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Genetic interactions that promote the floral transition in *Arabidopsis*

Francisco Madueño, Leonor Ruiz-García, Julio Salinas and José M. Martínez-Zapater

The transition from vegetative to reproductive development is marked by the establishment of the floral developmental program in the lateral primordia formed by the shoot apical meristem. This is achieved by the coordinate activation, in these primordia, of a set of genes that regulate floral initiation. The time of this activation is determined by a complex network of loci involved in several pathways that are dependent or independent from environmental conditions. In Arabidopsis, *the construction and characterization of double mutants is helping to understand the interactions between loci controlling flowering time and the genes that are required to initiate the process of flower development at the lateral primordia.*

Key words: *Arabidopsis thaliana* / floral transition / flowering time / flower initiation / late-flowering mutants

PLANT DEVELOPMENT is the result of the growth and differentiation taking place at groups of actively dividing cells known as meristems. Development from the shoot apical meristem proceeds through the reiterated production of primordia that will give rise to vegetative (leaves and lateral branches) or reproductive (flowers) organs.[1] In many species, the time of transition between vegetative and reproductive development, hereafter called the floral transition, is regulated by environmental factors like light and temperature.[1,2] This favors flowering being initiated under suitable environmental conditions, which ensures reproductive success.[2] Until recently, very little was known about the underlying mechanisms controlling the floral transition and flower development. However, the combination of genetic and molecular analysis in suitable plant species like, snapdragon (*Antirrhinum majus*) and the weed *Arabidopsis thaliana* is rapidly changing our understanding of these unique plant developmental processes.[3-9] In this article, we will describe *Arabidopsis* vegetative and reproductive development and then review the available information on the role of some of the loci that seem to be involved in the promotion of the floral transition and that have been identified by late flowering mutations. We will also summarize the current understanding of the genetic control of floral initiation in order to discuss the possible genetic interactions between the genes regulating this process and the loci involved in the promotion of floral transition, a topic that has not been previously reviewed.

From the Departamento de Biología Molecular y Virología Vegetal, Centro de Investigación y Tecnología, Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria, Ctra. de La Coruña Km 7, 28040 Madrid, Spain

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Several phases can be distinguished during *Arabidopsis* development

Arabidopsis belongs to a group of species that grow as rosettes during their vegetative development (Figure 1). During this stage, the shoot apical meristem produces lateral primordia, which give rise to leaves with axillary meristems, appearing at positions known as nodes.[6,7] The formation of the rosette is the consequence of the lack of internode elongation between consecutive nodes. Based on leaf size, shape and position, two phases can be distinguished during rosette development. In the juvenile rosette, leaves are small and round, and are initiated as opposite pairs, while later, in the adult rosette, leaves are big and ovalate, and appear following a spiral organization or phyllotaxy.[10,11] In Arabidopsis, the floral transition is marked by the generation of nodes where leaf development is inhibited and whose axillary meristems develop as flowers. This process is generally paralleled by the elongation of the last rosette internodes (bolting) which together with the elongation of internodes between flowers, give rise to the inflorescence (Figure 1). Based on node morphology at maturity, two phases can also be distinguished along inflorescence development.[12,13] In the early inflorescence, nodes bear lanceolate leaves with axillary

meristems, which will develop as secondary inflorescences. In the late inflorescence, nodes bear flowers that are not subtended by leaves. The shoot apical meristem of wild-type plants never differentiates into a flower; but keeps growing indefinitely, producing lateral floral meristems until senescence.[13] The time of floral transition is regulated by unknown endogenous signals and, in *Arabidopsis*, it is sensitive to environmental conditions that can hasten or delay the process.[6,14] Long photoperiods shorten the time of floral transition and, in some genotypes, temporal exposure to low non-freezing temperatures (vernalization) also causes rapid flowering.[15,16]

The process of floral transition causes dramatic changes in the fate of the lateral primordia produced by the shoot apical meristem. The initiation of leaves is inhibited, and the fate of the axillary meristem is reprogrammed to produce a flower instead of a secondary inflorescence.[7,13] This change in the developmental program involves: (i) supression of internode elongation; (ii) changes in organ identity to give rise to the typical floral organs, sepals, petals, stamens and carpels; (iii) changes in phyllotaxy, since floral organs appear in concentric rows (whorls), and (iv) inhibition of meristematic activity, as seen by the supression of axillary meristem formation and the differentiation of all the cells of the floral meristem. Flower development in *Arabidopsis* is a good example of a developmental process subjected to canalization. Once the flower developmental program is initiated, it proceeds independently from either the environmental conditions, the genetic background that determines the time of the floral transition or the position of the flower at the inflorescence. However, in other species, like *Impatiens balsamina*, a change in environmental conditions can produce a reversion from the flower developmental program to the vegetative program,[17] indicating the requirement for floral inductive conditions during all stages of the process of flower development.

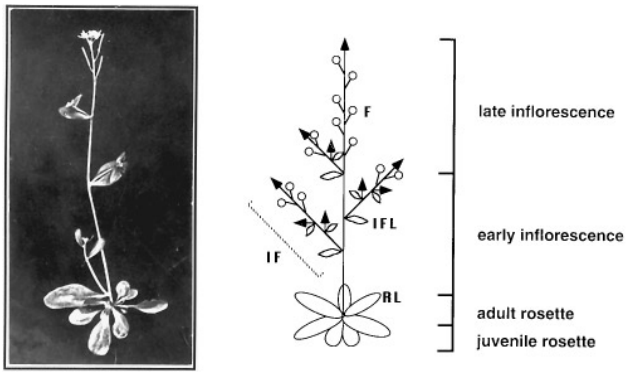

Figure 1. Left: wild-type Arabidopsis plant grown under photoperiodic inductive conditions. Right: Schematic representation of the wild-type plant indicating the different developmental phases. Arrowheads represent meristems. RL, rosette leaves; IFL, inflorescence leaves; IF, secondary inflorescences; F, flowers.

Genetic control of floral promotion

The isolation and characterization of mutations that affect flowering time in *Arabidopsis* has permitted the identification of more than 20 loci required to control the time of the floral transition under different environmental conditions.[6-9] Mutations in, at least, 12 of these loci produce a delay in the flowering time when plants are grown under inductive conditions (long photoperiods or continuous light), indicating that the corresponding loci could be involved in the promotion of floral transition.[18-23] These mutations not only delay the time of opening of the first flower but also increase the total number of leaves that are produced by the shoot apical meristem, which represents a developmental measurement of flowering time.[6,20] The morphological characterization of late flowering mutants at eight of these loci has revealed that they are not only affected in the timing of the floral transition but also show a significant elongation of all their developmental phases. The phase transition delays caused by these mutations are similar to the effect of short photoperiods on the development of wild-type plants (ref 11, Martínez-Zapater *et al*, manuscript in preparation). These results suggest that the corresponding loci are either transiently required at a very early stage in the life cycle of the plant, as was also suggested for the *GI* locus based upon the results of temperature shift experiments,[24] or they play a role during most of the life cycle. In this context, expression analyses of some of the genes, that have already been cloned (ref 25, and other articles in this issue), are in agreement with the second possibility.

In spite of the common phenotype of the late flowering mutants under floral inductive photoperiods, their morphological and physiological characterization has prompted their organization into, at least, two phenotypic groups.[6,8,20,26] The first group would include mutants *fca*, *fpa*, *fve*, *fy* and *ld*. These mutants are delayed in flowering time under any photoperiod,[20,27] and show a reduction in the elongation of inflorescence internodes (ref 11, and Martínez-Zapater *et al*, manuscript in preparation). Moreover, a vernalization treatment of germinating

mutant seeds strongly decreases their flowering time, rescuing the wild-type phenotype.[11,20,26,27] The current understanding of the role of the corresponding loci is that they could be involved in a constitutive pathway required to hasten plant development and for internode elongation. Consistent with this constitutive role, expression of the *LD* message is found in plants at different developmental stages and under different photoperiodic conditions.[27] The results from transgression analysis performed by M. Koornneef[6,20] also support the involvement of these loci in a single developmental pathway, since double mutants carrying mutations in two loci of this group do not flower later than the later parent.

A second group of late flowering mutants would include mutants like *co, fd, fe, fha, ft fwa* and *gi*. They show a phenotype characterized by a delay in flowering time under photoperiodic inductive conditions, while short photoperiods do not produce such a strong delay as in mutants of the first group.[6,20] Moreover, vernalization does not rescue the wild-type flowering time in these mutants.[8,20,26] This group of mutations has been interpreted as corresponding to loci involved in a developmental pathway responsible for the promotion of floral transition under photoperiodic inductive conditions.[6,8,20] In agreement with this interpretation, the level of the *CO* transcript, the only gene of this group that has been cloned, is higher in seedlings grown under long than under short photoperiods (ref 25, and Simon and Coupland, this issue). Differences in the responses to short photoperiods observed among these mutants identify *co, gi*, and *fha* as distinct from the rest of the mutants in this phenotypic group. Mutants *co, gi* and *fha* exhibit the same flowering time independently of the photoperiodic conditions, while the other mutants still maintain some photoperiodic responses.[8,25] Furthermore, we have recently found that when sucrose is provided to the shoot apex, it rescues the wild-type flowering time in all the late flowering mutants except in *fwa* and *ft* (Roldán *et al*, manuscript in preparation). Taken together, these results indicate that this second group of late-flowering mutants is not phenotypically homogeneous and the corresponding loci could be involved in more than one developmental pathway.

Although late-flowering mutations have a drastic effect on the timing of floral transition, they do not seem to alter the process of flower development.[6] Nevertheless, when late-flowering mutants are grown under short photoperiods, between 10% and 20% of the plants show abnormalities in the first flowers of the inflorescence. These flowers give rise to abnormal fruits (siliques) containing small inflorescences that, in some cases, can break through the carpels to produce abnormal secondary inflorescences (Figure 2) (Ruiz-García *et al*, unpublished observations). These structures could be considered as aborted floral transition events, and would represent failures of the canalization process under extreme conditions, suggesting the involvement of the genes identified by late-flowering mutations on the regulation of the floral initiation process. Similar structures also develop as a result of short-photoperiod-induced floral reversions that can be produced in plants heterozygous for strong mutant alleles at some of the genes involved in the process of floral initiation[28] (see later).

The process of floral initiation and its sensitivity to environmental conditions

The direct consequence of the floral transition in *Arabidopsis* is the activation of the floral program in the lateral primordia generated at the shoot apical meristem. Genetic analyses have led to the identification of at least five loci that regulate the process of flower initiation and development: *LEAFY* (*LFY*),[12,29-31] *APETALA1* (*AP1*),[32-34] *CAULIFLOWER* (*CAL*),[34,35] *APETALA2* (*AP2*)[36,37] and *UNUSUAL FLORAL ORGANS* (*UFO*).[38-40] Mutations in any of these

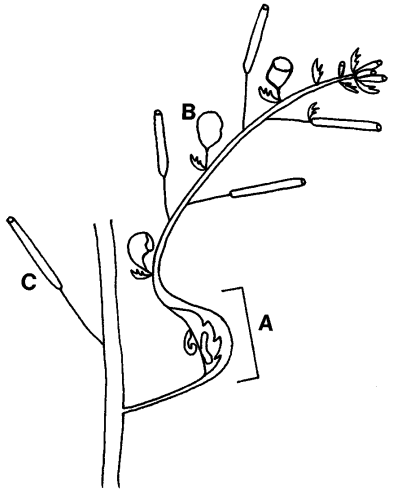

Figure 2. Schematic representation of abnormalities found occasionally instead of the most basal flowers in late-flowering mutants grown under short photoperiods. A, Broken carpel that gives rise to an abnormal secondary inflorescence; B, abnormal silique; C, normal silique.

loci interfere with the process of floral meristem specification, and result in the generation of inflorescences or modified flower structures, with inflorescence characteristics, in places where flowers should develop. The results of the genetic analyses and the molecular information already available for these genes have been reviewed in several recent articles.[4,7,9,28,41] Here, we will mainly refer to the results concerning the two loci, *LFY* and *AP1*, that have a more relevant role in the Floral Initiation Process or FLIP (as named by Schultz and Haughn, ref 13). In plants homozygous for strong *lfy* alleles, secondary inflorescences, subtended by leaves, are developed in place of the basal flowers of the wild-type inflorescence (Figure 3). In more apical positions of the inflorescence, these mutants contain flower-like structures, also subtended by leaves, whose organs are leafy, sepaloid or carpeloid structures that emerge in a spiral phyllotaxy (an inflorescence character).[12,13,29,30] Mutations at the *AP1* locus produce secondary inflorescences, that are not subtended by leaves, instead of the basal flowers of the inflorescence. In most apical positions *ap1* mutants show flower-like structures that are rather different than those found in *lfy* mutants. These structures exhibit whorled phyllotaxy as normal flowers although, due to the incomplete inhibition of axillary meristem formation, develop ectopic secondary flowers in the axils of first-whorl floral organs.[13,32,33]

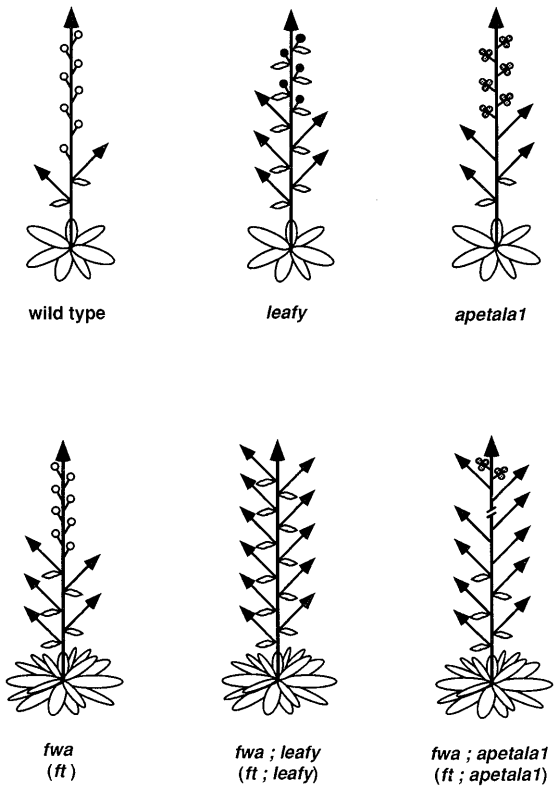

Figure 3. Schematic representation of rosette and inflorescence morphology of different Arabidopsis genotypes carrying mutations affecting floral initiation, flowering time or both. For simplicity, arrowheads represent meristems, open circles represent regular flowers and filled circles and clover-like symbols represent the abnormal flowers found in floral initiation mutants.

Double mutants of *lfy* and *ap1*, independent of the strength of the mutant alleles combined, show greatly enhanced flower-to-inflorescence transformations in which flower-like structures are only rarely observed.[13,29,34] This suggests that the products of these two genes reinforce each others activity to trigger the process of flower development at the axillary meristems.[29-31,34] Recent reports have shown that the constitutive overexpression of either *LFY* or *AP1* messages in transgenic plants produce a dramatic acceleration of the floral transition and the development of flowers in almost every available shoot meristem.[42,43] These results strongly support a crucial role of these two genes as switches of the initiation of flower development. The other mutations affecting FLIP have lesser phenotypic effects suggesting that the corresponding genes play secondary or partially redundant functions. In this way, the *CAL* locus is functionally redundant to the *AP1* locus and its mutations only produce a phenotype in *ap1* mutant backgrounds.[34,35] On the other hand, the effects of *ap2* or *ufo* mutations on FLIP are only evident when mutant plants are grown under short photoperiods or in double mutant combinations.[13,28,44]

Although mutations affecting FLIP delay the appearance of flower-like structures, and some of them almost completely prevent their development, they do not affect the time of bolting. Therefore, in contrast to the phenotype of late flowering mutants, FLIP mutants produce the same number of leaves in the rosette and in the inflorescence as the wild type.[13] Lfy mutants constitute a dramatic exception; these mutants produce an almost unlimited number of inflorescence leaves because this gene is required to inhibit leaf initiation in lateral primordia.[29] A feature common to all the FLIP mutants is that the canalization of flower development, which is responsible for the production of identical flowers all along the inflorescence development under any environmental condition, is broken. Consequently, the nature of the lateral structures produced by the inflorescences of FLIP mutants varies along their development, uncov-

ering the effects of developmental and environmental signals responsible for the induction of the FLIP itself. In this way, the floral character of the lateral structures produced by FLIP mutants increases acropetally, in a gradual manner.[12,29,30,44] Furthermore, the rate of change is very dependent on environmental variables, being slowed by conditions, such as short photoperiods or low growing temperature (e.g. 15°C), which produce a delay of the floral transition in wild-type plants.[13,34,44]

The acropetal increase of the floral character shown by lateral inflorescence structures in FLIP mutants is reminiscent of the gradual change of other morphological features, such as leaf size and shape, that is observed during the vegetative development of *Arabidopsis*.[10,11] In a similar way, the rate of this change is also dependent on the same environmental variables. Based on these observations, Schultz and Haughn have postulated the existence of a common mechanism, named as Controller of Phase Switch or COPS,[13] that would control all the phase transitions taking place during *Arabidopsis* development. Following the COPS hypothesis, the gradual morphological variation would be the result of a gradual change in the level of a factor(s) along with the development of the plant. When certain critical levels of the factor(s) are reached, specific phase transitions would be triggered at the shoot apical meristem. The level of the factor(s) would be increased or decreased by environmental conditions, and by mutations that affect flowering time, which is supported by the phase transition delays produced by both short photoperiods and late flowering mutations (ref 11, Martínez Zapater *et al*, manuscript in preparation). This raises many questions regarding the function of the loci that control flowering time and their interaction with FLIP genes. The construction and characterization of double mutants that are late flowering and altered in some of the FLIP genes should give some answers to these questions, as we summarize in the following section.

Genetic interactions between genes controlling flowering time and FLIP genes

Several laboratories are currently involved in the construction and characterization of *Arabidopsis* double mutant plants carrying mutations at genes controlling flowering time (hereafter called late loci) and FLIP genes. The results, although few, have already helped to outline a preliminary view of the genetic interactions between these genes. The results of experiments recently performed in our laboratory using complete loss-of-function alleles at loci *AP1* and *LFY*,[31] and late flowering mutations at loci *FWA, FT, FVE* and *FPA*, point toward the existence of at least two different ways of interaction between FLIP and these late loci (Ruiz-García *et al*, manuscript in preparation). The most striking results concern the phenotypes of double mutants for *lfy* and either *fwa* or *ft*. Although these double mutants bolt at the same time as their late-flowering parents, they show an extreme inflorescence phenotype, even stronger than that of *lfy, ap1* double mutants,[13,29,34] being completely unable to produce any flower-like structure (Ruiz-García *et al*, manuscript in preparation) (Figure 3). A similar phenotype has also been independently observed for *fwa, lfy* double mutants (A. Chaudhury, manuscript in preparation) and for *ft, lfy* double mutants.[45] These results indicate that, in the absence of the LFY and the FWA or FT functions, the other late loci are not able to promote the floral transition. Moreover, they suggest that mutations at either *FT* or *FWA* fully prevent the function of the other FLIP loci. Wild-type alleles of these loci are likely responsible for the development of the carpeloid organs found in the apical flower-like structures of *lfy, ap1* double mutants.[31]

In contrast, double mutants *fwa, ap1* or *ft, ap1* show an extreme Ap1 phenotype that could be reminiscent of the phenotype of double *ap1, cal1* mutants grown under non inductive conditions (Figure 3). In fact, *fwa, ap1* and *ft, ap1* double mutants are still capable of producing some fertile Ap1-like flowers at the end of an extended growth period. This suggests that, although *fwa* or *ft* mutations could block the function of FLIP loci like, *AP1, CAL, AP2* or *UFO* as shown above, they would not completely block the function of *LFY*. A reduction in the LFY function cannot be discarded in these double mutants since double mutants of *ap1* and *cal1* seem to show a reduction in the level of LFY expression as seen by *in situ* hybridizations.[34] Thus, in the absence of FWA or FT function, other late loci could be responsible for the establishment of the LFY function in the lateral primordia derived from the shoot apical meristem.

The double mutant phenotypes produced by the combination of *lfy* or *ap1* null alleles with *fpa* or *fve* mutations are not so extreme and informative as those of the above described double mutants. Both *fve* or *fpa* mutations seem to enhance the inflorescence phenotype caused by either *ap1* or *lfy* mutations (Ruiz-García *et al*, manuscript in preparation) in a similar way as

short photoperiods.[13,34,44] Plants carrying mutations at the late flowering gene *CO* and a weak mutant allele (*lfy-5*) at the *LFY* locus also show a similar enhancement of the Lfy phenotype.[25] Thus, although mutations in these late loci seem to delay the establishment of FLIP genes function in the lateral primordia produced at the shoot apical meristem, they do not seem to be required for FLIP in such an strict way as *FWA* or *FT*. Whether the lack of stronger double mutant phenotypes is due to the existence of redundant pathways, or to the weak nature of the *fve* and *fpa* mutant alleles used, remains to be seen.

In summary, these results suggest the existence of at least two developmental pathways to promote flower initiation in the lateral primordia. The first one would be represented by loci, like *FT* or *FWA*, that are required for the activity of most FLIP genes except *LFY*. The second one would be represented by loci like *FVE*, *FPA*, and perhaps *CO*, that seem to play a more general promoting role both on the establishment of LFY function and, through a pathway requiring FWA and FT functions, on the other FLIP functions. Mutants *fwa* and *ft* also show a different phenotype compared to the other late-flowering mutants studied in their response to the availability of sucrose at the shoot apex. As mentioned above, this availability can rescue the wild-type phenotype in all the mutants except in *fwa* and *ft*. Whether the function of both loci is required at the lateral primordia for the floral transition process remains to be shown.

The general picture that emerges from the characterization of late flowering and FLIP mutant phenotypes, and the corresponding double mutants, is that *Arabidopsis* development is a highly integrated process in which environmental conditions and specific mutations can alter the pattern of development all along the life cycle of the plant. Furthermore, if *Arabidopsis* development is an integrated process controlled by a common mechanism like COPS[7,13] there is no real need to postulate the existence of a specific floral hormone to explain the promotion of the floral transition. In fact, the same signals might be responsible for all the developmental phase transitions observed in *Arabidopsis*. It is tempting to speculate that some of the molecules responsible for the control of these transitions could be growth regulators, or other molecules, that would inform the shoot apical meristem of the nutritional and developmental status of the plant. Plant hormones like gibberellins and cytokinins, and metabolites like sucrose, have been repeatedly proposed and rejected as regulators of the floral transition process in different plant species,[2] and they certainly have an effect on the development of *Arabidopsis*.[6,14,46]

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