiens*. *Development* 124, 3343–3351

21 Krishnamoorthy, H.N. and Nanda, K.K. (1968) Floral bud reversion in *Impatiens balsamina* under non-inductive photoperiods. *Planta* 80, 43–51

22 Lubimenko, V.N. and Sceglova, O.A. (1931) Sur l'induction photoperiodique dans le processus du developpement des plantes. *Bull. Jardin Bot. Acad. Sci. URSS* 30, 1–52

23 Corbesier, L. *et al*. (1996) Design in *Arabidopsis thaliana* of a synchronous system of floral induction by one long day. *Plant J.* 9, 947–952

24 Chory, J. (1997) Light modulation of vegetative development. *Plant Cell* 9, 1225–1234

25 Hempel, F.D. (1996) The morphology of the transition to flowering in mustards. *Semin. Cell Dev. Biol.* 7, 391–400

26 Hempel, F.D. *et al*. (1998) Photoinduction of flower identity in vegetatively biased primordia. *Plant Cell* 10, 1663–1675

27 Okamuro, J.K. *et al*. (1996) Flowers into shoots: photo and hormonal control of a meristem identity switch in *Arabidopsis*, *Proc. Natl. Acad. Sci. U. S. A.* 93, 13831–13836

28 Blazquez, M.A. *et al*. (1997) *LEAFY* expression and flower initiation in *Arabidopsis*. *Development* 124, 3835–3844

29 Koornneef, M. *et al*. (1998) Genetic control of flowering time in *Arabidopsis*. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 49, 345–370

30 Levy, Y. and Dean, C. (1998) Control of flowering time, *Curr. Opin. Plant Biol.* 1, 49–54

31 Schultz, E.A. and Haughn, G.W. (1991) *LEAFY*, a homeotic gene that regulates inflorescence development in *Arabidopsis*. *Plant Cell* 3, 771–781

32 Kempin, S.A. *et al*. (1994) Molecular basis of the *cauliflower* phenotype in *Arabidopsis*. *Science* 267, 522–525

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

34 Mandel, M.A. and Yanofsky, M.F. (1995) A gene triggering flower formation in *Arabidopsis*. *Nature* 377, 522–524

35 Pidkowich, M.S. *et al*. (1999) The making of a flower: control of floral meristem identity in *Arabidopsis*. *Trends Plant Sci.* 4, 64–70

36 Parcy, F. *et al*. (1998) A genetic framework for floral patterning. *Nature* 395, 561–566

37 Weigel, D. *et al*. (1992) *LEAFY* controls floral meristem identity in *Arabidopsis*. *Cell* 69, 843–859

38 Mandel, M.A. and Yanofsky, M.A. (1995) The *Arabidopsis AGL8* MADS box gene is expressed in inflorescence meristems and is negatively regulated by *APETALA1*. *Plant Cell* 7, 1763–1771

39 Gustafson-Brown, C. *et al*. (1994) Regulation of the *Arabidopsis* floral homeotic gene *APETALA1*. *Cell* 76, 131–143

40 Liljegren, S.J. *et al*. (1999) Interactions among *APETALA1*, *LEAFY* and *TERMINAL FLOWER1* specify meristem fate. *Plant Cell* 11, 1007–1018

41 Wagner, D. *et al*. (1999) Transcriptional activation of APETALA1 by LEAFY. *Science* 285, 582–584

42 Busch, M.A. *et al*. (1999) Activation of a floral homeotic gene in *Arabidopsis*. *Science* 285, 585–587

43 Bowman, J.L. *et al*. (1993) Control of flower development in *Arabidopsis thaliana* by *APETALA1* and interacting genes. *Development* 119, 721–743

44 Ratcliffe, O.J. *et al*. (1999) Separation of shoot and floral identity in *Arabidopsis*. *Development* 126, 1109–1120

45 Blazquez, M.A. *et al*. (1998) Gibberellins promote flowering of *Arabidopsis* by activating the *LEAFY* promoter. *Plant Cell* 10, 791–800

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