The flowering hormone florigen functions as a general systemic regulator of growth and termination

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The florigen paradigm implies a universal flowering-inducing hormone that is common to all flowering plants. Recent work identified FT orthologues as originators of florigen and their polypeptides as the likely systemic agent. However, the developmental processes targeted by florigen remained unknown. Here we identify local balances between SINGLE FLOWER TRUSS (SFT), the tomato precursor of florigen, and SELF-PRUNING (SP), a potent SFT-dependent SFT inhibitor as prime targets of mobile florigen. The graft-transmissible impacts of florigen on organ-specific traits in perennial tomato show that in addition to import by shoot apical meristems, florigen is imported by organs in which SFT is already expressed. By modulating local SFT/SP balances, florigen confers differential flowering responses of primary and secondary apical meristems, regulates the reiterative growth and termination cycles typical of perennial plants, accelerates leaf maturation, and influences the complexity of compound leaves, the growth of stems and the formation of abscission zones. Florigen is thus established as a plant protein functioning as a general growth hormone. Developmental interactions and a phylogenetic analysis suggest that the SFT/SP regulatory hierarchy is a recent evolutionary innovation unique to flowering plants.

T he florigen paradigm was conceived from the study of photoperiod-sensitive plants but implies, in its general form, a universal graft-transmissible flowering signal that although activated in leaves by species-specific stimuli is common to all plants (1–3). Unequivocal evidence for the critical tenets of graft transmissibility and universality of the systemic mechanism was obtained in tomato. SFT, the FT homologue encoding florigen (4, 5), triggers graft-transmissible signals that complement late flowering in sft plants and substitutes for light dose stimuli in day-neutral tomato and tobacco, for short days in Maryland Mammoth tobacco and for long days in Arabidopsis (6). On the basis of the absence of SFT mRNA beyond the graft joints, we suggested that florigenic signals are generated by cell-autonomous SFT transcripts. This implicated the protein as a likely systemic agent (7), which was supported by strong circumstantial evidence (8–14). However, the developmental mechanisms targeted by florigen to transform vegetative meristems into reproductive organs remain unknown, and their study, by and large, is indifferent to florigen being a protein or RNA. A clue as to the target of florigen was inferred from the observation that overexpression of SFT induces, in addition to precocious flowering, an overall growth retardation (6). This seemingly trivial phenomenon associated with flowering in many plants might be the consequence of stress upon flowering, but because growth retardation and precocious flowering were triggered by a single gene, we hypothesized that they represent 2 facets of the same mechanism. In other words, boosting flowering is just 1 of the pleiotropic functions of florigen (6). To identify the developmental targets of florigen system-wide, we dissected its overall growth effects by using grafting in conjunction with mutants that sensitize organ-specific responses to florigen.

The tomato plant presents unique opportunities to study multiple aspects of florigen. Its shoots consist of developmental modules with homology to monopodial annuals but also feature regular vegetative/reproductive oscillations typical of woody sympodial perennials. Unlike other systems, tomato is photoperiod insensitive, thereby eliminating the influence of day length on the functional analysis of florigen. Finally, the ease of grafting and a fortunate battery of gene mutations can be used to monitor the effects of florigen on diverse aspects of growth. Pivotal to the system are SFT, encoding florigen (6), and SP, a homologue of TFL1 that promotes growth and represses flowering (15–19). Being members of the same gene family, these 2 CETS (CEN, TFL, SP) genes encode signaling factors with multiple options for protein–protein interactions (20). For example, the interaction with FD proved essential for the floral-inducing function of FT (21, 22). The interaction of the 2 genes with the same proteins (20) and the contrasting flowering modes of primary and sympodial apices of the self-pruning plants indicated to us that SP is a major component of the flowering response mechanism (7). Here we analyze the broad developmental consequences of changes in the SFT/SP balances as modified, in a context-dependent manner, by the mobile, graft-transmissible form of SFT, florigen. Florigen is thus established as a plant protein shown to function as a general growth hormone.

Results

Context-Specific Termination of Vegetative Growth in Shoot Apical Meristems by Florigen. WT tomato plants terminate with a primary inflorescence after forming 8 to 12 leaves, and subsequent sympodial units (SUs) consist of 3 leaves and a terminal inflorescence (Fig. 1A). Isogenic sp plants also terminate after 8 to 12 leaves, but subsequent SUs form progressively fewer leaves until the shoot is terminated by 2 consecutive inflorescences (SI Text, Fig. S1, and Table S1). It was inferred therefore that the flowering programs for the primary and sympodial shoots in tomato might be different (7).

Here we describe the role of SFT and its mobile form, florigen, in the 2 flowering programs. We show that both local SFT and florigen impact differential termination and flowering in the primary and sympodial apices. Unlike in sp, termination of the primary shoot in sft is delayed by 5 to 6 leaves resulting in a terminating vegetative inflorescence shoot that arrests the sympodial branching, thereby replacing the normal sympodial shoot system (SI Text and Fig. S1). Surprisingly, despite the opposite effects of sp and sft on WT shoot architecture, sft single mutants and sft sp double mutants are indistinguishable (23). Furthermore, overexpression of SP delays primary termination, increases the number of leaves per SU, and promotes leafy

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The differential response of primary and sympodial apices to SFT (Fig. 1 and Fig. S2) contrasted with their opposite sensitivity to the inactivation of SP. Whereas florigen, or a 200-fold excess of SFT (Fig. S3), failed to disrupt the regularity of sympodial cycles, this regularity readily collapses in sp plants. To analyze the contrasting response of primary and sympodial apices to overexpression of florigen and to sp inactivation, we bred sp plants overexpressing SFT. Primary termination in sp S5:SFT plants also occurred after 3 to 4 leaves, but the terminating inflorescence meristem produced no or at most 2 flowers, and sympodial branching was completely suppressed. Although distal auxiliary shoots were readily released from apical dominance in these plants, they produced only 1 or 2 leaves before terminating with a single flower or a blind apex (Fig. 1D). SFT is therefore a potent terminator of primary, sympodial, and inflorescence apices, but its role in sympodial and flower meristems is checked primarily by SP. Although 1 dose of SP in sp/+ heterozygotes is sufficient to maintain the 3-leaf cycles in WT plants, 2 doses are required to maintain it under excess of SFT, as suggested by the fluctuation in leaf numbers (between 1 and 3) per SU’s in sp/+S5:SFT plants (Table S1). Significantly, all features of sp S5:SFT were also induced in sp receptors by florigenic SFT signals emanating from a grafted 35S:SFT donor (Fig. 1E).

To examine the effect of other flowering genes on the differential response of the primary and sympodial apices to SFT and florigen, we bred additional flowering mutant lines that also expressed the S5:SFT transgene. falsiflora (fals, the tomato LFY), macrocalyx (mc, an AP1-like), blind (bl), and other mutant lines (Table S2) expressing the 35S:SFT transgene terminated, like S5:SFT, after 3 to 4 leaves (Fig. 1F and Fig. S3). However, the phenotypes of their mutant inflorescences and their proper sympodial patterns were maintained. The role of SP in checking SFT is therefore unique, such that SFT confers termination but not identity, and this function does not require the activity of FALS or MC.

To facilitate the analysis of perception and transmission of florigen by the different genotypes, uniflora (uf), a late-flowering, light-sensitive tomato mutant used previously to examine the universal functions of SFT (6), was combined with uf to generate a tester line that does not flower under any growth condition unless grafted with a florigen donor (Fig. 1G and H, SI Text, and Table S3). Grafting experiments showed that all mutant lines expressing 35S:SFT as graft donors induced efficient flowering in uf receptors and that as receptors, all these mutants responded to mobile florigen (Fig. S4 and Table S3). Thus, the sensitivities of the mutant lines to endogenous SFT and a mobile florigen are comparable. Together these results indicate that the tested genes are not required for the mobility or perception of florigen and reveal that the differential response of primary and sympodial meristems to florigen is maintained under a wide range of genetic backgrounds. Evidence pertinent to the 2 flowering programs in Arabidopsis and other plants is discussed in Fig. S2.
Florigen Regulates Stem and Leaf Meristems in an SP-Dependent Manner. Because of their developmental versatility, the compound leaves of tomato provide a highly sensitized forum to illustrate the effects of florigen as a general growth hormone (24) (Fig. 2B). SP and sp leaves of isogenic lines were indistinguishable except for a slightly reduced serration in the sp leaf margins. However, sft leaves displayed a distinct morphology and spacing of leaflets and carried additional folioles, suggesting a role for SFT in regulating midrib meristems (Fig. 2B). Leaves of 35S:SFT plants, although maintaining a compound architecture, usually lacked 1 pair of leaflets and were almost devoid of folioles. Strikingly, leaves of sp 35S:SFT plants were reduced to only 1 pair of lateral leaflets, and some were simple with entire margins (Fig. 2B). The arrest of lateral leaflet meristems suggests an early function of SP during leaf primary morphogenesis. In contrast, overexpression of SP induced larger blades with undulating surfaces, indicative of unregulated growth (Fig. 2B), an effect that was not displayed by sft 35S:SP plants (Fig. S2). As shown in Fig. 2C, all of these features were also induced by mobile SFT, suggesting that florigen is imported by leaves, where SFT is already expressed (see below), and arrest leaflet formation in an SP-dependent manner. Thus, in sft and sp 35S:SFT (i.e., under low or high SFT/SP ratios, respectively), SFT, SP, and florigenic SFT function as leaf meristem factors.

The response of stem meristems to different doses of SFT and SP is demonstrated in Fig. 2A. Although normal sympodial cycling in 35S:SFT plants resumed after primary termination, normal radial expansion of sympodial internodes was permanently suppressed, indicating a context-specific function of florigen. In general, a high SFT/SP ratio promoted growth restriction at the shoot apical meristems (SAMs) and lateral leaflet meristems, leading to a faster floral transition and reduced leaf complexity respectively. But higher SFT/SP ratio as in sp 35S:SFT plants resulted in the complete suppression of both vegetative and inflorescence meristems (Fig. 1D).

Florigen Mediates Its Own Distribution by Regulating Sink–Source Relations. The reiterated phase transitions along the tomato shoot and its evergreen nature and day length insensitivity require that the distribution of florigen be regulated by a dynamic balance on a daily basis. To characterize the balance between the dynamic needs for endogenous local SFT and its mobile form in the shoot system, we determined the minimal source required to induce a flowering response in the tomato bush. A single mature 35S:SFT donor leaf induced flowering in sft uf receptor shoots for 2 months and in apices 2 m above the graft points (Fig. 2D). Therefore, every mature leaf is capable of exporting florigen to all parts of the tomato bush.

The developmental expression of SFT and SP at the whole-plant level was studied by comparing each gene’s age-dependent expression gradients within sequentially growing leaves along primary shoots. Initially a series of leaves was collected from WT and WFTNFT seedlings, having 10 leaves larger than 1 cm and a primordial inflorescence. Leaves of all ages were collected 4 h after dawn, corresponding to the diurnal SFT peak (Fig. S3). As shown in Fig. 2E, SFT and SP display opposing age-dependent expression gradients in which SFT RNA was relatively high in expanded mature leaves and SP RNA relatively high in the immature leaves. For comparison, profiles of other relevant genes were also included (Fig. 2E). Note that the age-dependent gradients of SFT and SP are independent of plant age, and similar series of leaves taken from postflowering plants will display the same gradients.

As shown in Fig. 2F, the expression patterns of SP were not altered in sft plants and vice versa, suggesting that these patterns are not interdependent. We next explored the possibility that overexpression of one of the genes will affect the endogenous expression of the other (Fig. 2G). Here, the expression of 35S:SFT or 35S:SP results in high (200-fold and more) but equal expression in all leaves; the endogenously differential expression of SP and SFT, respectively, in old and young leaves was maintained. We therefore inferred that the functional antagonism between SFT and SP does not primarily involve mutual transcriptional feedback loops.

Age-dependent gradients were also evident in leaflets along the proximal–distal axis of immature leaves up to 15 cm long. But when leaves reached approximately three quarters of their final size (approximately 25 cm), all their major leaflets expressed SFT equally (Fig. 2H).

Removal of mature leaves delays flowering in tomato (7), and we speculated that by the time intraleaf expression gradients have
leveled off (Fig. 2F), leaves become better exporters of florigen. Because SP promotes growth, its inactivation, or an elevated SFT/SP ratio brought about by imported florigen, are likely to accelerate leaf maturation, converting leaf status from sink to source and leading to early release of florigen. This conjecture is supported by experiments comparing 3SS:SFT and sp 3SS:SFT donors. Donors of both genotypes were either regular-growing shoots with leaves, axillary buds and growing apices, or 2-leaf stem sections with apices and axillary buds removed. Regular sp 3SS:SFT donors were significantly more effective inducers of both uf sp and uf sp receptors, and, as expected, sp receptors were more responsive (compare Fig. 2 D and I). However, when sp and SP donors having mature leaves only were compared, they were equally effective (Table S3). Therefore, the superiority of sp donors can be attributed to their early maturation: 3SS:SFT donors continue to generate sink organs in the form of new branches, but branching is suppressed in sp 3SS:SFT donors (Fig. 1D). Florigen can therefore be seen as a hormone that by regulating leaf maturation in an SP-dependent manner, adjusts its own distribution at the whole-plant level.

Restrictions on the long-range function of FT are imposed by the cells and tissues in which SP is expressed (13). In Arabidopsis plants expressing 3SS:amiR-FT/SFT (Fig. S3), generating in both species effects that were similar to conventional loss of function. As shown in Table S3, it was possible to exploit the advantages imposed by the cells and tissues in which SFT is expressed (Fig. 3 A–E and Fig. S5). In contrast to the 3SS and the phloem-specific SUC2 promoters, expression of SFT, driven by 3 early leaf-specific promoters (BLS, FIL, and 650), although inducing precocious flowering, failed to generate graft-transmissible florigenic signals (see Fig. S5 for further discussion).

**Florigen Links the Transition to Flowering with Leaf Architecture.** To understand the genetic basis for phenotypic responses resulting from changing SFT/SP ratios, we examined the effects of different SFT/SP balances in mutant backgrounds with altered leaf morphology. Leaves of trifoliate (tf) plants form only 1 pair of lateral leaflets (24), but we observed that leaves of tf sp plants gradually lose their lateral leaflets, resembling the sequential reduction of leaflets in the compound leaves of the flowering rose shoots (Fig. 4A). A dysfunctional TF therefore sets higher sensitivity thresholds for changing SFT/SP ratios.

The effect of tf on the trifoliate leaf is analogous to its mild effect on WT leaves (compare Fig. 4B with Fig. 2B), but if SFT is inactivated in tf sp background the effect of sp is completely suppressed: tf sft sp and tf sft leaves were indistinguishable from each other (Fig. 4B). Significantly, tf and tf sp leaves were also indistinguishable (Fig. S2), suggesting that the allelic status of SP is irrelevant in sft leaves as in tf shoots (Table S1).

However, when SFT was overexpressed in tf (i.e., if 3SS:SFT), a functional SP, as expected of the sensitized tf background, was no longer sufficient to support the formation of lateral leaflets, and almost all leaves were simple (Fig. 4D and Fig. S4). As shown in Fig. 4E, if tf did not affect the expression profiles of SFT or SP.

To determine whether mobile florigen also inhibits leaflet meristems in the sensitized tf leaves, if 3SS:SFT donor shoots were grafted onto tf and tf sft sp receptors. Both receptors bear trifoliate leaves (Fig. 4B) and have a similar SFT/SP balance, but with functional and dysfunctional SFT and SP genes, respectively. As shown in Fig. 4 C and D, florigen induced simple leaves in both tf and tf sft sp receptors. In addition, shoots of the tf receptors shifted to 2-leaf SUs, as in if 3SS:SFT (Fig. 4C). If tf sft sp receptors, florigen complemented the sft gene and induced tf sp-like shoots. Thus, trifoliate leaves monitor both SFT/SP and sft/sp as balanced 1:1 ratios, and both genotypes are similarly modified by imported florigen, which is the ultimate manifestation of the florigen-dependent SFT/SP regulatory hierarchy.

The tomato leaf is initiated as a terminal leaflet and an elongated rachis. Independent pairs of lateral leaflets, each capable of duplicating the compound pattern, are then formed (24). In WT and tf, SFT and florigen regulate, in an SP-dependent context (Fig. 2), the arrest of lateral leaflets in a gradient opposite to their formation. The same is observed in tf sp or tf leaves importing florigen or overexpressing SFT (Fig. 4). In all these cases the terminal leaflets and their elongated rachis are unaffected, suggesting distinct regulatory mechanisms for the initiation of classes of leaflets. We surmise that florigen, mediated by the SFT/SP ratio, regulates the leaflet initiation gradient in conjunction with auxin (see SI Text and below).

**Florigen Links the Vegetative/Reproductive Balance in the Inflorescence with the Generation of Abscission Zones.** Abscission zones (AZs) mark the sites where plant organs, particularly fruits and leaves (Fig. 5A), are eventually separated from the main body of the plant. The formation of AZs is regulated by day length, auxin, and ethylene, and the deciduous habit is considered an important innovation of angiosperms (25, 26). In tomato, formation of floral pedicel AZs is preceded by site-specific cell divisions (27), and AZs are completely missing from jnlessless1 (j1) and j2 pedicels (Table S2).

mc, sft, and bl condition partial vegetative inflorescences similar to those seen upon overexpression of SP (17) (Table S2). In the course of studying the interactions between mc, bl, and florigen, we noticed that AZs in floral pedicels of mc, bl, and sft were incomplete, irregular, or mislocated. We found that similar to j1, floral pedicels of sft mc, sft bl, or mc bl double mutants completely lacked AZs (Fig. 5 B and C), indicating that mc and
J1 supplement experiments showed that mobile florigenic signals, which com-
rose toward flowering. 

Leaf architecture in Fig. 4.

Bl/SP regulatory hierarchy. 

Fig. 4. Leaf architecture in tf plants is determined by the florigen-dependent.

SFT/SP regulatory hierarchy. (A) Left: A flowering tf plant. All leaves have 3
leaflets. Middle: A tf sp flowering shoot with a gradual reduction in leaf complex-
ity. Right: Stepwise elimination of leaflets in compound leaves of the garden
rose toward flowering. (B) Inactivation of TF sensitizes the growth response of
leaves to changing SFT/SP ratios (ratios are listed below the corresponding im-
egages). SFT/SP ratio (tf, far left) and spf/3 ratio (tf, far right) result in similar
trifoliolate leaves. High ratios, as in overexpression of SFT or inactivation of SP
(leaves 3 and 4 from left, respectively) induced simple LANCEOLATE-like leaves
(24, 36). Note that tf sp leaves (second from left) have low SFT/SP ratio but are
indistinguishable from tf sp leaves (right-most), and both have extended
rachises and additional leaflets characteristic of tf leaves (Fig. 3). (C and D) Mobile
florigen modulates the SFT/SP balance to generate 2 leaf SUs and to arrest leaflet
meristems. (C) Florigen donor induced slender stems, simple leaves, and a reduced
number of leaves per SU in a tf receptor (Inset). (D) A systemic reconstitution of
high SFT/SP balance in tf leaves. Left: The contribution of florigen by a WT donor
is insufficient to reduce the complexity of tf receptor leaves. Middle: Systemic
reconstitution of a high SFT/SP ratio in tf sp leaves induces simple leaves. Right:
Mobile florigen elevates the level of SFT in tf sp leaves, thereby inducing
"reversion" to a simple architecture. (E) tf does not affect expression gradients of
flowering genes.

bl act redundantly with sft in specifying these tissues. Grafting
experiments showed that mobile florigenic signals, which com-
plement tf, penetrate floral pedicels to rescue AZs in sft mc and
sft bl pedicels (Fig. 5 D and F). Although SFT, SP, MC, BL, and
J1 are all expressed in pedicels and floral buds (Fig. 5E), the

corresponding proteins did not interact in any combination in
yeast 2-hybrid tests (data not shown).

AZs serve as a classical example of a morphogenetic trait
regulated by auxin (25, 26). Auxin is also involved in regulating
radial expansion of stems, formation of leaflet meristems, and
inflorescence patterning processes but is not essential for any of them (30).

Fig. 5. Florigen links the formation of AZs with inflorescence genes. (A) Normal
AZ of mature tomato fruit. (B) mc bl floral pedicels produce no AZs. (C) SFT
is required for the formation of AZs in an mc background. (D) Graft-
transmissible signals donated by a 35:SFT scion restore AZs in mc sft floral
pedicels. (E) Genes involved in generating floral AZs are expressed in WT floral
pedicels. YP, young pedicels before AZs can be observed; MP, mature pedicels
with developed AZs; YF, young flowers with pedicels removed. (F) Summary of
long-range complementation tests of AZs by florigen.

Discussion
Florigen as a Growth Hormone and the Evolution of the SFT/SP
Regulatory Hierarchy. The need for plant organs to respond
effectively to changing environmental signals dictates quantita-
tive regulatory schemes with inherent potentials for reversibility.
Such tenets are satisfied by florigen functioning as a general
growth hormone. SFT and SP modulate a variety of signaling
pathways but are not directly involved in the fate of cells or
organs. Rather, they regulate the balance of diverse growth
processes (20) and thus facilitate the potential for plasticity in
growth, not much different from the proposed role of the
Notch/Wnt system that pleiotropically modulates multiple sig-
naling processes but is not essential for any of them (30).

FT is an integrator of all flowering pathways (4), but as shown
here (Figs. 1, 2, and 4), SFT is imported by organs in which it is
already produced, with the exception of the SAM. It was recently
suggested that genes of the autonomous pathway carry pleiotropic
vegetative functions (31). Mutations in SOC1 and FUL, 2 major
flowering genes in Arabidopsis, affect vegetative functions (32).
Likewise, GA promotes flowering (33) and modifies growth. Given
our finding that florigen is not only a flowering-specific agent,
perhaps there are no designated flowering genes at all. One reason
could be that "flowering" is an arbitrary external description (34)
that does not match the internal description of the plant. By the
same token, the mechanism requiring movement of florigen from
leaves to apices (21, 22) may not be specific to flowering. Rather it
is one aspect of a more general mechanism in which the florigen
hormone is exploited to change local SFT/SP balances. If correct, florigen may regulate flowering in species in which SFT orthologues are normally expressed in apices, just as it regulates leaflet meristems in the compound leaf.

In shoots and leaves, meristems react to florigen in an SP-dependent manner. Here we offer an evolutionary scenario for the generation of this mechanism in flowering plants. Consider the following. (i) Overexpression phenotypes define SFT as a growth retardant (6). It terminates primary SAMs, sympodial SAMs, and inflorescence meristems and arrests stem and leaflet meristems (Figs. 1, 2, and 4). (ii) The arrest of meristematic activities by SFT/florigen is alleviated by even the low WT level of SP. SP is therefore an amazingly potent inhibitor of SFT. (iii) SP is functionally relevant only in the presence of a functional SFT. (iv) Expression of FT orthologues is correlated with photoperiodic bud setting in slow-growing conifers (35). With their highly conducive vasculature, fast-growing meristems, and need for a rapid response to environmental signals, the ability of flowering plants to exploit new habitats likely required high levels of FT/florigen, but these levels were also detrimental. We speculate that SP evolved specifically to alleviate the detrimental effects of SFT. This is supported, as shown in Fig. 6, by the absence of genes of the SP/TFL1/CEN clade from all known nonflowering plant genomes.

Methods

Plants were grown and grafted as specified previously (6). Monogenic lines and WT cultivars were obtained from the Rick Center at University of California, Davis. Combinations mentioned in the text and in Tables S1–S3 were specifically bred for this work. Cloning, RNA isolation, and PCR were performed as previously described (6). A list of primers, fragment sizes, and number of cycles for each gene is given in Table S4. Confocal imaging and microscopy were performed as described previously (39).

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