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Review

The transition to flowering in tomato

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Abstract Tomato (*Solanum lycopersicum* L.) is a very important commercial crop and also a useful model to study the transition to flowering in a sympodial perennial plant. Here we try and summarize past and recent progress in understanding the environmental cues that affect the initial transition to flowering in this species and the genes that are involved in this transition and additional transitions occurring on the sympodial shoot. Environmental cues discussed are daylength, light intensity and growth temperature. In the last eight years much progress has been made in identifying the genes and in analyzing genetic interactions of the different mutations. Most of the genes isolated so far seem to play similar roles in *Arabidopsis* flowering. For example, mutations in the tomato *SINGLE FLOWER TRUSS* gene cause late flowering and this gene was recently shown to encode a protein similar to that encoded by the Arabidopsis *FT* gene. *FT*-like proteins seem to act as major flower promoters in diverse species. We also discuss issues in tomato flowering that we believe still require further research.

Key words: *Solanum lycopersicum* L, Flowering, SINGLE FLOWER TRUSS, Environmental cues, Flowering-time mutants.

The genus Lycopersicon is native to western South America. Mexico appears to have been the site of domestication and the source of the earliest introductions of tomato (*Solanum lycopersicum* L.; previously named *Lycopersicon esculentum* Mill.). Likely environmental conditions in its natural habitat are little (25 mm) rainfall, high relative humidity, temperatures ranging from 10 to 24°C and photoperiods ranging from 11.5 to 12.5 hours per day (Cooper 1972). Tomato is a perennial plant usually grown as an annual.

There are several excellent reviews on tomato flowering (Wittwer and Aung 1969; Picken et al. 1985; Atherton and Harris 1986; Dieleman and Heuvelink 1992; Kinet and Peet 1997; Lozano et al. 2000; Lifschitz and Eshed 2006). Here our focus will be on the timing of the initial transition to flowering and how environmental conditions affect this timing. Our current knowledge in tomato will be briefly compared to that known in other species, especially *Arabidopsis*.

Unlike most model systems, such as *Arabidopsis* and rice, in tomato vegetative and reproductive phases alternate regularly along the compound (sympodial) shoots of tomato. The primary vegetative apex is terminated by an inflorescence (Sawhney and Greyson 1972), after 6–12 leaves have formed. As described below, the number of leaves depends on genetic background and on environmental cues. Upward growth then continues from a new vegetative shoot arising from the upper-most (proximal) side (axillary) bud (meristem) of the youngest leaf just below the terminating inflorescence. From then on, the stem is composed of reiterated units, sympodial segments, each with three nodal leaves and a terminal inflorescence. The position of the last leaf formed before the transition appears later on above the inflorescence. This is due to a partial fusion of its petiole with the new vegetative shoot arising from its axillary bud which displaces the inflorescence axis sideways, and places the leaf above it (Figure 1). Lateral branches emerging from axillary buds of other leaves, normally produce more leaves before appearance of the first flower, and are not partially fused to their host leaf petiole. This is an interesting point since although the plant has clearly already gone through a first transition to flowering, new branches do not seem to flower as quickly as sympodial branches (Lifschitz and Eshed 2006).

The developmental 'time' of transition can be best measured by counting the number of leaves (or nodes) formed on the initial apical meristem before it is terminated with the production of the first inflorescence. In this review we will not mention conditions or

Abbreviations: LFY, LEAFY; FT, FLOWERING LOCUS T; SOC1, SUPPRESSOR OF OVEREXPRESSION OF CO 1; AP1, APETALA 1; CO, CONSTANS; FA, FALSIFLORA; SFT, SINGLE FLOWER TRUSS; MC, MACROCALYX; FLC, FLOWERING LOCUS C; UF, UNIFLORA; AN, ANANTHA; SP, SELF PRUNING; J, JOINTLESS; BL, BLIND; S, COMPOUND INFLORESCENCE; LS, LATERAL SUPPRESSOR.
This article can be found at http://www.jspcmb.jp/

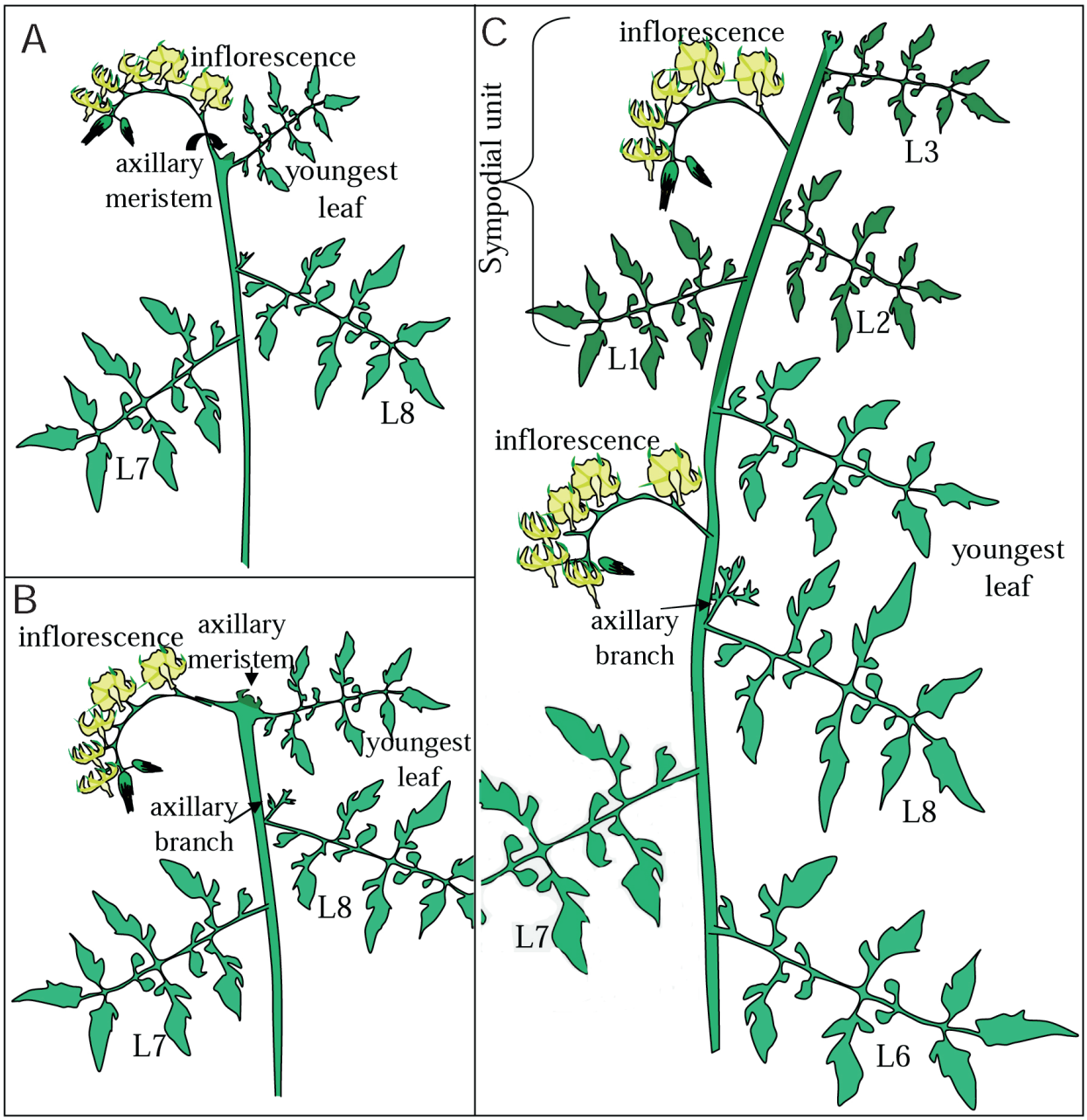

Figure 1. Growth of the compound tomato shoot. (A) The primary apical meristem produces 6–12 leaves (9 leaves in the figure) and terminates once it produces an inflorescence. The axillary meristem of the uppermost leaf (youngest leaf) starts to develop and will form the first sympodial segment. (B) The axillary meristem (dark green) starts to produce leaves. Its growth slowly displaces the position of the inflorescence to a lateral position. (C) The sympodial branch terminates after three leaves when the meristem produces an inflorescence. The next sympodial bud, formed by the axillary meristem in the axil of the youngest leaf (L3) will develop into the next sympodial unit. Lateral shoots in the axillaries of other leaves will flower after several leaves and will later maintain a sympodial growth pattern.

mutations which only result in a delay in the appearance of the flower, without a significant change in leaf number. Although this is a very important agricultural trait, in most cases it is likely due to a change in the rate of vegetative development.

Integrators of the transition to flowering

Our current knowledge on the molecular genetic mechanisms of flowering in *Arabidopsis* is mostly based on analysis of mutations that affect flowering time (Koornneef et al. 1991). In *Arabidopsis* a complex network of regulatory pathways controls flowering time in response to diverse environmental signals. These pathways converge on a small set of flowering-time genes, called floral integrators, such as *LEAFY* (*LFY*; Weigel and Nilsson 1995; Ruiz-Garcia et al. 1997), *FLOWERING LOCUS T* (*FT*; Kardailsky et al., 1999; Kobayashi et al. 1999); *SUPPRESSOR OF OVEREXPRESSION OF CO 1* (*SOC1*; Lee et al. 2000;

Samach et al. 2000; Corbesier and Coupland 2005), and APETALA 1 (AP1; Mandel et al. 1992; Mandel and Yanofsky 1995; Wigge et al. 2005) whose expression is controlled by different environmental signals and is closely correlated with flowering time. In *Arabidopsis* flowering is promoted in long day conditions. An increase in daylength causes a gradual accumulation of the CONSTANS (CO) protein (Putterill et al. 1995; Suarez-Lopez et al. 2001; Valverde et al. 2004) in leaves, which can directly activate *FT* expression in the vascular bundles of the leaves (Samach et al. 2000; Takada and Goto 2003; An et al. 2004). *FT* activity is detected in the leaf (Teper-Bamnolker and Samach 2005) and in the meristem (Wigge et al. 2005; Abe et al. 2005) suggesting that RNA (Huang et al. 2005) or protein can systemically reach the apical meristem.

In tomato, the *FALSIFLORA* (*FA*) gene (Table 1; Stubbe 1963; Molinero-Rosales et al. 1999) encodes a protein similar to LFY (Weigel et al. 1992) and its *Antirrhinum majus* homolog FLORICAULA (Coen et al. 1990). A mutation in this tomato gene causes a significant delay in flowering time. The number of leaves formed on each of the initial sympodial segments is also increased. In the *fa* mutant flowers are replaced by leaf producing inflorescences (http://tgrc.ucdavis.edu/Images/fa.GIF), quite similarly to the *lfy* phenotype in *Arabidopsis.* There are several alleles of *lfy* and only one described allele of *fa* (Allen and Sussex 1996; Molinero-Rosales et al. 1999). Several weak *lfy* alleles eventually produce some abnormal flowers, and abnormalities are due to an additional role of LFY in floral organ identity (Schultz and Haughn 1993). The total lack of flowers in the *fa* mutant is likely due to the severe mutation in the gene, a deletion causing a frame shift, leading to a truncated protein of 187 aa instead of 412 aa (Molinero-Rosales et al. 1999).

In tomato the *SINGLE FLOWER TRUSS* (*SFT*) gene encodes a protein similar to *Arabidopsis* FT (Lifschitz et al. 2006). The gene was previously named *SP3D* (Carmel-Goren et al. 2003) and *TFT* (Teper-Bamnolker and Samach 2005). Plants containing mutant *SFT* alleles produce many more leaves before initiating their first flower (Kerr 1982; Molinero-Rosales et al. 2004; Lifschitz et al. 2006; Quinet et al. 2006a). With the transition to flowering one (origin of gene name) or two flowers with some leaf-like sepals are made yet unlike wild type plants the primary vegetative apex does not terminate. The meristem goes on to produce additional leaves and flowers, continuing to form the main axis of the plant (Molinero-Rosales et al. 2004; Lifschitz et al. 2006). While in wild type plants, the proximal axillary bud develops into the new vegetative shoot, development of this shoot is suppressed in the *sft* mutant (Molinero-Rosales et al. 2004; Lifschitz et al. 2006).

When over-expressed in *Arabidopsis SFT* causes early flowering similar to plants overexpressing *FT* (Teper-Bamnolker and Samach 2005). *SFT* overexpression also causes early flowering in tomato (after 3–5 leaves) and short-day tobacco plants grown under non-inducing conditions (Lifschitz et al. 2006). Interestingly, similar to *Arabidopsis* FT, TFT originates a systemic flowering signal, elegantly proven using grafting experiments with the *35S*:*SFT* lines (Lifschitz et al. 2006). In these experiments there was no evidence supporting the possibility that RNA was moving through the grafts, as was suggested in *Arabidopsis* (Huang et al. 2005).

The meristem of the primary vegetative apex seems to be very responsive to high levels of SFT since *35S*:*SFT* tomato plants flower with very few leaves. Interestingly, the meristems of the sympodial segments might be less responsive as the number of leaves in each segment is only reduced from three to two. In *Arabidopsis* over-expression of *FT* resulted not only in early flowering but also in morphological changes in the leaves (Teper-Bamnolker and Samach 2005). In tomato high transcript level of *SFT* affected the character of the compound leaf and the stem (Lifschitz et al. 2006). It was suggested that SFT is involved in controlling growth rates and that flowering is one pleotropic effect of SFT function (Lifschitz and Eshed 2006).

The *sft fa* double mutant produced more than 100 leaves after 1 year of growth with no formation of an inflorescence (Molinero-Rosales et al. 2004). This suggests that similar to *LFY* and *FT* in *Arabidopsis* (Ruiz-Garcia et al. 1997), *SFT* and *FA* act in parallel pathways to promote flowering in tomato. *35S*:*SFT* plants 'flower' with a similar number of leaves in the *fa* mutant background, yet the nature of the 'flower' is that of an *fa* 'flower'—a leafy inflorescence (Lifschitz and Eshed 2006). Overexpression of both FT and LFY in *Arabidopsis* cause the plant to make flowers immediately after cotyledons (Kardailsky et al. 1999; Kobayashi et al. 1999). It would be interesting to see what the consequences of a similar combination in tomato would be.

A mutation in the *MACROCALYX* (*MC*) gene encoding an AP1/ SQUAMOSA-like MADS box transcription factor (Robinson and Tomes 1968; Vrebalov et al. 2002) causes formation of an indeterminate inflorescence, with leaf-like sepals. The gene (also named *LeMADS-MC*) is adjacent to the *RIPENING-INHIBITOR* (*RIN*) locus (also named *LeMADS-RIN*) and was identified with the cloning of *RIN*. The *rin* mutation is a result of a deletion that fuses both genes together. Antisense *MC* transgenic tomato plants have an indeterminate inflorescence together with leaf-like sepals, confirming the role of the *MC* gene. Overexpression in tomato of the *Arabidopsis AP1* gene caused early flowering with initially little effect on the flowering of the sympodial shoot. Later on, the

Table 1. Some mutations affecting flowering in tomato

Mutation			Protein similarity		Description	Affect on first flowering		Inflorescence identity	Flower structure	Sympodial flowering	Lateral meristems	Double mutants										
		citation		citation			environment					*an*	*bl*	*s*	*fa*	*j*	*ls*	*sp*	35S:SP	*sft*	35S:SFT	*uf*
anantha	*an*	8	unknown		1, 5, 18, 19, 29	–		+	+			19	29		1	19, 29		19	19			
blind	*bl*	23	R2R3 MYB	25	14, 15, 20, 29						+	29				29	25, 29	25				21
compound inflorescence	*s*		unknown		20	+	+	+														21
falsiflora	*fa*	27	LEAFY	16	1	+		+	+	+		1								17		
jointless	*j*	2	MADS	13	6, 20, 28, 29			+				19, 29	29				29	22				21
lateral suppressor	*ls*		VHIID	26	12, 29						+		25, 29			29		29				
macrocalyx	*mc*	24	MADS	30				+	+													
self pruning	*sp*	32	CETS	19	29					+		19	25			22	29		19	17	10	21
single flower truss	*sft*	9	CETS	11	17, 20	+		+	+						17			17			11	10
uniflora	*uf*	7	unknown		3, 4, 5, 11	+	+	+					21	21		21		21		10	11	

Allen and Sussex 1996 (1); Butler 1936 (2); Dielen et al. 1998 (3); 2001 (4); 2004 (5); Emery and Munger 1970 (6); Fehleisen 1967 (7); Helm 1951 (8); Kerr 1982 (9); Lifschitz and Eshed 2006 (10); Lifschitz et al. 2006 (11); Malayar and Guard 1964 (12); Mao et al. 2000 (13); Mapelli and Lombardi 1982 (14); Mapelli and Kinet 1992 (15); Molinero-Rosales et al. 1999 (16); 2004 (17); Paddock and Alexander 1952 (18); Pnueli et al. 1998 (19); Quinet et al. 2006a (20); 2006b (21); Rick and Sawant 1955 (22); Rick and Butler 1956 (23); Robinson and Tomes 1968 (24); Schmitz et al. 2002 (25); Schumacher et al. 1999 (26); Stubbe 1963 (27); Szymkowiak and Irish 1999 (28); 2006 (29); Vrebalov et al. 2002 (30); Williams 1960 (31); Yeager 1927 (32).

transgenic lines showed earlier termination due to immediate flowering of the sympodial shoot (Ellul et al. 2004)

Environmental effects on tomato flowering time

Environmental conditions can affect the timing of the transition from vegetative to reproductive development of many species (Bernier and Perilleux 2005), including tomato. Based on experiments in controlled conditions, it seems that the environment can affect flowering time in tomato only after cotyledon expansion of the germinating seedling.

Temperature

The best studied effect of temperature on flowering-time is the response to extended exposure to low temperatures called "vernalization". To avoid flowering before the end of winter, several annuals will not flower before experiencing a certain long period of cold temperatures. Great progress in understanding the molecular mechanism of vernalization in *Arabidopsis* was recently reached (Sung and Amasino 2005; Baurle and Dean 2006; Dennis et al. 2006). The *FT* and *SOC1* integrators are transcriptionally repressed by the MADS box transcription factor FLOWERING LOCUS C (FLC) (Hepworth et al. 2002; Helliwell et al. 2006; Searle et al. 2006). In *Arabidopsis* ecotypes which respond to vernalization, vernalization relieves *FLC* transcriptional repression in both the leaf (*FT*) and meristem (*SOC1* and *FD*) allowing photoperiod-dependent production of systemic signals in the leaves and conferring competence on the meristem to respond to these signals (Searle et al. 2006). The expression of *FLC* is down-regulated by cold temperature through stable changes in chromatin structure caused by specific changes in histone modification at the *FLC* locus. A long period of cold leads to an increase in expression of the *VERNALIZATION INDEPENDENT 3* (*VIN3*) gene. *VIN3* encodes a PHD finger protein required for histone deacetylation at the *FLC* locus (Sung and Amasino 2004), a first step in a series of modifications leading to a fixed epigenetic state of transcriptional repression.

In tomato seed 4°C vernalization shows no effect on the number of nodes preceding the first inflorescence or the number of flowers (Calvert 1957; Wittwer and Teubner 1957). This is not surprising, since the species originated from regions that do not have extremely cold winter conditions. Still, mild ambient temperatures during seedling growth do cause earlier flowering. For example, when comparing flowering time of the 'Ailsa Craig' variety grown under warm (27°C) or low (10°–15°C) temperatures, the number of leaves till the first inflorescence was reduced from 14 to 8 leaves in the lower temperature regime (Calvert 1957). The early flowering response (reduction in leaf number till first flowering) to low temperatures seems to be limited to the first nine days after cotyledon expansion, termed the 'sensitive phase' (Calvert 1957; Wittwer and Teubner 1957). The responsive tissue seems to be the aerial part of the plant (Phatak et al. 1966). Since high temperatures cause an increase in rate of leaf production (Calvert 1959), the exact time in which the first inflorescence reaches anthesis is a 'balancing act' between the number of leaves till the first flower, and the rate of their production. Since the number is reduced by low temperatures, and the rate is increased by high temperatures, faster flowering can be achieved by combining a period of low temperatures followed by a period of high temperatures (Wittwer and Teubner 1957). In some species there is evidence for a time-of-day-sensitivity to temperature. For example, in *Arabidopsis*, warm night temperatures caused a more significant reduction in flowering time compared to warm day temperatures (Thingnaes et al. 2003; Paltiel et al. 2006). A similar experiment in tomato did not reveal any significant differences between day or night cold (10°C) temperature treatments under 21°C ambient growth conditions (Calvert 1957; Calvert 1964b).

Temperature also affects the number of flowers in each inflorescence (Hurd and Cooper 1967). Here, the 'sensitive phase' affecting the first inflorescence seems to be eight to twelve days after cotyledon expansion (Lewis 1953). The effect of low temperature on flower numbers is not perturbed when followed by high temperatures. For example in one experiment seedlings after cotyledon expansion were grown under low temperatures (10°–13°C) for two weeks and then high temperature (21°–24°C) for four weeks and the cold effect was not nullified (Lewis 1953; Wittwer and Teubner 1957). Both Lewis (1953) and Calvert (1957) found that extending the length of the treatment resulted in an increased number of flowers up to the 5th inflorescence. Further studies will hopefully provide the genetic basis for low-temperature-dependent induction of flowering in this species. It would be interesting if this process is also controlled through epigenetic regulation, similar to low-temperature-dependent vernalization in *Arabidopsis.*

Light

A plant is exposed to varying light conditions due to changes in the relative position of the sun (seasonal and daily), and position and quality of objects (clouds, other plants etc.) which filter the light of the sun before it reaches the plant. These changes are measured in the total quantity of light the plant receives during the day (termed 'daily light integral'), daylengths (photoperiod) and light quality (wavelengths). We will discuss each of these factors below.

In *Antirrhinum majus* L. increasing the light intensity decreases the flowering time in days and in leaf number, when the intensity is too low, it may lead to delay of flowering for over two years (Cremer et al. 1998; Munir et al. 2004). When tomato is grown under glasshouse conditions with no artificial light, an obvious delay in flowering occurs under winter conditions (Goodall 1937; Calvert 1964a). Experiments under controlled environmental conditions clearly showed that the number of leaves till flowering decreases (by ~2 leaves) with an increase in daily light integral under a certain photoperiod (Calvert 1959). This effect of light is less pronounced under low temperatures (discussed above) that also promote flowering (Calvert 1959; Hussey 1963). This suggests that both environmental stimuli might work on the same molecular target. In such a scenario, by exposing the plant to one stimulus, saturation is reached so that the plant no longer responds to the other stimulus.

Mutations in the *UNIFLORA* (*UF*) gene (Fehleisen 1967; Dielen et al. 1998; Dielen et al. 2001; Dielen et al. 2004; Lifschitz et al. 2006) cause late flowering when measured by number of days and number of leaves preceding the first inflorescence. The *uf* mutation doesn't affect the rate of the leaf production. Late flowering of the mutant was much less pronounced when plants were grown during the summer (Dielen et al. 1998; 2004) suggesting that under high daily light energy integrals, loss of UF is compensated by other genetic pathways. This was proven by growing plants under controlled environmental conditions with different light conditions (Dielen et al. 2004). Under low daily light energy integrals many *uf* mutant plants do not flower, and those that do, may produce the first flower after more than 40 leaves. This suggests that UF is a major component (still with unknown function) in the molecular mechanism controlling floral transition in tomato (Dielen et al. 1998; 2004; Lifschitz et al. 2006).

The single-flower phenotype of this mutant occurs independently of light intensity and might be due to two major events. One possibility is that the apical meristem is now terminated by one flower instead of an inflorescence, so that UF is involved in inflorescence meristem identity in addition to its role in flowering time (Dielen et al. 1998). The other possibility is that unlike the wild type the inflorescence meristem is also producing leaves, similar to what was described for *sft*, so that the new leaves produced above the flower are made by the initial apical meristem and not by sympodial growth of the side meristem. This was termed a "pseudoshoot" (Lifschitz et al. 2006).

In this second model one can speculate that a general low "competence" to flower caused by the loss of UF causes both a delay in the first flower that is formed, and an incomplete transition of the apical meristem to an inflorescence meristem. Still, these two phenotypes are not always linked in *uf*. No known environmental treatment can rescue the single flower phenotype while high light can reduce late flowering. Also, while introducing high levels of *SFT* can cause the *uf* mutant to make first flowers after only three leaves (Lifschitz et al. 2006), the single flower phenotype is not completely rescued, even though extra flowers are made. The fact that high levels of SFT can cause only partial rescue of one phenotype (single flowers) while causing a total reversion of the flowering time phenotype (early instead of late flowering) might suggest SFT as a downstream target of UF, but probably not it's only target.

Under intermediate light energy integrals *uf* plants develop lateral branches in the axil of leaves 8 to 13, where normally the wild type plant initiates an inflorescence. It was suggested that branch formation is a result of a temporary release from apical dominance since upper nodes do not form lateral branches (Dielen et al. 1998; Dielen et al. 2004). It was also suggested that *uf* mutants undergo a partial evocation but then go back to vegetative growth, unable to finish the flowering process (Dielen et al. 1998; Dielen et al. 2004). It has been recently reported (Lifschitz and Eshed 2006) that the double mutant *sft uf* does not flower, suggesting that both genes have a mutual target but reach it using independent pathways.

One of the predictable changes in the environment in regions far from the equator is a gradual change in daylength. Many annuals use these changes in photoperiod to correctly time their transition to flowering. Some plants will not flower unless exposed to a long enough or short enough daylength while others flower faster as days become longer or shorter. As mentioned above, in *Arabidopsis* flowering is promoted in long day conditions via CO activation of *FT* transcription. Overexpressing the *CO* gene causes very early flowering even under short day conditions in *Arabidopsis* (Onouchi et al. 2000).

Tomato produces flowers under both short and long photoperiods, and is considered by many as a photoperiod insensitive plant (Lifschitz et al. 2006). In fact, several experiments under controlled conditions have shown a slight significant reduction in leaf number under short days in many cultivars (Reinders-Gouwentak 1954; Wittwer 1963; Morgan et al. 1971; Hurd 1973; Aung 1976; Kinet 1977). The photo-morphogenetic response to photoperiod is correctly assayed when the additional hours of light provided in the long day treatment are of very low intensity in wavelengths that are less efficient for photosynthesis, and more efficient for a photoperiodic response (for example, low intensity incandescent light). This treatment is termed extended short days. Since, as discussed above, an increase in daily light integral reduces flowering time, in some

experiments in which long photoperiods were provided with high light intensity, the overall delaying effect of long photoperiods might have been reduced by the promotive effect of the additional light.

Overexpressing the *Arabidopsis CO* gene or tomato *CO-like* genes in tomato did not seem to affect flowering time, even when one of the tomato genes does have a strong effect on *Arabidopsis* flowering (Ben-Naim et al. 2006). This might suggest that *CONSTANS-like* proteins are not linked to flowering in tomato, yet perhaps those that do have not been characterized yet.

Flowering time in *Arabidopsis* is strongly attenuated by light wavelength, where red light acts to delay flowering and blue and far-red light act to expedite the transition to flowering. The role of light is most likely through it's effect on CO protein stability and perhaps also CO-independent affects on *FT* and other genes expression (Halliday et al. 2003; Valverde et al. 2004; Paltiel et al. 2006). Light is perceived by the plant through photoreceptors, the main ones being the red/ far-red light phytochrome (PHY) receptors and the blue light CRYPTOCHROME (CRY) receptors (Samach and Pineiro 2002; Cerdan and Chory 2003; Lin and Shalitin 2003).

Thorough genetic analysis have been performed on tomato photoreceptor, chromophore and signal transduction mutants (Van Tuinen et al. 1995; van Tuinen et al. 1995; Kendrick et al. 1997; Lazarova et al. 1998a; Lazarova et al. 1998b; Ninu et al. 1999; Perrotta et al. 2000; Weller et al. 2000; Weller et al. 2001; Davuluri et al. 2004). As far as we know there is no clear flowering time phenotype associated with mutations in these genes. Over-expression or silencing of the tomato blue light receptor CRY2 had no effect on the developmental timing of the transition to flowering, although the rate of leaf production was altered causing a delayed appearance of flowers (Giliberto et al. 2005). We find it quite interesting that there are no clear flowering time phenotypes described for these mutants, even though flowering is affected by light intensity and photoperiod.

Additional genes involved in tomato flowering

Compared to *Arabidopsis*, the number of loci described in the literature to have an effect on flowering time in tomato is relatively small. Relatively few QTL tests were performed to detect early flowering loci (Honma et al. 1963). Past screens for flowering time mutants in tomato were much less extensive and perhaps were performed under environmental conditions in which subtle phenotypes are not clear. A recent screen identified 41 mutations affecting flowering time (Menda et al. 2004). These and other mutations can be seen in the website: "Genes that Make Tomatoes" (http://zamir.sgn.cornell.edu/mutants/). Another excellent source for mutations is the "C.M. Rick Tomato Genetics Resource Center" (TGRC; http://tgrc.ucdavis.edu/index.aspx; Chetelat 2005). An additional argument may be that *Arabidopsis* flowering is sensitive to more environmental cues and therefore there are more genes directly involved in flowering time. Aside from the genes directly involved in the control of flowering time, many mutations in *Arabidopsis* have pleotropic effects, including changes in flowering time. With our growing knowledge of the pathways of *Arabidopsis* flowering, we understand more about some of these mutations. For example, photoperiodic flowering in *Arabidopsis* requires an intact circadian clock. Therefore any mutation that perturbs the clock has an effect on flowering time (Mizoguchi and Coupland 2000; Samach and Coupland 2000). Gene expression patterns reveal that tomato has a circadian clock (Ben-Naim et al. 2006; Facella et al. 2006), yet since photoperiod has little effect on flowering of tomato, mutations in the clock apparatus may not influence tomato flowering. Another example may be vernalization. In *Arabidopsis*, vernalization causes epigenetic modifications in histones surrounding the *FLC* locus, encoding a floral repressor, leading to a shut down of transcription of this gene, and a loss of repressor activity. Thus, any mutation that modifies epigenetic regulation is likely to have a strong flowering time effect (Sung et al. 2006). Since vernalization does not affect tomato flowering, it is possible that histones surrounding flowering time loci in tomato are not modified by the environment. Perhaps all mutations which affect epigenetic regulation in tomato have no flowering time effect.

In the *anantha* (*an*) mutant (Helm 1951; Paddock and Alexander 1952; Allen and Sussex 1996; Pnueli et al. 1998; Dielen et al. 2004) the transition to flowering is normal, as well as sympodial flowering, yet flowers are replaced by a meristematic tissue that keeps on dividing leading to a cauliflower-like structure (http://tgrc.ucdavis.edu/Images/an-LA0536-flowers.jpg). *fa* is completely epistatic to *an*: the double mutant looks like an *fa* single mutant (Allen and Sussex 1996).

In *the self pruning* (*sp*) mutant caused by a mutation in a CETS family protein with a similar sequence as the *Arabidopsis TERMINAL FLOWER 1* protein (Yeager 1927; Pnueli et al. 1998) the sympodial shoot no longer makes three leaves before termination. Termination of consecutive sympodial shoots occurs with less leaves and ends with an inflorescence followed by an additional inflorescence leading to a determinate growth pattern. This mutation was extensively used in breeding programs. The number of leaves formed before the first transition to flowering is not affected by this mutation in a wild type or in a *sft* mutant background (Molinero-Rosales et al. 2004). Still, the mutation did seem to

reduce the number of leaves formed before flowering in a *uf* background (Quinet et al. 2006b). Overexpression of SP in wild-type and *sp* plants resulted in development of extra leaves in the inflorescence (Pnueli et al. 1998). The *an sp* double mutant produces *anantha* inflorescences (Pnueli et al. 1998). Overexpression of *SP* in the *an* background changed the fate of the *an* meristems to production of leaves or shoots, similar in appearance to *fa* inflorescences (Pnueli et al. 1998). This and the an *fa* phenotype suggests that *FA* might normally act to increase or activate AN and to lower SP levels or activity.

The *jointless* mutant (*j*), is caused by deletion in a MADS-box gene, named after its lack of an abscission zone on flower pedicels (Butler 1936; Emery and Munger 1970; Szymkowiak and Irish 1999; Mao et al. 2000; Quinet et al. 2006a; Szymkowiak and Irish 2006). Although *j* mutants did seem to flower later, the differences in leaf number were not statistically different (Quinet et al. 2006a). The inflorescence reverts to vegetative indeterminate growth after forming one to three flowers (http://tgrc.ucdavis.edu/Images/j-LA3033,-bud-compii.jpg; Rick and Sawant 1955; Szymkowiak and Irish 2006). Introducing the *sp* mutation into the *j* background terminates the sympodial meristem before formation of leaves (Rick and Sawant 1955). Introducing the *an* mutation into the *j* background, upon the 'floral transition' the meristem continues to initiate leaf primordia, yet, unusually, the axillary meristem formed between the new leaf and the apical meristem develops at the same rate as the apical meristem, resulting in two meristems and a leaf from each meristem in each cycle of activity (Szymkowiak and Irish 2006). Interestingly, the *an j* double mutant also produced fruit-like structures made of leaves and vegetative shoots (Szymkowiak and Irish 2006), as if the plant is still capable of planning a fruit structure although with the wrong organs. The leaves formed after transition are much more developed compared to the leaves formed in single *an* mutants, suggesting that JOINTLESS is involved in repression of leaf development.

In the *blind* (*bl*) mutant (Rick and Butler 1956; Mapelli and Lombardi 1982; Mapelli and Kinet 1992), caused by a loss of function of an R2R3 class *Myb* gene (Schmitz et al. 2002), axillary buds are not formed due to lack of lateral meristem initiation (http://tgrc.ucdavis.edu/Images/bl-LA0059-flwr-comp.jpg). Shoot growth is terminated early after formation of an inflorescence. In some alleles a sympodial shoot is not formed (Szymkowiak and Irish 2006). RNAi inhibition of the gene in tomato causes transformation of side shoots into leaves (Schmitz et al. 2002). Introducing *bl* into the *uf* background reduced the late flowering phenotype of *uf*, yet the influence of other genetic loci introduced by this cross on this phenotype could not be ruled out (Quinet et al. 2006b). Introducing *bl* into the *j* background causes an interesting synergistic phenotype: with the transition to flowering, one terminal fertile flower with leaf-like sepals is produced, with no side branches or sympodial shoots (Szymkowiak and Irish 2006). Thus, in the absence of these two genes a very simple yet fertile tomato plant is formed, capable of making normal leaves and one flower with leaf-like sepals which terminates the meristem. Again, the *bl j* double mutant phenotype suggests that JOINTLESS is involved in repression of leaf development. The *bl an* double mutant causes the replacement of flowers, normally formed in *bl* with enlarged meristems producing leaf primordia. Thus, a flower will ultimately be replaced by a shoot containing 17–18 leaves. Similar to *j an* double mutants, fruit-like structures are formed in the *bl an* double mutant, and are even more developed in this background, reaching a stage of ripening similar to real fruit (Szymkowiak and Irish 2006).

The *compound inflorescence* mutant (*s*; chromosome 2) produces a highly branched inflorescence bearing up to 200 flowers. This is caused by the inflorescence meristem generating two inflorescence meristems instead of one floral and one inflorescence meristem as occurs in wild type plants. This leads to a ramification of the inflorescence structure. The number of leaves till flowering is slightly higher in the mutant when plants are grown under winter conditions, suggesting a flowering time phenotype revealed under low light integral (Quinet et al. 2006a). Indeed, crossing this mutation to the *uf* mutation described above, causes even later flowering than that of single mutants under low light conditions (Quinet et al. 2006b).

In the *lateral suppressor* (*ls*) mutant caused by loss of function of a VHIID type protein (Williams 1960; Malayer and Guard 1964; Schumacher et al. 1999) axillary meristems are mostly absent yet reappear in the axils of leaves situated just before the terminated meristem, allowing sympodial growth. Additional mutations in *SP*, *BL* and *J* were additive (Szymkowiak and Irish 2006). The *ls bl* double mutants lacked all axillary buds including a sympodial meristem. The *ls j* double mutants lacked all axillary buds on the main stem and on the leafy inflorescence formed due to the *j* mutation (Szymkowiak and Irish 2006).

The one flower inflorescence phenotype of *uf* was still found in double mutants of the *uf* mutant together with *s*, *sp*, *bl* and *j*, suggesting that the *uf* mutation is genetically epistatic to the other mutations regarding this phenotype (Pnueli et al. 1998; Quinet et al. 2006b). The determinate character of *sp* was still found in double mutants with *an*, *bl*, *j*, *ls*, *sft* and *uf* (Rick and Sawant 1955; Pnueli et al. 1998; Schmitz et al. 2002; Molinero-Rosales et al. 2004; Quinet et al. 2006b; Szymkowiak and Irish 2006).

Final remarks

The study of flowering in tomato initiated quite a few years ago, and mutations, mostly affecting inflorescence identity, have been identified. In the last eight years much progress has been made in identifying the genes and in analyzing genetic interactions of the different mutations. Most of the genes isolated so far seem to play similar roles in *Arabidopsis* flowering. Remaining major goals would be to understand molecular mechanisms of response to different environmental cues, and how flowering of the main and sympodial shoots are differentially controlled in tomato.

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