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The *SEP4* Gene of *Arabidopsis thaliana* Functions in Floral Organ and Meristem Identity

Gary Ditta, Anusak Pinyopich, Pedro Robles, Soraya Pelaz, and Martin F. Yanofsky*
Section of Cell and Developmental Biology
University of California, San Diego
9500 Gilman Drive, Department 0116
La Jolla, California 92093

Summary

The ABC model of flower organ identity is widely recognized as providing a framework for understanding the specification of flower organs in diverse plant species [1]. Recent studies in *Arabidopsis thaliana* have shown that three closely related MADS-box genes, *SEPALLATA1* (*SEP1*), *SEP2* and *SEP3*, are required to specify petals, stamens, and carpels because these organs are converted into sepals in *sep1 sep2 sep3* triple mutants [3, 4]. Additional studies indicate that the SEP proteins form multimeric complexes with the products of the B and C organ identity genes. Here, we characterize the *SEP4* gene, which shares extensive sequence similarity to and an overlapping expression pattern with the other *SEP* genes. Although *sep4* single mutants display a phenotype similar to that of wild-type plants, we find that floral organs are converted into leaf-like organs in *sep1 sep2 sep3 sep4* quadruple mutants, indicating the involvement of all four *SEP* genes in the development of sepals. We also find that *SEP4* contributes to the development of petals, stamens, and carpels in addition to sepals and that it plays an important role in meristem identity. These and other data demonstrate that the *SEP* genes play central roles in flower meristem identity and organ identity.

Results and Discussion

Flower development in *Arabidopsis* is controlled to a large degree by members of the MADS-box family of transcription factors. The ABC model of flower organ specification states that the individual and combined activities of the A-, B-, and C-class organ identity genes specify the identity of the four organ types with A alone specifying sepals, A and B together specifying petals, B and C specifying stamens, and C alone specifying carpels [1]. The A-class activity is encoded by the *APETALA1* (*AP1*) and *APETALA2* (*AP2*) genes, B-class activity is encoded by the *APETALA3* (*AP3*) and *PISTILLATA* (*PI*) genes, and C-class activity is encoded by the *AGAMOUS* (*AG*) gene. All of these genes encode putative transcriptional regulators, and, with the exception of *AP2*, all encode proteins belonging to the MADS-box gene family [2]. More recently, three closely related *SEPALLATA* (*SEP*) MADS-box genes have been incorporated into this model. In a *sep1 sep2 sep3* (*sepallata*) triple mutant, all flower organs are converted into sepals despite the fact that the onset of B and C gene expression is not affected in the triple mutant [3, 4]. The conversion of floral organs into sepals can be viewed as reversion to a more leaf-like state, an idea consistent with Goethe's hypothesis in the late 18th century that floral organs are essentially modified leaves.

Protein-protein interaction data, together with the fact that *sep1 sep2 sep3* triple mutants closely resemble BC double mutants (*ap3 ag* or *pi ag*), indicate that the SEP proteins interact with the products of the B and C organ identity genes to direct petal, stamen, and carpel development [1, 4, 5]. In spite of the fact that the *SEP* genes are known to be expressed early in cells that will later give rise to sepals [6, 7] and the observation that the SEP3 protein can interact with the AP1 A-class protein [4, 5], no alterations in sepal development were observed in *sep1 sep2 sep3* triple mutants. These data raise the possibility that a redundant gene might mask the roles of *SEP1*, *SEP2* and *SEP3* in sepal development and therefore suggest that inactivation of this gene in the presence of *sep1 sep2* and *sep3* mutations might result in alterations to sepal development. One candidate for a gene that acts redundantly with *SEP1*, *SEP2*, and *SEP3* is *SEP4* (previously called *AGL3*) [7, 8], which shares extensive sequence similarity with the other *SEP* genes and displays an RNA expression pattern that is qualitatively similar to the other *SEP*s during early flower development (Figure S1 in the Supplemental Data available with this article online, and Figures 1A–1C). As was found for *SEP1*, *SEP2*, and *SEP3*, RNA for *SEP4* could not be detected in inflorescence meristems or in stage 1 flower meristems. *SEP4* RNA was first observed throughout stage 2 flower primordia (Figure 1A), and by late stage 3 to early stage 4, expression was restricted to modest levels in sepals and to a conical region within the central dome in whorl 4 (Figure 1B). Although expression could still be detected early in stage 6 in emerging carpel primordia, it was not detected thereafter (Figure 1C). We used a PCR-based method to identify a T-DNA insertion mutant within the first intron of *SEP4* at position +307 relative to the translational start. Both RNA blotting (data not shown) and in situ hybridization analyses (Figure 1D) confirmed that this allele, designated *sep4-1*, is an RNA null.

First Whorl Organs Acquire Leaf-Like Characteristics in *sep1 sep2 sep3 sep4* Mutants

Inspection of *sep4* single-mutant plants did not reveal any phenotype that could be distinguished from wild-type. To determine if the *SEP4* gene acts redundantly with other *SEP* genes, we generated a *sep1 sep2 sep3 sep4* quadruple mutant (Supplemental Data). In contrast to *sep1 sep2 sep3* triple-mutant flowers consisting of only sepals [3], the quadruple mutant produced flowers possessing only leaf-like organs. The conversion of sepals to leaf-like organs was most striking on the adaxial (inner) surfaces of these organs. Whereas wild-type se-

*Correspondence: marty@ucsd.edu

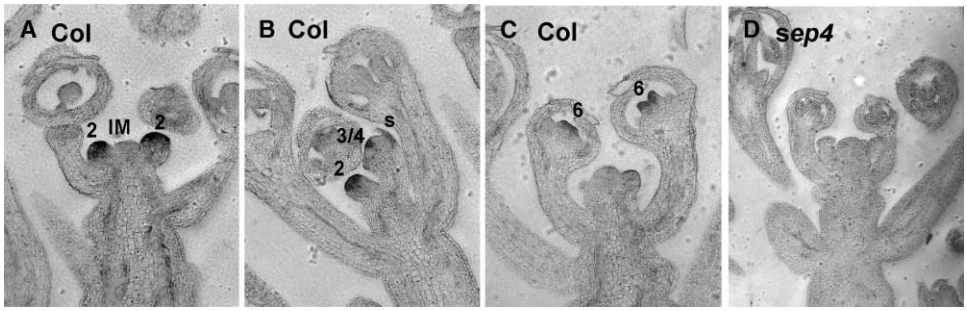

Figure 1. RNA In Situ Analysis of *SEP4* Expression

Expression is first detected and is strongest during stage 2 (A and B) but is not seen in the inflorescence meristem. By stages 3–4 (B), expression is localized to a conical region within the central dome and can be weakly detected in sepals. Expression persists into stage 6 in carpels but is no longer visible in sepals (C). Expression was not detected at later stages. No expression is seen for a *sep4* mutant also determined to be an RNA null by Northern blot analysis (D). Abbreviations are as follows: 2, 3, 4, and 6 = stages of *Arabidopsis* flower development; IM = inflorescence meristem; s = sepal.

pals have an abundance of elongated rectangular cells aligned parallel to one another (Figure 2A), the adaxial surfaces of *sep1 sep2 sep3 sep4* floral organs (Figure 2D) were found to consist almost entirely of irregular puzzle-shaped cells typical of those normally found on leaves (Figure 2C). Although the cells on the surfaces of *sep1 sep2 sep3* triple-mutant sepals (Figure 2B) were similar to those found on wild-type sepals, close inspection revealed cells with slightly irregular edges, suggesting a partial adoption of leaf fate. The adaxial surfaces of first-whorl organs on plants with other *sep* genotypes such as s*ep1 sep 2 sep4* and *sep3 sep4* were unaffected (data not shown), suggesting that all four *SEP* genes must be inactivated for the adaxial surface of sepals to adopt a leaf-like fate.

In contrast to the leaf-like appearance of the adaxial epidermal cells of *sep1 sep2 sep3 sep4* mutant flower organs, the abaxial (outer) epidermal cells of these organs appeared to have a mixture of both leaf- and sepal-like characteristics. The most obvious leaf-like feature was an abundance of branched (stellate) trichomes, particularly on the outermost (first whorl) organs (Figures 2G and 2J, yellow arrows). Although less frequent, stellate trichomes were also observed on adaxial surfaces in the quadruple mutant. Stellate trichomes are a feature of rosette and cauline leaves in *Arabidopsis* and are seldom observed on sepals of either wild-type (Figures 2E and 2H) or the *sep1 sep2 sep3* triple mutant (Figures 2F and 2I), both of which typically have simple, unbranched trichomes. Although the number of trichomes declined in later-arising flowers of the quadruple mutant, such trichomes when present were predominantly of the branched type. Close inspection of cells on the abaxial surface of the quadruple mutant revealed features similar to the sepals of both wild-type and the *sep1 sep2 sep3* triple mutant, such as the overall arrangement of epidermal cells and the presence of large, elongated cells (Figures 2H–2J, blue arrows). Another characteristic feature of leaves in *Arabidopsis* is that they have small appendages at their base called stipules, which are not present at the base of sepals. Structures resembling stipules were observed at the base of the leaf-like organs in the flowers of *sep1 sep2 sep3 sep4* quadruple mutants.

Taken together, these data indicate that sepals adopt features of leaves in the *sep1 sep2 sep3 sep4* quadruple mutant and suggest that the four *SEP* genes have overlapping activities required for normal sepal development. The leaf-like phenotype of organs in the *sep1 sep2 sep3 sep4* quadruple mutant resembles that seen for ABC mutants of genotypes *ap2-1 ap3-1 ag-1*, *ap2-1 pi-1 ag-1*, or *ap2-2 pi-1 ag-1* [1]. The fact that floral organs default to leaf-like organs when developmental specification is removed supports the notion that flower organs represent modified leaves.

Although floral organs in the *sep1 sep2 sep3 sep*4 mutant resemble leaves, it is clear that they also retain some features characteristic of sepals, suggesting the involvement of a *SEP*-independent function in specifying sepal identity. However, the fact that the *sep2-1* and *sep3-2* alleles are derived from the insertion of the autonomous transposable element En-1 (see the Supplemental Data available with this article online) leaves open the possibility that these residual sepal characteristics are the result a partial restoration of *SEP* activity after excision of the En-1 element.

SEP4 Contributes to Organ Development in All Four Whorls

The conversion of sepals into leaf-like organs in the *sep1 sep2 sep3 sep4* quadruple mutant indicates that *SEP4* contributes to sepal identity. To determine if *SEP4* also contributes to petal, stamen, and carpel identity, we introduced *sep4* into plants that were homozygous for mutations in *SEP1* and *SEP2* and heterozygous for mutation in *SEP3* but wild-type for *SEP4*. Such *sep1 sep2 sep3*/+ plants develop flowers that look normal except for a reduction in the number of stamens (Figures 3A and 3B). When a *sep1 sep2 sep3*/+ *sep4* mutant was generated, profound changes were observed in early-arising flowers (Figure 3C). Both organ number and organ identity were affected in a highly variable manner. Dissection of the individual organs of a typical early-arising flower from this mutant (Figure 3C) revealed two aberrant sepal-like organs, one sepal-petal hybrid, six abnormal stamens, and carpels with stellate trichomes on the valves (Figure 3C, inset). Stellate trichomes are never observed on wild-type carpels but were frequently

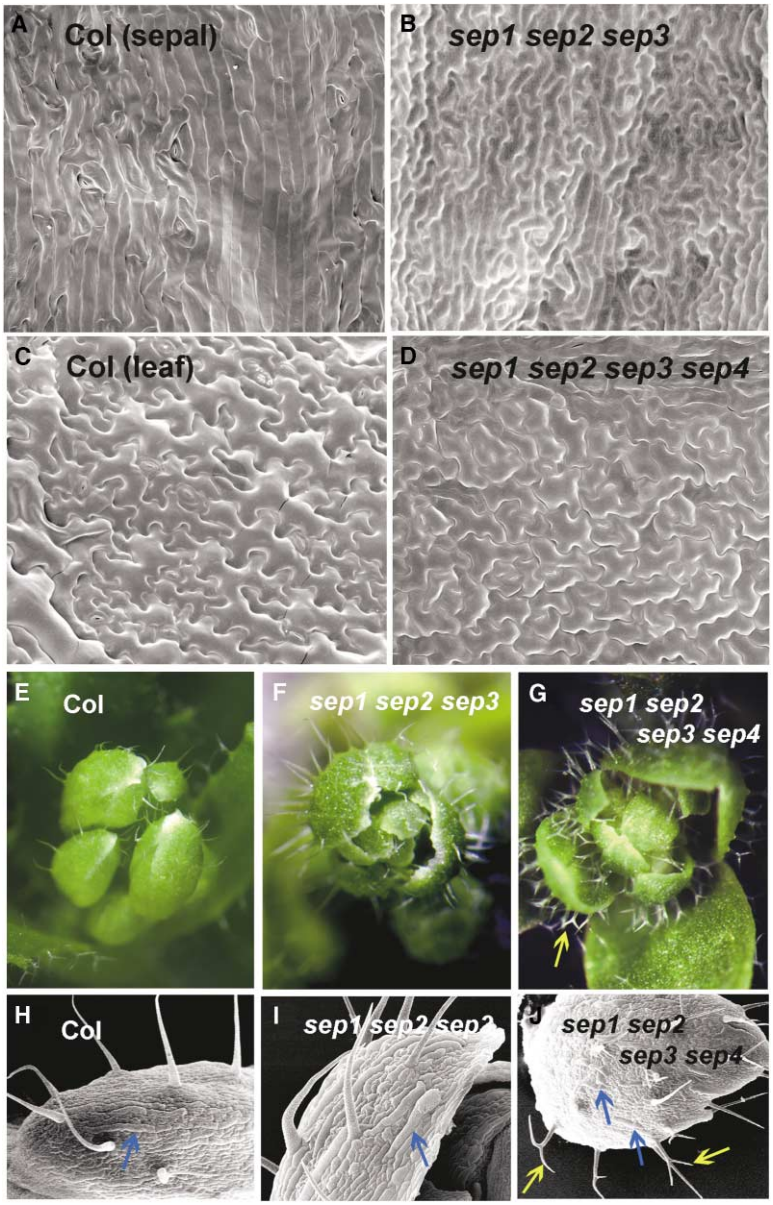

Figure 2. Floral Organs Resemble Leaves in *sep1 sep2 sep3 sep4* Mutants

The adaxial surface of wild-type (A) and *sep1 sep2 sep3* (B) mutant sepals consists primarily of elongated rectangular cells in a parallel arrangement. In contrast, cells on the adaxial surface of leaves have an irregular and interlocking shape (C). Cells on the adaxial epidermis of first whorl organs in *sep1 sep2 sep3 sep4* closely resemble those of leaves (D). Stomata are present in each instance (A–D). Sepals from flowers of wild-type (Col) (E and H) and *sep1 sep2 sep3* (F and I) have simple unbranched trichomes on the abaxial surface whereas the first whorl organs of *sep1 sep2 sep3 sep4* (G and J) have predominantly branched stellate trichomes (yellow arrows). The rest of the abaxial surface, including the epidermal cell structure (blue arrows) appears equivalent in each case (H–J).

observed on those of *sep1 sep2 sep3*/+ *sep4* plants. The gynoecium was often supported by an elongated gynophore, and many flowers displayed a loss of fourth-whorl determinacy such that flowers arose within flowers (Figure 3D). This loss of floral meristem determinancy is a well-documented feature of situations in which C-function activity is compromised [9]. The severe distortions of floral organ development that were seen in early flowers lessened over time so that later-arising flowers appeared more normal. However, even later-arising flowers displayed significant perturbations of organ development (Figure 3E). Eventually, plants with a *sep1 sep2 sep3*/+ *sep4* genotype set viable seed.

These data suggest that the *SEP* genes contribute to floral organ development in a redundant and additive fashion. While a *sep1 sep2 sep3*/+ mutant has enough remaining *SEP* function to support relatively normal flower development, the removal of *SEP4* lowers overall *SEP* activity below a threshold critical for proper floral organ development. Because the loss of *SEP4* can affect the development of all four organ types, it is apparent that *SEP4* plays a role in petal, stamen, and carpel development as well as in sepal development. Since previous work [3, 4] and work presented here show that *SEP1*, *SEP2*, and *SEP3* play roles in specifying the development of sepals, petals, stamens, and carpels, we conclude that all four *SEP* genes are important for the development of all four whorls of *Arabidopsis* flowers.

It should be noted that, whereas the data indicate overall *SEP* functionality to be fundamentally additive, individual *SEP* genes nevertheless contribute disproportionately to organ development. The removal of two copies of *SEP4* from a *sep1 sep2 sep3*/+ background (described above), for example, has a less dramatic effect on flower development than does removal of the single remaining copy of *SEP3* (*sep1 sep2 sep3*), whereas a

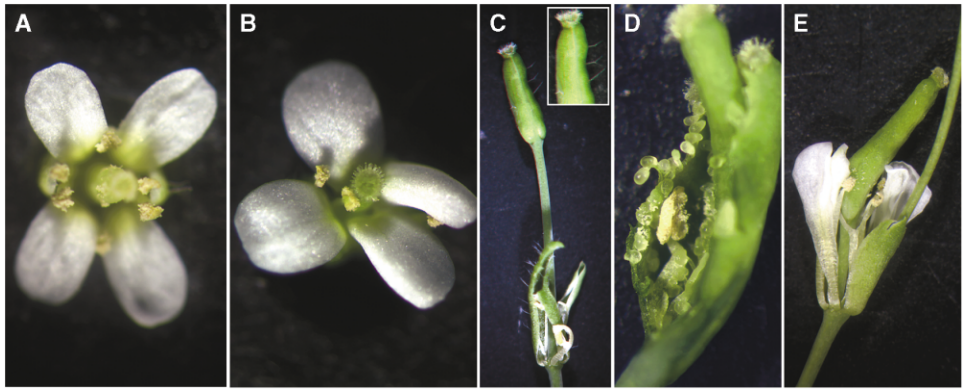

Figure 3. *SEP4* Functions in All Four Floral Whorls

A Col wild-type flower is shown in (A). *sep1 sep2 sep3*/+ flowers are fertile and set seed but have a reduced number of stamens (B). An early-arising flower from a *sep1 sep2 sep3*/+*sep4* plant shows striking alterations in organ development that are variable from flower to flower (C). As well as variations in organ number and identity, the elongated pedicel and stellate trichomes on the carpel valves (inset) of this flower are typical of many flowers. Another frequent occurrence is a loss of floral determinacy such that flowers arise within flowers. In (D), a carpel has been opened to reveal the additional carpel and stamens contained alongside developing ovules. Later-arising flowers show a less severe phenotype (E). Even here, however, a split petal occurs as a first whorl organ, in addition to a bifurcated stamen, a bent carpel, and overall reduced organ number. An elongated pedicel of an axilary flower can be seen on the right.

sep1 sep2 sep4 mutant shows no significant perturbation of floral organ development (data not shown). These observations suggest that *SEP3* is significantly more critical for flower development than *SEP4*. Whether this difference is attributable primarily to variations in protein function or to RNA expression, however, is unknown. It is worth noting that in vitro work has shown SEP3 to be a significantly stronger transcriptional activator than either SEP1 or SEP2 [4].

SEP4 Is Involved in Maintaining Meristem Identity

In addition to alterations in organ identity, *sep1 sep2 sep3* triple mutants also display a slight loss of flower meristem identity as evidenced by occasional secondary flowers in the axils of first-whorl organs [3] (Figure S2A). The loss of floral meristem identity is even more extreme in the case of *sep1 sep2 sep3 sep4* quadruple mutants (Figure S2B). To further examine the meristem identity role of the *SEP* genes, and *SEP4* in particular, we combined various *SEP* mutations with mutations in the *AP1* and *CAULIFLOWER* [*CAL*] meristem identity genes. Mutations in *AP1* lead to the development of flowers in the axils of first-whorl organs, and this has been interpreted as a partial conversion of flower meristems into inflorescence meristems [10] (Figure 4B). This conversion is nearly complete in *ap1 cal* double mutants, in which flower meristems behave like inflorescence meristems and continuously elaborate new meristems, resulting in a "cauliflower" phenotype [10] (Figure 4D). Although *ap1 cal* double mutants elaborate meristems for a considerable period of time, flowers resembling those of *ap1* single mutants eventually appear and set seed. Because *cal* single mutants cannot be distinguished from wild-type, it appears that all of the functions of *CAL* are encompassed by those of *AP1*.

Remarkably, when we constructed the *ap1 sep1 sep2 sep4* quadruple mutant, we found that these plants produced a *cauliflower* phenotype similar to *ap1 cal* mutants, even though the plants were homozygous for the wild-type *CAL* gene (Figure 4F). One explanation for these results is that the SEP proteins are required for CAL protein activity just as they have previously been shown to be required for activities of the B- and C-gene products. We also found that an *ap1 sep4* double mutant had a meristem identity defect intermediate between that of *ap1* and *ap1 cal* mutants. Inflorescences of *ap1 sep4* plants appeared cauliflower-like early after bolting but soon began to produce *ap1*-like flowers on their periphery and maintained this general appearance (cauliflower-like center, *ap1*-like periphery; Figure 4C) for a considerable time before the cauliflower-like character diminished. The intermediate nature of the *ap1 sep4* phenotype suggests that a *sep4* mutation has a similar but less severe effect on maintenance of floral meristem identity than does a *cal* mutation and again suggests that SEP4 protein is required for full activity of CAL protein.

Interestingly, we found that the *sep4* mutation enhanced the meristem identity defect of *ap1 cal* cauliflower mutants (Figure 4E). Although inflorescences of *ap1 cal sep4* mutants looked indistinguishable from cauliflower structures formed by either *ap1 cal* or *ap1 sep1 sep2 sep4,* the *ap1 cal sep4* triple mutant inflorescences nevertheless took 7–10 days longer than *ap1 cal* mutants did to begin producing flowers (unpublished data). In *ap1 cal sep4* mutants, the increased flower meristem identity defect cannot be attributed to a decrease in CAL function. However, since *ap1 cal sep4* mutants eventually produce flowers, it is clear that one or more additional genes must be playing a role similar to *CAL* in promoting flowering. The *FUL* gene, which is a close relative of both *AP1* and *CAL*, has been shown to function in specifying floral meristem identity and to be required for the eventual flowering of *ap1 cal* mutants [11]. Therefore, one interpretation for the delay in flowering of *ap1 cal sep4* is that loss of SEP4 activity results in a decrease of FUL activity such that the *ap1 cal sep4* mutant has a phenotype intermediate between that of

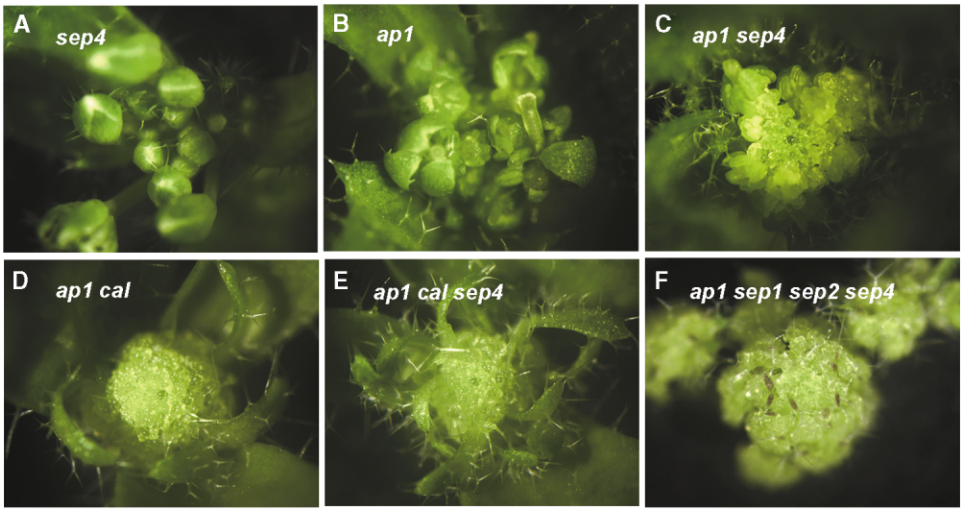

Figure 4. *SEP4* Functions in Meristem Identity

sep4 inflorescences look completely normal (A). An *ap1-15* mutant (B; strong allele of Col) lacks petals and shows a complex structure due to the appearance of additional flowers in the axils of first whorl organs. The center of an *ap1 sep4* inflorescence has a cauliflower-like appearance but is surrounded on its periphery by flowers (C). This architecture is maintained for a prolonged period. An *ap1 cal* flower has a classic cauliflower appearance (D). An *ap1 cal sep4* flower looks essentially the same as *ap1 cal* although there are more frequent bract-like organs interspersed as displayed here (E). As discussed in the text, resolution of the cauliflower head and the initiation of fruit development takes longer to occur in *ap1 cal sep4* than in *ap1 cal*. An *ap1 sep1 sep2 sep4* mutant forms a cauliflower-like inflorescence in the presence of a wild-type *CAL* allele (F).

ap1 cal (slow to produce flowers) and *ap1 cal ful* (never produces flowers). *SEP4* appears to have a more important role than the other *SEP* genes in specifying meristem identity because other combinations of *sep* alleles with *ap1*, such as *ap1 sep1 sep2* or *ap1 sep3* did not display the cauliflower-like character (data not shown).

Overexpression of SEP4 Promotes a Floral Fate

Loss of function studies showed that *SEP4* acts to promote flower meristem identity. To determine if *SEP4* is sufficient to promote a floral fate, we generated transgenic plants in which the *SEP4* gene was ectopically expressed from the CaMV35S promoter. In contrast to wild-type inflorescence meristems, which are capable of producing many flowers prior to senescence, constitutive expression of *SEP4* resulted in the production of fused terminal flowers in place of inflorescence meristems (Figure S3C). These terminal flowers frequently consisted of a fusion of multiple flowers that had apparently originated from the inflorescence meristem at around the same time, and they typically had many more than the number of organs that a single flower would normally produce. The fact that the inflorescence meristem terminated after producing these flowers indicates that the ectopic expression of *SEP4* was sufficient to convert the normally indetermimate inflorescence meristem into a floral meristem. These phenotypes are similar to what has been observed for terminal flower (*tfl*) mutants, in which lack of *TFL* activity allows the inflorescence meristem to be consumed in the production of terminal flowers rather than be maintained in order to produce additional flowers [12], and supports the proposed role of SEP4 in promoting flower meristem identity.

SEP Function and Flower Development

As well as interacting with ABC proteins [4, 5], SEP3 forms transcriptionally active complexes with other proteins related to AGAMOUS known to be important for ovule development, including STK, SHP1, and SHP2 [13]. These studies, together with demonstrated roles for *SEP* genes in specifying ovule, meristem, and organ identity ([3–5, 13, 19] and this work), show that the *SEP* genes play a broad and pivotal role in flower development in *Arabidopsis*. *SEP* orthologs in other plant species are likely to play equivalently important roles in flower development. In *Petunia*, for example, *SEP* orthologs *FBP2* and *FBP5* were shown to function redundantly and to be essential for B and C function activity. Whorls 2, 3, and 4 acquired sepaloid characteristics and thus became more leaflike in an *fbp2 fbp5* double mutant [14]. *SEP* orthologs have been identified in eudicot, monocot, as well as gymnosperm species [15–18]. Such widespread and ancient structural conservation suggests that *SEP* genes have been important for plant reproduction since before the appearance of Angiosperms over 140 million years ago. The primary role of SEP proteins appears to be in facilitating the formation of specific transcription complexes and in conferring upon these complexes transcription activation potential [4, 19]. This suggests that the other MADS-box proteins within such complexes contribute primarily to promoter selection, although it has not been ruled out that SEP proteins might influence this process as well. As summarized in the "floral quartet" model, a wide array of heteromeric complexes can be derived from various combinations of SEP proteins and other MADS-box proteins [20]. Because each of these is likely to possess a unique level of transcriptional activation capability [13], it is clear that the *SEP* gene family endows the cell with

an exquisitely sensitive mechanism for controlling floral gene expression.

Supplemental Data

Supplemental Data including Supplemental Experimental Procedures and two figures are available at http://www.current-biology.com/cgi/content/full/14/21/1935/DC1/.

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