How plants pattern flowers: Lessons from molecular genetic studies of flowering in *Arabidopsis thaliana* a model plant

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Understanding mechanisms that regulate when, where and how flowers are formed would elucidate cell-fate determination in plants. Some of the advances made towards deciphering genes controlling floral induction, meristem specification and floral organ patterning are reviewed here. Studies beginning in the early 1980s of mutations in *Arabidopsis thaliana* or *Antirrhinum majus* that alter either floral induction or floral meristem fate or floral organ fate were start points for these analyses. Cloning and functional characterization of the corresponding genes has illustrated how transcription factors of the MADS box gene-family or factors involved in cell–cell signalling regulate cell fate. These studies reveal evolutionarily conserved elements in the flower development pathway.

General background

Developmental biology pertains to the process by which genes/gene products in the fertilized egg control cell fate in the embryo, so as to determine organismal form and behaviour. Several questions central to developmental biology are: How do cells arising from divisions of the fertilized egg become different from each other? How are these cells programmed to generate complex organs with many differentiated cell types? Much of the excitement in this field comes from our expanding understanding of how genes control these developmental processes in model experimental systems, and also from discoveries of the general principles of development. However, until about a decade ago much of the knowledge on the genetic control of development came from studies on model animal systems like *Drosophila melanogaster*, *Caenorhabditis elegans*, *Xenopus laevis*, *Mus musculus*, etc. Plant development differs in a few crucial ways from animal development; the most obvious one is the absence of cell migration due to the rigid structure of the plant cell wall. Therefore major changes in shape cannot be achieved by movement or invagination of sheets of cells, a common mechanism in animal development. Also programmed cell death does not contribute in a major way to plant development, though it is involved in a few specific events. Thus plant development occurs largely by altered rates and planes of cell division and by cell enlargement. A second significant difference is that post-embryonic development in higher plants gives rise to all of the adult structures of the plant through the activity of specialized groups of cells called meristems. This is much unlike animal embryogenesis, where organogenesis is near complete and post-embryonic development is very limited. These plant meristem cells divide repeatedly to replenish themselves and to generate initials or meristems for all plant tissues: shoots, stems, leaves and roots. Organogenesis is therefore a continuous process in plant development.

In flowering plants, the fertilized egg undergoes a stereotypic pattern of cell divisions to form an embryo where an apical–basal axis and a radial pattern are defined. In the embryo, the shoot apical meristem and the root meristem form the polar axis and from these meristems originate all adult structures of the plant. The radial axis in the embryo establishes the outer epidermis, the presumptive vascular tissue in the centre and the presumptive cortex that lies between these tissues. Another important difference between plant and animal development is the ability of a single somatic cell from an adult plant to generate a new fertile plant. Thus some differentiated plant cells retain totipotency, a feature recognized in higher plants for several decades. Yet, how cell fate is determined in plants is still largely unknown, and remains a fascinating avenue of research. Since most structures in plants result from controlled cell division, a reasonable assumption is that cell fate could be dictated by its lineage. However, it is known that a plant cell's fate can be altered by its position in the meristem, suggesting that cues from neighbouring cells dictate fate. The best illustrated case is the study of the effects of cell ablations in the root, where laser ablation of a single cell resulted in neighbouring cell occupying the space and assuming the fate of the dead cell. In summary, the control over the rate and direction of cell division combined with the capability of cells to

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sense positional information is crucial for the generation of the body plan in plants[1,2].

Arabidopsis thaliana, an experimental model for plant development

Experimental analysis of genetic control of development in higher plants has benefited greatly from the use of the crucifer weed *Arabidopsis thaliana*, a member of the Brassicaceae family, as a laboratory model system. This species is a representative angiosperm, the most abundant plant group today. This diverse group of modern plants with about 250 million species has evolved in about 200 million years. These plants generate complex reproductive structures: flowers, whose diverse forms are important criterion in taxonomic classification; yet, in each species flower formation and floral structure is noticeably consistent in pattern. *Arabidopsis,* a temperate weed had been sporadically used for genetic experiments. The early 1980s were a turning point in its experimental use, which began with the realization of its small genome; so far the smallest amongst all plants[3,4]. This feature coupled with the emergence of molecular genetic tools to study plant growth and development made *Arabidopsis* the obvious choice for complete genome sequence and also for functional genomics[5,6]. Some of these molecular tools were the generation of high density integrated genetic maps with morphological and molecular markers, development of facile procedures for germline transformation of *Arabidopsis* and the adaptation of maize Ac-Ds transposable elements to tag genes based on their expression patterns or phenotypes. These methodologies are revealing gene hierarchies and functions in diverse cellular pathways, for example, plant defence, environmental response; hormonal response, flowering, root development to name a few. In this review are summarized the current understanding of genetic networks that control flower development, specifically floral meristem formation and floral organ patterning, largely from the studies on this model plant. These studies are paralleled by those done in *Antirrhinum*, a second model experimental plant for the flower development pathway. In the last decade, the study of mutants defective in floral development pioneered in the laboratories of Elliot Meyerowitz, Maarteen Koorneef, Enrico Coen and Heinz Saedler has uncovered a complex network of regulatory factors in both model plant species that direct flower formation and pattern therein.

In *Arabidopsis*, upon seed germination the shoot apical meristem (SAM) produces on its flanks primordia/meristems for leaves. The leaves are generated in a spiral fashion and are separated by short lengths of stem (internode) (Figure 1). Upon floral induction SAM reorganizes to form an inflorescence meristem that first produces a few spirally placed leaf primordia with axillary second-order inflorescence meristems. These leaves are called cauline leaves and will be separated by long internodes. After this the inflorescence meristem produces meristems for individual flowers, again in a spiral fashion. The floral meristem is determinate in its development, unlike indeterminate inflorescence meristem. Each floral meristem specifies formation of concentric rings (whorls) of floral organ primordia in an invariant order: sepals, petals, stamens and carpels from the periphery to the centre of the flower. The result is a plant with a basal rosette of leaves and racemose inflorescence, where individual flowers bear organs in the pattern (sepals)4, (petals)4, (stamens)6, and (carpels)2.

Floral induction: Mutations and genes controlling flowering time

The switch in apical meristem from a vegetative to an inflorescence meristem depends on critical cues, both environmental and genetic. Day length is by far the most important environmental determinant for flowering in most species. Long days promote flowering in *Arabidopsis*, while short days result in delayed flowering. Mutations in *Arabidopsis* that cause precocious or delayed transition to flowering[7–9] have provided starting points for genetic analysis of this complex step. Mutations at about twenty loci have been reported to specifically alter flowering time. Not surprisingly, many of them define components of general signal-transduction cascades required for light perception or plant hormone signalling. These *Arabidopsis* mutants reveal three partially redundant pathways involved in floral induction, of which photoperiod sensing is one. Grafting experiments in other plant species done several decades ago have suggested that the photoperiod perception occurs in leaves and results in long-range change in the shoot apical meristem[10]. Phytochromes the receptors for far-red/red light are involved, since over-expression of some of the members of this class leads to early flowering and independence from day length regimes[11,12]. In addition, blue light photoreceptors play a role since mutations in *CRY2* that encodes the blue-light photoreceptor result in delayed flowering[13,14]. The photoperiod pathway also includes an inhibitory element, encoded by the PHYTOCHROME B (*PHYB*) gene, whose inactivation causes constitutive early flowering, with greater effects in short days. The mechanism by which photoperiod signal is transduced to the shoot meristem is poorly understood. Complex interactions between phytochrome, day length and flowering time genes have been proposed based on analysis of flowering time in plants with single, double or triple mutations in these genes[7,9] (Figure 2). Some of these genes have been characterized molecularly. One such flowering time gene, *CO,*

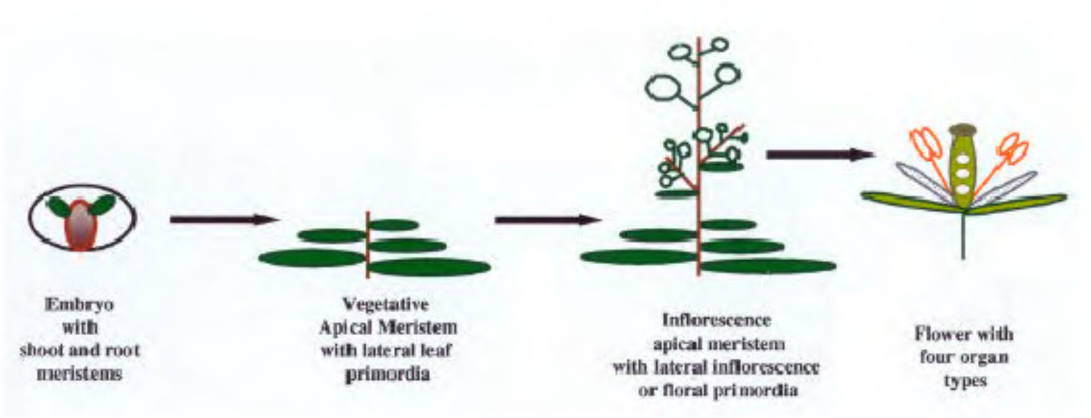

Figure 1. Different stages in the life of an *Arabidopsis* plant, particularly with regard to the shoot meristematic cells and the lateral organs/structures produced thereof. Four organ types in individual flowers borne on the racemose inflorescence are also shown.

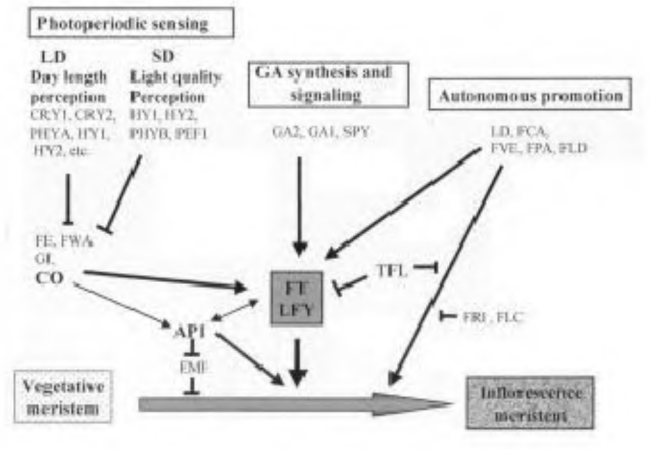

Figure 2. Interactions between components of three different signalling pathways that control the transition of the *Arabidopsis* vegetative shoot apical meristem to an inflorescence apical meristem. Some of the genetically defined regulatory factors are shown, as are their interactions (→, positive interactions; , negative interactions).

mediates flowering in response to inductive photoperiods, mutations here delay flowering in long days and the gene is transcriptionally upregulated in response to inductive photoperiod[15]. *CO* levels are apparently critical for transition of the apical meristem, since ectopic high levels of *CO* expression can induce flowering in otherwise inappropriate regimes. The predicted CO protein is a Zn-finger-containing transcription factor, and is an upstream regulator of genes that specify floral meristem fate, i.e. *LFY*[16]. However, the number of steps between activation of *CO* and change in meristem fate remains unknown.

The second pathway that induces flowering is dependent on the plant hormone gibberellin (GA); mutants defective in synthesis or perception of GA are delayed for flowering[17]. Further, exogenous application of GA promotes flowering in unfavorable conditions. One mechanism of GA action is activation of *LFY,* a key gene that confers floral identity to incipient meristems[18]. The last pathway is the autonomous pathway for flowering, components of which have been identified as mutants with delayed flowering that are not dependent on photoperiod. *FCA*, *FVE* and *LD* are some of the positive regulators in this pathway, while *FRI* and *FLC* are some negative regulators. *FCA* encodes an RNA-binding protein suggesting that it might regulate gene expression post-transcriptionally[19]. The non-autonomous mode of action of *FCA* suggests that it could regulate the production of a diffusible substance[20]. *LD* also codes for a regulatory factor, the predicted LD protein has homeo-domain and glutamine-rich stretches, attributes of transcription activators[21]. The *FRI* locus was inferred to be a negative regulator of the autonomous pathway, from crosses between naturally occurring late flowering ecotypes and the common laboratory ecotypes that are early flowering. Late flowering was correlated to the presence of dominant *FRI* allele[9]. *FRI* was shown to repress the same pathway that *FCA* and *LD* depend on, i.e. the autonomous pathway.

Convergence of multiple pathways leading to floral induction is exemplified by the behaviour of the late flowering mutant, *ft*, which is regulated by both the photoperiod-dependent and independent pathways[7]. *FT* is at least partially downstream of *CO*, because *FT* transcripts are significantly delayed in *co* mutants. Also ectopic expression of *FT* rescues flowering defects of *co* mutants. However, unlike *CO, FT* is not normally a positive regulator of the floral meristem identity gene *LFY*. A suggested mode of action is that *FT*, a flowering time gene, acts in parallel to the *CO–LFY* pathway to establish floral identity for meristems. The biochemical

mode of action of FT is unclear, but a suggested function is in signalling because of its sequence relatedness to mammalian proteins that bind hydrophobic ligands[22, 23]. An exciting hint comes from studies on a human protein, that is cleaved to generate a stimulatory peptide HNCP; future experiments should reveal if FT bears a similar function. Interestingly, one of the plant proteins with sequence similarity to FT is TFL1, mutations in which lead to terminal flowers, i.e. early flowering. Like FT, CO also regulates *TFL1*. The behaviour of plants that ectopically express *TFL* and or *FT* suggests that while these proteins have opposing effects on flowering the balance between their activities underlies responses to inductive conditions.

In summary, while some of the factors that contribute to floral induction are being characterized, the signalling factor or factors crucial for this transition in behaviour of the apical meristem are still largely unknown. It is likely that integration of several inputs from each one of the inductive pathways is needed before a transition to an inflorescence meristem occurs.

Specification of floral meristem identity

Floral induction in *Arabidopsis* is characterized by a change in the identity of meristems being allocated on the flanks of the shoot apex. The early arising meristems, after induction, are specified as leaves with axillary inflorescence meristems, the later arising meristems develop as individual floral primordia. The characterization of *Arabidopsis* and *Antirrhinum* loss-of-function mutants that perturb the fate of these meristems has led to the identification of several genes that control floral meristem identity. In *Arabidopsis*, *LEAFY* (*LFY*), *APETALA1* (*AP1*), *APETALA2* (*AP2*) and *CAULIFLOWER* (*CAL*) are genes required for this step. The meristem defects in *lfy* loss-of-function mutants include an increase in number of leaves with secondary inflorescences and also the formation of abnormal shoot-like flowers in the place of solitary flowers[24,25]. The *ap1* mutants produce shoots at the first few positions of the inflorescence axis that are normally occupied by flowers. Later they produce branched flowers in place of solitary flowers[26,27]. Because plants with null alleles of *ap1* still produce functional reproductive floral organs, these mutants are only partially defective in conferring floral meristem identity. The phenotypes of *lfy* and *ap1* single mutants together with the more severe non-flowering phenotype of the double mutants demonstrate partial redundancy of gene function. The *Arabidopsis cal* loss-of-function mutation has no phenotype of its own, but it enhances *ap1* phenotype, producing a large number of branching meristems[27]. *LFY* and *AP1* are sufficient to specify floral fate after induction, because constitutive expression of *LFY* or *AP1* results in formation of flowers in the place of leaves or second-order inflorescence meristems[28,29]. The *Arabidopsis ap2* mutations also enhance flower meristem defects of *ap1* and *lfy* mutants, indicating that *AP2* also contributes floral meristem identity[26,27,30,31].

Several of these *Arabidopsis* genes have counterparts in the other experimental model plant *Antirrhinum*. *FLORICULA (FLO)* and *SQUAMOSA (SQUA)* of *Antirrhinum* are sequence homologues of *LFY* and *AP1*. However the relative contribution with regard to determining floral fate varies slightly between these two species. *FLO* and *LFY* are probably orthologous genes; they share 70% identity in amino-acid sequence, and are unique to the plant kingdom with no sequence homologues as yet among all currently known animal and microbial genes. Additionally, both predicted proteins have biochemical characteristics of transcriptional regulators: proline-rich domains and acidic stretches[25,32]. High level LFY expression occurs on the flanks of the inflorescence apical meristem even before flower meristems are morphologically visible. This expression marks cells on the flanks of the apical meristem as incipient floral primordia. This upregulation of *LFY* transcripts occurs concomitant with floral induction, prior to which only very low levels of *LFY* are expressed in newly arising lateral meristems that form leaves or shoots[33]. Cloning and analysis of the expression patterns of *LFY* sequence homologues from diverse dicot species, a monocot and a gymnosperm reveal an evolutionarily conserved function in specifying reproductive fate to meristems[34–38]. However, additional species-specific functions are also implied since loss-of-function mutations in *UNIFOLIATA*, a pea *LFY* homologue, affect leaf development in addition to causing defects in floral meristems[36].

AP1 and *SQUA* genes are likely sequence homologues and they encode putative transcription factors that are members of MADS-domain containing gene family[39,40]. The *CAL* gene in *Arabidopsis* is a related redundant gene[41]. Consistent with its role in floral fate, *AP1* transcripts are found in early floral meristems and their appearance occurs later than that of *LFY* transcripts. Several additional lines of evidence place *AP1* downstream of *LFY*. Notably, *AP1* expression is delayed and reduced in *lfy* mutants; also, constitutive *LFY* expression results in earlier than normal activation of *AP1*. Very recently, these suggestions on the hierarchial relationship between *LFY* and *AP1* were proven by the following experiments: *AP1* was shown to be a direct *in vivo* transcription activation target of *LFY*; additionally, LFY protein was shown to bind sequence elements in the promoter of *AP1* (refs 42, 43).

In both *Arabidopsis* and *Antirrhinum* inflorescence meristems continue to be maintained apically, while flower meristems are initiated only from the flanks of inflorescence meristems. Consequently, these plants

normally produce lateral flowers and not terminal flowers. But mutations in the homologous genes *CENTRORADIALIS* (*CEN*) of *Antirrhinum* and *TERMINAL FLOWER 1* (*TFL1*) of *Arabidopsis* result in terminal flowers, i.e. the inflorescence meristem is now specified as a floral meristem[44–46]. In both mutants, some flowers are formed by the inflorescence before the generation of the terminal flower. *TFL1* and *LFY* seem to be antagonists because the *tfl1* phenotype is similar to *lfy* and *ap1* gain-of-function mutants. As mentioned earlier, the TFL1 and CEN proteins have homology to *FT* and mammalian phosphatidyl ethanolamine-binding proteins. Both *CEN* and *TFL1* are expressed just below the domes of the inflorescence meristem. Expression of these genes seems to maintain indeterminate growth by inhibiting expression of floral meristem identity genes in the dome of inflorescence meristem.

Patterning of floral organs: The ABC model

Several *Arabidopsis* homeotic genes have been identified that control the pattern of four floral organs on the newly established floral meristem. The genes fall into three classes, each of which is necessary for floral organ specification in two adjacent whorls (Figure 3). Mutations in *ap1* and *ap2* loci affect the organ identity in the outer two whorls. The *ap1* null mutants have sepals converted to bracts, but no petal formation occurs at all[26,27]. In plants with severe *ap2* alleles, the first and second whorl organs, if present, are converted from sepals and petals to carpel and stamens, respectively[47,48]. Both *AP3* and *PI* genes are required for correct development of the second and third whorl organ types[48]. In *ap3* and *pi* mutants, sepals replace petals and carpels replace stamens. The *AG* gene is required for determining the identities of stamens and carpels[48,49]. In *ag* mutant flowers, the six stamens are converted to petals and carpel is replaced by a second *ag* flower such that the pattern of sepals, petals and petals are repeated more than five times. This phenotype indicates that *AG* function is also required to generate a determinate floral meristem. The number of organs is altered in *ap2* and *ag* mutants indicating that these genes also function in regulating organ primordia initiation.

Mutations affecting organ identity have also been isolated in other plants, a large number being in *Antirrhinum*. The major contributions here have come from the laboratories of Coen and Carpenter in England and that of Saedler in Germany. These studies capitalized on the transposon-induced mutations that alter floral meristem, or floral organ. Such transposon-induced mutations provided an easy start point to cloning the gene and thereafter its functional characterization. Many of these *Antirrhinum* homeotic conversions are similar to those found in *Arabidopsis* mutants. The *deficiens* (*def A*) and *globosa* (*glo*) mutants of *Antirrhinum* have phenotypes

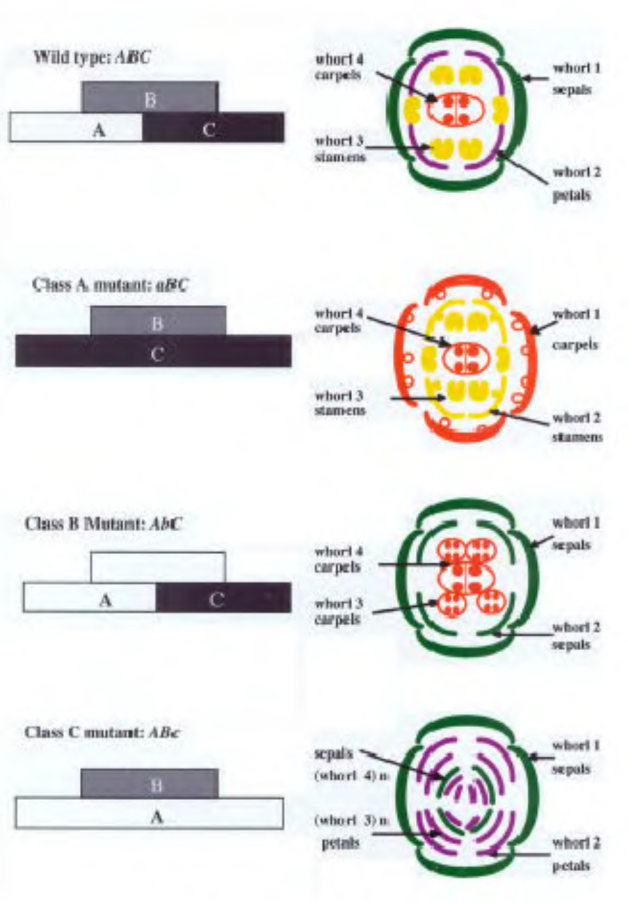

Figure 3. Phenotypes of loss-of-function homeotic mutations that affect organ identity in flowers. The ABC model for specifying floral organ identity based on mutant phenotypes and from genetic and molecular studies on interactions between these regulators is also shown. The model explains how the combined and individual expression of three classes of regulators A, B and C specifies four organ types.

similar to the *ap3* and *pi* mutants in *Arabidopsis*. Similarly, mutants of another *Antirrhinum* gene *plena* (*ple*) show conversion of stamens to petals and carpels to additional floral organs as in *ag* mutant[50,51]. Yet another system used to dissect floral patterning is petunia, wherein mutants with similar phenotypes have been studied. The petunia, *blind* mutant produces flowers with sepals exhibiting carpel features, together with conversion of petals to stamenoid organs[52], similar to the flowers of weak *ap2* mutants. Another petunia floral mutant, green petal (*gp*), has flowers with the petals converted to sepals[53]. Model plant species for the grasses have usually been maize or rice. The phenotype of the *silky* mutant of maize is similar to *ap3* or *pi* mutants of *Arabidopsis*[54], hinting at similar control mechanisms in grasses. These kinds of observations together with the fact that these floral organs occur in an invariant order in almost all extant flowering plants indicate an evolutionarily conserved ground plan that dictates floral organ patterning.

Genetic analysis of such loss-of-function floral homeotic mutants in *Arabidopsis* and in *Antirrhinum* forms the basis of the ABC model for floral organ patterning that was first proposed by Coen and Meyerowitz[49,55]. This model proposes that floral organ identity is regulated by three classes of master genes, A, B and C (Figure 3). These genes act in overlapping domains, each of which extends over two adjacent whorls[56–59]. The model suggests that the A and C functions specify sepals and carpels, respectively, whereas the combined activities of A and B, and B and C specify petals and stamens, respectively. The model further suggests that the A and C activities are mutually antagonistic, such that in Class A loss-of-function mutants, the C domain expands to include all whorls, the converse occurring in Class C loss-of-function mutants. All but one of these genes encode putative transcription factors that contain an ancient evolutionarily conserved DNA-binding domain called the MADS box. The proposed domains of A, B and C gene action were supported by the observed expression patterns for several of the cloned homeotic genes in wild type flowers as well as flowers mutant for one A, B or C function; this being the case for the *Arabidopsis AP1*, *AG*, *PI*, *AP3* and as well the *Antirrhinum SQUA*, *DEFA*, *GLO* and *PLE* genes. Therefore, organ identity is controlled to a great extent at the level of transcription of these regulators in specific domains of the floral meristem.

Both *AP1* and *AP2* are examples of genes with class A activity; in the absence of either gene, sepals and petals fail to develop properly. Although early *AP1* RNA expression occurs throughout the young flower primordia, it later becomes localized to sepals and petals, consistent with its role in determining the identity of these two organs[40] (Figure 4). The absence of *AP1* RNA from stamens and carpels arises from negative regulation by the C-function gene *AG*[60]. Thus the transcriptional regulation of *AP1* is responsible for its domain of expression and therefore its Class A activity. In contrast, while *AP2* function in specifying organ identity is restricted to the two outer whorls, *AP2* RNA is present in all four whorls of the flower as well as in the inflorescence meristem and the vegetative meristem[61]. Thus, post-transcriptional regulation of *AP2* is suggested for its Class A activity. Also, unlike other floral organ identity genes (*AP1*, *AP3*, *PI* and *AG*) the predicted AP2 protein does not contain a MADS box. It belongs to a different class of transcription factors since it bears domains with similarity to a class of DNA-binding proteins such as the ethylene response element binding (EREB) proteins[61].

The Class B floral organ identity genes are necessary for the proper development of the petals and stamens, normally found in the second and third whorls of the flower. *AP3* and *PI* are two Class B genes necessary and sufficient for B function activity[62–64]. Both are MADS-box gene products and mutations in either *AP3* or *PI*

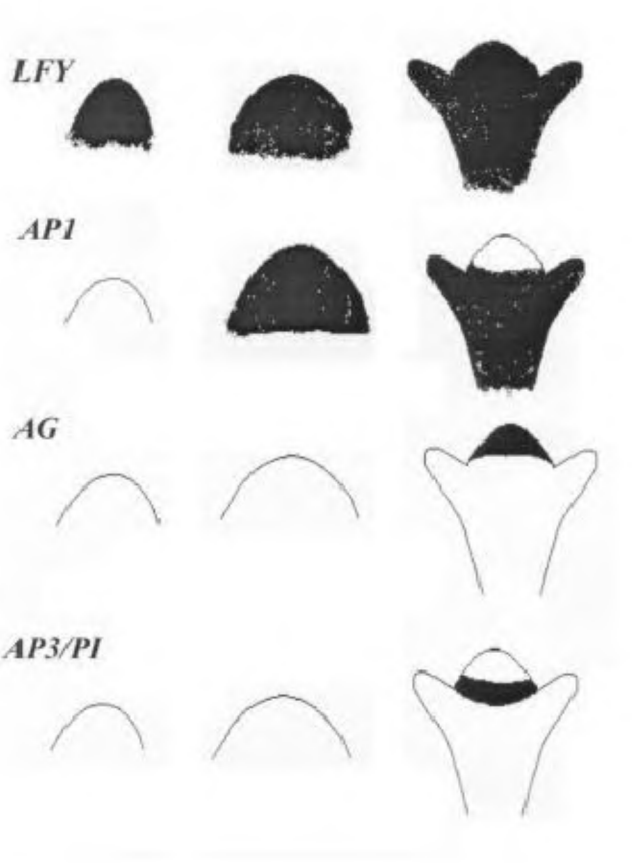

Figure 4. Domains of expression of *Arabidopsis* floral meristem identity gene *LFY* and organ identity genes during early stages of flower morphogenesis. (left column) Very early floral primordia; (Center column), Primordia at slightly later stages of development, yet before any organ primordia are discernible; (Right column), Flowers where sepal primordia only have been initiated. Floral meristem identity gene *LFY* is expressed throughout the flower primordia in all these stages. *AP1* is first expressed in the young floral meristem after *LFY* expression. *AP1* is later restricted to sepal and petal primordia. *AG* expression is restricted to the stamen and carpel primordia. *AP3/PI* expression is confined to petal and stamen primordia. The figure also shows that floral organ identity genes express only after floral meristem is specified.

result in homeotic transformation of petals to sepals, and of stamens to carpels. Additionally, *AP3* and *PI* RNAs accumulate in the second and third whorls of flower meristems at the stage of sepal formation (Figure 4). Their continued expression is maintained by autoregulation[63,65,66]. Furthermore, B function activity requires the formation of AP3/PI heterodimers, which are then nuclear localized[67–69].

In *Arabidopsis* the *AG* gene is necessary and sufficient for development of stamens and carpels. This gene too, codes for a MADS box-containing factor and the expression of its RNA in only the third and fourth whorl primordia suggests transcriptional regulation[70]. In addition, *AG* activity is responsible for the exclusion of *AP1* RNA from the two inner whorls[60]. *AG* is also required for the determinacy of the flower primordium, since *ag*

mutant flowers are indeterminate and continue to add new whorls of organs, giving rise to the 'flower within a flower' phenotype. An important prediction of this model was the antagonistic nature of A and C functions. This antagonism is supported by the observation that the expression of *AG* expands outward to all floral whorls in the *Arabidopsis ap2* mutant flowers[71]. In fact, ectopic expression of C type genes in a variety of species leads to the formation of carpeloid structures in the first whorl and stamenoid structures in the second whorl as can be predicted by the ABC model[52,72–74]. Thus, the ABC model appears to be applicable to many flowering plants. Though simple and attractive, the ABC model for organ identity is constantly being revised to account for emerging observations of gene interactions.

Studies with mutants affecting floral organ numbers are providing clues to controls on cell proliferation in meristems and within floral whorls. *Arabidopsis SUPERMAN* (*sup*) mutant flowers are normal in the outer three whorls: sepals, petals and stamens, but produce additional whorl(s) of stamens at the expense of the carpel whorl[49]. Based on the expression pattern of *SUP* in wild-type flowers and B loss-of-function mutants, a proposed model for *SUP* action is that it controls cell proliferation differentially in the third and fourth whorls[49,75,76]. In contrast to the flower-specific effects of *sup*, the *Arabidopsis clv* mutants have enlarged shoot and floral meristem resulting in extra floral organs[77,78]. Cloning of two members of this group, *CLV1* and *CLV3*, indicates that they may be involved in cell–cell signalling to control proliferation[2,79,80]. The *Arabidopsis PAN* gene defines yet another class of regulators that control organ numbers, *pan* loss-of-function mutants have altered number of petals without any change in petal size or whorl boundaries[81]. The *PAN* gene encodes a transcription factor of the bZIP class, and is a member of a larger gene family[82]. The role of the *PAN*-like genes is still unknown. Much less is known about individual organ differentiation and of cell type specification within each organ, and this includes analysis of genes that produce asymmetric flowers. The only genes identified in the latter class are the *CYC* and *DICH* genes that are required for asymmetric development of petals and stamens in the zygomorphic *Antirrhinum* flower[83].

Modular organization of MADS protein

The coding regions of most of the floral regulatory genes share nucleotide and amino acid sequence similarities with the DNA binding and dimerization domains of two previously identified transcription factors: Mini Chromosome Maintenance gene product of yeast (MCM1)[84] and the mammalian Serum Response Factor (SRF)[85]. This region of homology has been termed MADS box that stands for *MCM1*, *AG*, *DEFA*, *SRF* genes that are important developmental regulators found first in animals and yeast and subsequently in plants[57,58] (Figure 5). The plant MADS domain-containing proteins have additional regions with moderate sequence similarity. One such is the K domain, with predicted structural similarity to the intermediate filament protein keratin, which has the ability to form amphipathic α-helices. A predicted role for the K domain is to mediate protein–protein interactions[58,86]. In addition, plant MADS domain proteins have two divergent regions, the I region (for Inter domain) that lies between the MADS and K domains, and the C region that contributes the most C-terminal portion of the protein.

Several of these plant proteins have been shown to bind DNA in a sequence specific manner; the consensus elements being related[67,87–89]. Nevertheless, there are some differences in the consensus-binding sequences, and different proteins may have different affinities for the same DNA sequences[87,88,90]. While minor differences in DNA-binding properties of different MADS box proteins are known, these features do not reflect the functional differences of the proteins. Studies with chimeric proteins indicate that the region outside the MADS domain, namely the I and K regions, are important for the *in vivo* specificity of these proteins[58,91,58].

While the DNA-binding properties of these ABC genes have been investigated to some extent, the targets of these genes in defining organ identity are less well established. So far, one direct target gene that is upregulated in response to AP3 has been identified as the *NAM* gene. This gene is regulated by AP3/PI heterodimer binding to sequences in its intron[92]. Similar studies are likely to shed light on the mechanism of action of these DNA-binding regulators.

Temporal and spatial regulation of the organ identity genes

The expression of the organ identity genes occurs in a distinct pattern of overlapping domains. Genes that establish the initial domain of activity of these floral organ identity genes and those that maintain this pattern have been studied to some extent. Current data suggest complex regulation at several different levels. The activity of the meristem identity gene, *LFY*, is central to the initiation of *AP1*, *AP3*, *PI* and *AG* transcription. By generating activated and/or inducible versions of *LFY*, the transactivation function has been demonstrated. This function is different from its role in specifying floral fate and the data show that different mechanisms are used for activation of ABC genes[22,42,43]. Because the early expression of *LFY* in the floral primordium is uniform, the eventual regions of activation of the ABC genes have been a question of interest. A plausible mechanism is the involvement of co-factors (co-

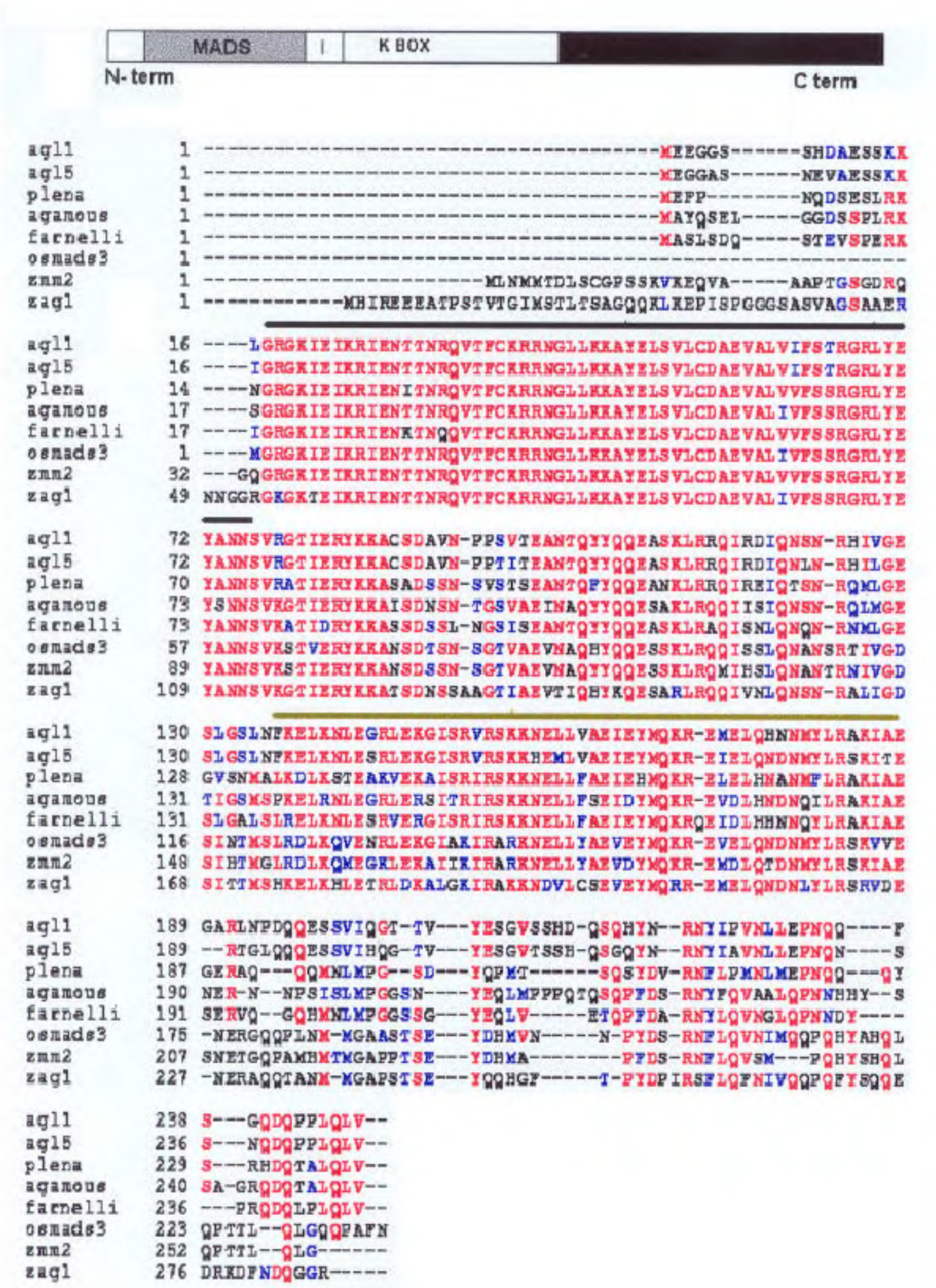

Figure 5. Modular organization of floral MADS box-containing transcription factors. Schematic representation of the general organization of MADS box factors, with the DNA binding MADS box, the K-box with potential to form amphipathic α-helices, and the gene specific Intermediate (I), and C terminal regions. Aligned in ClustalW (1.8), are a few C-function factors, including the *Arabidopsis AG*, the *Antirrhinum PLENA*, the maize *ZAG1, ZMM2* and rice *OsMADS3*. A few other sequences with similarity to the above factors are also shown. The amino acid residues in red are identical, those in blue are conservative substitutions and those in black are non-identical. The black bar above the text depicts the MADS box, whereas the light green bar depicts the K-box.

regulators) that confer region-specific activation of the ABC genes. The *UFO* gene product in *Arabidopsis* is suggested to function as a co-regulator with LFY for activation of B genes in the second and third whorls. Observations supporting the above suggestion are: *AP3* and *PI* expression requires *UFO* activity, as evidenced from studying their gene expression patterns in *ufo* loss-of-function and gain-of-function mutants[93,94].

Another mechanism of control occurs through the interactions among the organ identity genes themselves, the negative regulation between *AP1/AP2* and *AG*, discussed in an earlier section, being one example. The other two candidates which negatively regulate *AG* are *LEUNIG* (*LUG*) where mutations result in transformation of sepals to carpel[95] and *CURLY LEAF* (*CLF*), mutations here cause ectopic expression of *AG* and, to a lesser extent, *AP3*. The latter data indicate that the normal function of *CLF* is to stably repress *AG* transcription in leaves and stems and that it is required to maintain repression during later stages of flower development. *CLF* encodes a protein with similarity to the Polycomb group of genes that have a role in fate determination through chromatin reorganization[96].

Evidence for post-transcriptional regulation of *AP3* and *PI* for the sustained and restricted expression in the second and third whorl has been found. *AP3* and *PI* are both required for persistent, although, not their initial expression. In constitutively expressing *AP3* transgenic lines, *AP3* RNA can be detected in all four whorls, but the protein can be detected only in whorls two, three and four, implying post-transcriptional regulation[66]. In addition, constitutive expression of both *AP3* and *PI* from the CaMV 35S promoter leads to the first whorl sepals developing as petals, and stamens developing at the expense of the fourth whorl, carpels[64]. Epigenetic mechanisms for control of organ identity genes have also been uncovered. The regulators of ABC genes are subject to control by epigenetic mechanisms. Alleles at the *sup* locus that result from hypermethylation at the *SUP* locus result in reduced or no expression of SUP transcripts and thus a *sup* loss-of- function phenotype[97,98]. Chromatin methylation also influences expression of *AP3* and *AG* genes, since reduced genome-wide methylation results in alteration of the expression of these genes[99]. More recently, it has been found that hypermethylation at *AG* and thereby its silencing occurs in transgenic lines bearing antisense-versions of *MET*1 encoding the *Arabidopsis* methyltransferase gene[98].

In addition, there could be one or more plant growth regulators that could provide positional information, since alteration in the levels of growth regulators are known to affect many aspects of floral development[100]. Further, studies also show changes in the expression levels of the homeotic genes relative to changes in the concentration of the growth regulators. For example, differences in the levels of LFY protein and RNA activity were observed, in response to changes in GA concentration[18]. Also overproduction of cytokinin in the floral meristems of tobacco results in a marked decrease in the expression of the tobacco homologues of organ identity genes *DEFA*, *GLO* and *PLE*[101].

Genes involved in floral development in other species

Although *Arabidopsis* and *Antirrhinum* are distantly related dicotyledonous species, the regulatory mechanisms directing floral morphogenesis are generally very similar[50,55,102]. This apparent similarity suggests that a common floral development programme operates in all angiosperms, although differences may exist with respect to the genome organization and in expression pattern of these genes. Based on sequence similarity, putative meristem identity genes and organ identity genes have been cloned from several dicotyledonous and monocotyledonous species and even gymnosperm species. Class C organ identity genes have been cloned from *Brassica napus* – mustard (*BAG*)[103], *Lycopersicum esculentum* – tomato (*TAG1*)[104], *Nicotiana tabacum* – tobacco (*NAG1*)[74], *Petunia hybrida* – petunia (*pMADS*)[52], *Cucumis sativus* – cucumber (*CAG1* and *CAG3*)[105], *Zea mays* – maize (ZAG1 (ref. 106); *ZMM2* (ref. 107) and a gymnosperm *Picea mariana* – black spruce (*SAG1*)[108]. Further, in some instances, these *AG* homologues appear to be functional orthologues. Their ectopic expression brings about similar phenotypic effects in diverse species. For example, *BAG* expression in tobacco or *Arabidopsis*[40] and *CAG* expression in *Arabidopsis*[109] give similar phenotypes. Multiple *AG* homologues exist in cucumber, petunia and maize. These genes might reflect aspects of organ identity that are species-specific[107,109]. At least for the two maize genes, *ZAG1* and *ZMM2*, it is evident that they each have partial C function with distinct but non-overlapping activities[107,110]. Putative Class B organ identity genes have been cloned from petunia (*pMADS2, FBP1*)[111,112] and rice (*OsMADS2* (ref. 113); *OsMADS16* (ref. 114)) among other species. Also known are MADS box genes that control the fate of only one whorl, rather than the effects on two adjacent whorls as seen for the *Arabidopsis* genes. The *GREEN PETAL* (gp) of petunia is an example, where its function is restricted to only the second whorl, while its RNA is found in both second and third whorls of the flower[53]. Multiple genes represent Class A function even in *Arabidopsis*. Some candidates for Class A functions are known from maize (*ZAP1*)[115], rice (*OsMADS1*)[116] and other species. Together, all of these data suggest that in general the ABC model for floral organ specification is conserved in diverse flowering plants.

To some extent, regulators of ABC genes are also conserved through evolution. *LFY* homologues have been identified from several species. Also, the *LFY* function in conferring reproductive fate to meristems has been shown to operate in distantly-related plants. Some of the *LFY* homologues known are from pea (*unifoliata*)[35], rice (*RFL*)[34,117], petunia (*ALF*)[37], *Pinus radiata* (*NEEDLY*)[35], and also from a primitive group of angiosperms[38]. Strikingly, in many species *LFY* homologues are expressed to high levels in vegetative meristems and organ primordia. A role for this vegetative expression is clear only for the pea *LFY* homologue where it plays a role in leaf architecture, in addition to controlling floral meristem identity. A role in inflorescence branching is suggested for the rice *LFY* homologue, *OSL*, because of its high levels of expression in inflorescence branch primordia without any expression in the vegetative meristem[34,117]. Another *Arabidopsis* regulator of ABC genes, *SUP*, plays an evolutionarily conserved role in controlling cell proliferation in a distantly-related plant, rice[76]. This report indicates that the mechanisms that regulate regional cell proliferation might be conserved. In addition, reverse genetics approaches in several plant species has also begun to identify new genes that could function in flower development. Future functional analysis of these genes will illustrate their specific roles.

In summary, molecular genetic analysis of inflorescence and floral patterning is beginning to shed light on how these events are controlled, and on elements in this patterning that are evolutionarily conserved.

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