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Florigen Coming of Age after 70 Years

The report that *FT* mRNA is the long-sought florigen, or at least part of it (Huang et al., 2005), has attracted much attention and was ranked the number three breakthrough of 2005 by the journal *Science* (Anonymous, 2005). This exciting discovery has brought to center stage one of the major outstanding questions in plant biology: What is the nature of florigen? In this essay, I summarize the classical experiments that led to the florigen hypothesis and how molecular-genetic approaches combined with physiological methods have advanced our understanding of florigen. I also discuss the possible universality of florigen and some of the remaining questions regarding flowering and other photoperiod-controlled phenomena involving long-distance signaling in plants.

FLORIGEN AS A PHYSIOLOGICAL CONCEPT

Julius Sachs (1865) may be considered the father of the flower hormone concept. From his well-known experiments with partially darkened *Tropaeolum majus* and *Ipomoea purpurea* plants, he concluded that leaves in the light produce flower-forming substances in very small amounts, which direct the assimilates to form flowers in darkened shoots. However, more convincing evidence in support of flower-forming substances did not appear until after the discovery of photoperiodism, the response of plants to the relative length of day and night (Garner and Allard, 1920). A seminal finding with photoperiodically sensitive plants was that daylength is perceived by the leaves, whereas flower formation takes place in the shoot apical meristem (Knott, 1934). This finding implies that a long-distance signal moves from an induced leaf to the shoot apex. Later, it was shown that this signal can also be transmitted from a flowering partner (donor) via a graft union to a non-flowering partner (receptor). Chailakhyan (1936) introduced the term ''florigen'' (flower-former) for this floral stimulus, which he defined as specific substances with a regulatory function. Grafting experiments between related species, but of a different photoperiodic response type (e.g., a short-day plant [SDP] and a long-day plant [LDP]), provided evidence for exchangeability of florigen among different response types. This earlier work showing that florigen is functionally conserved in different species has been extensively reviewed (Lang, 1965; Zeevaart, 1976). The *Crassulaceae* family has representatives of SDPs, LDPs, long-short-day plants (LSDPs; require long days [LDs] followed by short days [SDs] to flower), and short-long-day plants (SLDPs; require short days followed by long days to flower), which are all graft-compatible and can transmit the floral stimulus in every possible graft combination (see examples in Figure 1). Thus, the dogma emerged that florigen is universal in plants (at least in closely related species and different photoperiodic response types). However, despite numerous attempts to extract florigen and several reports of extracts with flower-inducing activity, which all turned out to be nonreproducible, florigen remained a physiological concept rather than a chemical entity. As a result, the florigen hypothesis fell into disrepute, and a rival hypothesis, proposing that flowering would be induced by a specific ratio of known hormones and metabolites, gained favor (Bernier, 1988; Bernier et al., 1993).

MOLECULAR-GENETIC STUDIES OF FLOWERING

As the physiological-biochemical approaches to flowering had begun to stagnate, along came molecular genetics with a new approach to the study of flowering. Isolation and characterization of mutants with respect to their flowering response, mainly in the facultative LDP *Arabidopsis thaliana*, became the mainstay of flowering research. Mutants flowering later than wild-type plants involve positive regulators of flowering, and early flowering mutants have lost repressors of flowering. Studies of epistatic relationships among the flowering genes have resulted in a network of four response pathways that control flowering in *Arabidopsis*: the photoperiod, vernalization, autonomous, and gibberellin (GA) flowering response pathways (Mouradov et al., 2002; Périlleux and Bernier, 2002; Komeda, 2004; Corbesier and Coupland, 2005). In most of this genetic work, the role of florigen in flowering was ignored until recently, presumably because it was not obvious that any of the identified flowering genes was involved in production of or response to florigen. In this context, I will restrict the discussion mainly to the photoperiod pathway. Two genes central to LD-induced flowering in *Arabidopsis* are *CONSTANS* (*CO*) and *FLOWERING LOCUS T* (*FT*). *CO* encodes a nuclear zinc-finger protein, which in response to LD induces transcription of *FT*, encoding a RAF-kinase-inhibitor-like protein. Neither of these genes is expressed to any extent in the shoot apex. Expression from meristem-specific promoters of *CO* does not promote flowering, but early flowering is induced in plants in SD when *FT* is overexpressed in the meristem. Expression of *CO* only in the phloem is sufficient to generate a phloem-mobile signal, as shown by grafting experiments with *Arabidopsis* (An et al., 2004; Ayre and Turgeon, 2004). An et al. (2004) speculated that FT protein might be the mobile signal or, alternatively, that FT controls the synthesis of a mobile, small substance that induces flowering.

In the vernalization pathway, flowering is promoted in response to a prolonged exposure to low temperature (vernalization). In cold-requiring accessions of *Arabidopsis*, the MADS

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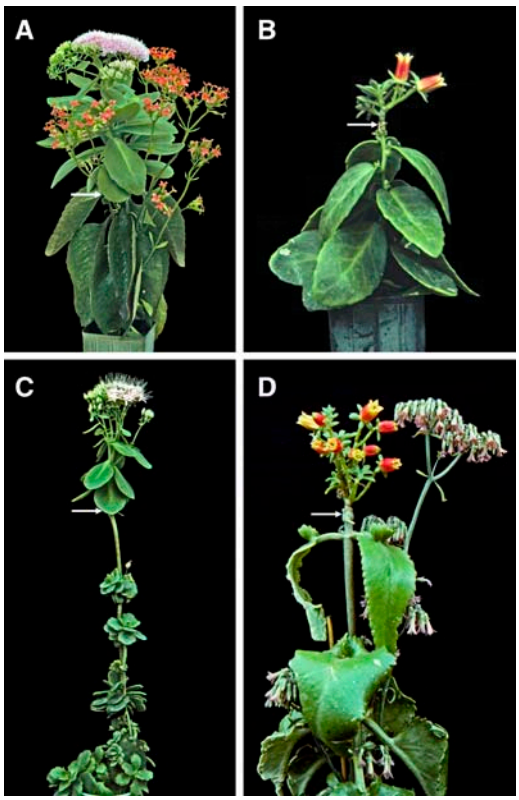

Figure 1. Four Examples from the Crassulaceae in Which Flowering Is Induced in a Noninduced Scion by Transmission of Florigen from a Florally Induced Stock.

In each case, the stock (below the graft union) is the donor, and the scion (above the graft union) is the receptor. Arrows point toward the graft unions. In each case, the appropriate photoperiodic conditions were used to induce flowering of the donor plant, whereas the receptor was in a noninductive photoperiod. None of the control grafts with noninduced donors caused flowering in the receptors (data not shown).

(A) The SDP *Kalanchoë blossfeldiana* as donor for the LDP *Sedum spectabile* as receptor (Zeevaart, 1958).

(B) The LDP *S. spectabile* as donor for the SLDP *Echeveria harmsii* (my unpublished data).

(C) The LSDP *Bryophyllum crenatum* as donor for the LDP *S. spectabile* (my unpublished data).

(D) The LSDP *B. daigremontianum* as donor for the SLDP *E. harmsii* (Zeevaart, 1982).

box gene *FLOWERING LOCUS C* (*FLC*) is highly expressed, and *FT* in the leaf is repressed by the FLC protein. Vernalization reduces *FLC* expression, thus inducing *FT*, which can now act as a stimulus for flowering. In addition, in the shoot apex, *FLC* expression inhibits response to the FT signal. Thus, vernalization in *Arabidopsis* acts to allow (1) production of the FT signal in leaves and (2) response to the signal in the apical meristem (Searle et al., 2006).

PHYSIOLOGY AND GENETICS CONVERGE

It was expected that the physiological-biochemical and molecular-genetic approaches would ultimately come together and give rise to a unifying theory of flowering. The finding that *CO* via activation of *FT* regulates the synthesis of a mobile, flower-inducing stimulus was strong support for the florigen hypothesis (see above). Huang et al. (2005) investigated the possibility that the mobile stimulus is *FT* mRNA (or at least part of it). These workers conducted a set of elegant experiments using induction of a single *Arabidopsis* leaf combined with sensitive molecular techniques and microdissection of shoot apices to show that *FT* under the control of a heat shock promoter was transiently induced in the heated leaf and that *FT* mRNA was detected in the shoot apex 6 h later. The conclusion from these results is that *FT* mRNA is the limiting factor for flowering; it is produced in the leaf and moves to the apical meristem, where its arrival is correlated with flower formation. Thus, *FT* mRNA fulfills the definition of florigen (at least in *Arabidopsis*). The objection can be raised that FT itself is not the final stimulus, but only induces another factor essential for flowering that moves along with *FT* transcripts from leaf to shoot apex (An et al., 2004; Huang et al., 2005; Wigge et al., 2005). However, it is unlikely that FT plays such a role in the leaf phloem. FT acts in the shoot apex by forming a complex with the basic domain/leucine zipper protein FLOWERING LOCUS D (FD). This FT/FD heterodimer then activates the downstream floral meristem identity gene *APETALA1* (*AP1*) (Abe et al., 2005; Wigge et al., 2005). Moreover, expression of *FT* from a meristem-specific promoter will induce early flowering in SD, indicating that in such transgenic plants no signal from the leaf is required for early flowering. This is strong evidence that *FT* mRNA is the only essential factor for floral initiation that moves from leaf to shoot apex. It is, of course, possible that FT protein also moves from the induced leaf to the shoot apex. In fact, FT protein has recently been identified in phloem exudate from inflorescence stems of *Brassica napus* (Giavalisco et al., 2006). If both mRNA and protein move from an induced leaf to the shoot apex, the question is: Which one is necessary for flowering, or are perhaps both required?

In some species, production of florigen appears to continue after the plants are no longer exposed to the inductive photoperiod. This phenomenon is illustrated by induced leaves of the SDP red *Perilla*, which were still effective donors in grafting experiments 3 months after they had been moved from SD to LD (Zeevaart, 1958). There are also species (e.g., *Xanthium strumarium* and *Bryophyllum daigremontianum*) in which flowering receptor shoots become effective donors themselves. This phenomenon, called indirect induction or non-localized induction, suggests that florigen has self-perpetuating properties (for review, see Zeevaart, 1976). The results by Huang et al. (2005) provide further insight into these phenomena. These workers reported that a few hours after the heat shock–inducible *FT* transgene was induced, native *FT* mRNA also started to accumulate both in the induced leaf and in the shoot apex. This

finding suggests that there is positive feedback whereby *FT*, once induced, further enhances its own expression, both in the donor leaf and in the apical meristem.

MOVEMENT OF FLORIGEN/*FT* mRNA

Florigen moves in the phloem along with photoassimilates (e.g., King and Zeevaart, 1973). The velocity calculated for the movement of *FT* mRNA in *Arabidopsis* was 1.2 to 3.5 mm/h (Huang et al., 2005), which is in the same range as measured for export of florigen from cotyledons of the SDP *Pharbitis nil* induced by a single dark period (Imamura and Takimoto, 1955; Zeevaart, 1962a). This rate is much slower than the movement of sugars in the phloem (50 to 100 cm/h). However, with adult plants of *P. nil* and much longer distances between donor leaves and receptor buds than in seedlings, velocities of florigen movement were much closer to the values for assimilate movement (Takeba and Takimoto, 1966; King et al., 1968).

A priori, it would be expected that mRNA molecules, probably forming a complex with a protein, would move more slowly than assimilates. The earlier values for velocities of florigen were based on the time it took for florigen to move out of an induced leaf and initiate a flowering response. This approach would obviously underestimate the velocity because it is based on flowering response, which presumably requires a threshold value of florigen and does not measure the first molecules arriving at the shoot apex. It is surprising, therefore, that with the direct measurement of *FT* mRNA arriving at the apex (Huang et al., 2005) no higher velocities were found than with the physiological approach.

Movement of RNAs and proteins in the phloem is now well established (Lucas et al., 2001). *FT* mRNA is produced in the companion cells and then has to move through the sieve elements to the shoot apex to induce flowering. From the termination of the protophloem strands in the shoot apical meristem, it then has to traverse, presumably symplastically, a series of meristematic cells to reach its target, the shoot apex. However, movement of *FT* mRNA all the way from source leaf to the shoot apex proper may not be necessary. As discussed above, *FT* mRNA, once produced, induces production of more *FT* mRNA via an autoregulatory feedback loop (Huang et al., 2005). So, it is conceivable that *FT* mRNA that exits from the protophloem induces expression of *FT* throughout the apex, thus making it superfluous for RNA molecules to move from the protophloem ends across many cells to the apex.

IS THE CO→FT SIGNALING PATHWAY UNIVERSAL FOR CONTROLLING FLOWERING?

The tenet of the florigen hypothesis is that florigen is the same in SDPs, LDPs, day-neutral plants (DNPs), LSDPs, and SLDPs. Grafting experiments can be performed only between closely related species, but results of interspecific and intergeneric grafts between different photoperiodic response types support this idea (see above). Thus, regardless of which environmental cues are required for floral induction, the end product, florigen, is the same and, by implication, regulation of *CO* and *FT* expression is central to flowering in all plant species. Indeed, the CO→FT combination in the flowering response pathway appears to be highly conserved, regardless of response type. For example, in SDP rice (*Oryza sativa*), the ortholog of *FT*, *Hd3a*, promotes flowering downstream of *Hd1*, the ortholog of *CO* (Kojima et al., 2002). Increased expression of *Hd3a* occurs in darkness; suppression by night interruption inhibits flower initiation (Ishikawa et al., 2005). Thus, the photoperiod pathway for flowering is conserved between SDP rice and LDP *Arabidopsis* and most likely in other species as well (Hayama and Coupland, 2004). Therefore, the differences between SDPs and LDPs appear to reside in how the genes in the flowering pathways function and are regulated. It remains to be shown, of course, that FT is the universal systemic transmissible signal (mRNA or protein) that is required for flowering.

Little work on flowering has been performed with DNPs because their flowering cannot be controlled at will. However, recent work with tomato (*Solanum lycopersicum*) demonstrates that flowering in this DNP is also induced by a transmissible signal, generated by the ortholog of *FT*, *SINGLE-FLOWER TRUSS* (*SFT*) (Lifschitz et al., 2006). Overexpression of *FT* or *SFT* in day-neutral tobacco (*Nicotiana tabacum*) or tomato induced early flowering in both species. Moreover, overexpression of *SFT* induced flowering in the SDP Maryland Mammoth tobacco in LD and in *Arabidopsis* under SD. Transmission of florigen via grafts was obtained from tomato overexpressing *SFT* (donor) to *sft* mutant plants, to Maryland Mammoth tobacco in LD, and to a tomato mutant *uf* that does not flower under low irradiance. *SFT* was expressed in the leaves, and its protein was mainly localized in the nuclei of leaf cells. No evidence was obtained for movement of *SFT* mRNA from donor leaves to receptor shoots, so that it was proposed that in tomato, florigen is a signal downstream of *SFT* (Lifschitz et al., 2006). Removal of *SFT* donor shoots promptly reverted *sft* receptors to mutant phenotype, indicating that *SFT* mRNA is very short-lived in the receptors (if it crosses the graft union at all) and also that, unlike in *Arabidopsis* (see above), an *SFT* autoregulatory loop does not function in tomato. So, although there may be differences between different species and photoperiodic response types, all have in common that either *FT*, or a product of *FT*, is the flower-inducing signal.

There are many examples of successful transmission of florigen between different species (see above), but there are also many examples in which the receptor shoots did not flower (Zeevaart, 1976). Does this mean that in the latter case florigen is not functionally conserved? The work with tomato provides an answer to this question. Transgenic plants overexpressing *SFT* under control of the 35S promoter were strong donors, but wild-type tomato could not complement *sft* mutant plants in grafting

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experiments (Lifschitz et al., 2006). This result makes it clear that the level of florigen in wild-type plants is too low to induce flowering in the receptor plants. Thus, the failure to induce flowering in receptor shoots is not due to nonidentity of florigen but due to insufficient production of florigen in the donor and/or rapid decay of florigen in the receptor.

Unlike herbaceous plants, trees flower only after a long juvenile phase that may last many years. A recent report shows that expression of *FT* is also a prerequisite for flowering in trees. Ectopic expression of an *FT* ortholog in aspen (*Populus* spp) resulted in early flowering and thus drastically shortened the juvenile phase. Moreover, expression of the *FT* ortholog increased with age of the trees (Böhlenius et al., 2006). Work by Hsu et al. (2006), reported in this issue of *The Plant Cell*, also shows that in juvenile *Populus deltoides* a critical level of *FT2* expression is necessary before flowering will occur. In addition, LD-induced transcription of *FT2* in spring is closely associated with floral initiation in mature trees. These results with trees provide further evidence that the CO→FT system for control of flowering time is widespread and not restricted to herbaceous plants.

GAs AND FLOWERING

GA can induce or promote flowering in many LDPs that grow as a rosette in SD. However, not all rosette plants can be induced to flower by GA, although applied GA always causes stem elongation. By contrast, GA does not induce flowering in SDPs grown in noninductive LD conditions. Because results of grafting experiments indicate that florigen is exchangeable between LDPs and SDPs, it was concluded early on in work on the role of GA in flowering that GA cannot be florigen (see Zeevaart, 1983). In the LDP *Lolium temulentum*, GA causes floral initiation without first causing stem elongation, and GAs, especially GA_5 and GA_6, are endogenous signals transmitted from an induced leaf to the shoot apex. These GAs have been assigned a role as florigen in grasses (King and Evans, 2003), but this role appears to be restricted to a certain group of plants, temperate grasses, just as the flower-inducing effect of ethylene is limited to the family of the *Bromeliaceae* (see Zeevaart, 1976, 1978). Florigen was meant to indicate a universal flower hormone. At present, FT-regulated flowering appears to be widespread, and it would be preferable, therefore, to restrict the term florigen to the *FT*-induced transmissible signal(s).

The effect of GA on flowering raises the question about the relationship between GA and *FT* expression. In *Arabidopsis*, GA activates the floral meristem identity gene *LEAFY* (*LFY*) (Blázquez et al., 1998) but does not regulate expression of *FT* (Moon et al., 2003). In support of separate GA and *FT* flowering pathways, King et al. (2006) also found that an increase in *FT* mRNA in *L. temulentum* in LD occurred independently of GA. LFY is conserved in plants (Maizel et al., 2005), so that with respect to the GA response pathway the question is: What is the effect of GA on expression of *LFY* in LDPs and SDPs that do not flower in response to applied GA?

A TRANSMISSIBLE FLOWER-INHIBITING SIGNAL OF FLOWERING

In addition to flower-promoting florigen, there is also evidence that noninduced leaves can inhibit flowering. Some of these inhibiting effects can be explained in terms of source-sink relationships between induced leaves and receptor buds. Noninduced leaves between donor leaves and receptor buds can prevent florigen from reaching the target receptor buds, as demonstrated by correlating transmission of florigen with ^{14}C-photoassimilate translocation in *Perilla* (King and Zeevaart, 1973). One may call this phenomenon nonspecific inhibition due to interference with florigen movement. However, there is also evidence for specific inhibition of flowering by a mobile signal. In grafting experiments with various tobaccos, both the flowering SDP Maryland Mammoth and LDP *Nicotiana sylvestris* promoted early flowering in day-neutral tobacco. But when the donors were kept in noninductive daylengths, Maryland Mammoth had only a slight flower-delaying effect in the day-neutral tobacco, whereas *N. sylvestris* suppressed its flower formation. These responses indicate that the LDP *N. sylvestris* in SD produces a transmissible flower-inhibiting signal that is absent (or present at a much lower level) in the SDP Maryland Mammoth tobacco (Lang et al., 1977).

Can this physiological evidence for a flower inhibitor now be interpreted in molecular-genetic terms? Loss-of-function mutants that flower earlier than wild-type plants have lost a repressor of flowering. One such mutant in *Arabidopsis* is *tfl1*, which flowers very early with a terminal flower. Interestingly, TFL1 has homology with FT, and change of a single amino acid can convert TFL1 as a repressor of flowering to an activator of flowering (Hanzawa et al., 2005). This raises the question: Does *TFL1* mRNA, like *FT* mRNA, also move in the phloem as a signal counteracting FT? Although TFL1 may be moving in the phloem, it is probably not a flower-regulatory signal because *TFL1* is already highly expressed in the shoot apical meristem, where it interacts antagonistically with the floral meristem identity genes *LFY* and *AP1/AP2* (Shannon and Meeks-Wagner, 1993; Ratcliffe et al., 1999). Thus, at present, there is no known gene function that is specifically associated with a transmissible flower inhibitor.

OTHER TRANSMISSIBLE PHOTOPERIODIC SIGNALS

There are other phenomena in plants besides flowering that are under photoperiodic control and involve long-distance signaling. Tuberization in potato (*Solanum tuberosum*) is induced by SD. Gregory (1956) showed transmission of a tuber-inducing stimulus from an induced to a noninduced shoot. When *Nicotiana* spp of different photoperiodic response types were grafted on

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tuberless *Solanum andigenum*, the SDP Maryland Mammoth tobacco induced tubers in SD only, the LDP *N. sylvestris* in LD only, and the DNP Trapezond tobacco in both SD and LD (Chailakhyan et al., 1981). It is clear from these results that only flowering donors could induce tuber formation in *S. andigenum*, raising the possibility that florigen and the tuber-forming stimulus are interchangeable. Thus, it is not too far-fetched to propose that tuber formation is also under control of the CO→FT pathway. In more recent work, overexpression of *Arabidopsis CO* in potato inhibited tuber formation, and this inhibitory effect was perceived in the leaves of transgenic plants (Martínez-García et al., 2002). Results with overexpression of *FT* should further clarify the possible role of the CO→FT signaling pathway in tuber formation.

Several phenomena in woody species, such as cessation of apical growth, bud dormancy, cambial activity, cold acclimation, and leaf fall in deciduous species, occur in the fall under shortening photoperiods. In *Betula pendula*, a northern ecotype had a longer critical photoperiod and greater photoperiodic sensitivity for growth cessation than a southern ecotype, resulting in earlier dormancy and cold acclimation (Li et al., 2003). As demonstrated with actively growing seedlings of certain woody species, the locus of perception for dormancy is the leaves, whereas the buds respond with dormancy, a situation reminiscent of photoperiodic induction of flowering (see Wareing, 1957). Therefore, it is not surprising that *CO* is the mediator between the shortening daylength and low expression of the ortholog of *FT* in aspen trees, resulting in growth cessation and bud dormancy (Böhlenius et al., 2006). This shows that the CO→FT combination not only plays a critical role in flowering but can mediate vegetative growth as well.

PERSPECTIVE

Discoveries usually give rise to many new questions. This is also the case with the finding that FT plays a pivotal role in inducing flowering. It is important to determine whether FT itself (RNA or protein) (Huang et al., 2005) or its product (Lifschitz et al., 2006) is the mobile flower-inducing signal. This question needs to be resolved, probably using plants overexpressing *FT* because in wild-type plants, expression of *FT* may be too low for easy detection.

Physiological experiments indicate that production and persistence of florigen vary among species. For example, different varieties of the SDPs *P. nil* and *X. strumarium* differ in the number of inductive cycles required for flowering. These differences are based on differences in production of florigen as well as on differences in sensitivities of the shoot apex to florigen (see Zeevaart, 1976). Can these differences now be explained in terms of FT expression, FT transport, or response of the shoot apex to FT?

Like trees (see above), herbaceous plants also have a juvenile phase, although usually of short duration. In grafting experiments, it could be shown that in red *Perilla* and in *B. daigremontianum*, juvenility is due to inability of juvenile leaves to produce sufficient florigen, whereas apical meristems of juvenile plants can respond to florigen with flowering (Zeevaart, 1958, 1962b). Therefore, juvenility in these herbaceous plants resides in the leaves. Thus, it would be expected that there is an acropetal gradient of increasing expression of *FT* in induced leaves in these plants. As indicated by these few examples, it is anticipated that many of the classical observations on physiology of flowering can now be studied from a molecular-genetic perspective and will ultimately lead to a general theory of flowering with FT perhaps as the common signal.

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