Abstract

The floral transition in plants is regulated by a complex interplay among environmental and endogenous signals that determine a critical transition in the activity of the shoot apical meristem (SAM) from vegetative to reproductive growth. During this transition, the size, morphology, and identity of the SAM undergo notable changes. In *Arabidopsis thaliana* (Arabidopsis), the APETALA2 (AP2) transcription factor plays a dual role in regulating both SAM development and identity. Before the floral transition, AP2 represses the transcription of MADS-box family genes *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*) and *FRUITFULL* (*FUL*). As plants age, the floral repressor AP2 is gradually downregulated at the post-transcriptional level by miR172, and at the transcriptional level by SOC1 and FUL. This mutual repression between AP2 and SOC1/FUL regulates the timing of the transition of Arabidopsis into the floral phase and the initiation of inflorescence development.

The *soc1 ful* double mutant exhibits reversion of the inflorescence meristem to vegetative growth, with the formation of rosette leaves instead of flowers on the primary inflorescence. By genetic analysis, AP2 was identified as a key factor in this reversion. The triple *ap2 soc1 ful* mutant did not show the inflorescence reversion phenotype of the *soc1 ful* double mutant. Moreover, introduction of an *rAP2* (miR172-resistant *AP2*) transgene into the *soc1 ful* background, caused a more severe reversion of the inflorescence. These findings demonstrate that impairing repression of AP2 in the *soc1 ful* double mutant causes reversion of the inflorescence to the vegetative state.

To test the effect of AP2 on inflorescence development, a chemically inducible expression system was used to analyse its effects at different developmental stages and in specific spatial domains. Induction of *rAP2-mScarlet* in the inflorescence meristem from the *FD* gene promoter caused reversion to the vegetative state, accompanied by a reversion of the shape of the meristem to that characteristic of floral transition. This alteration in meristem shape was associated with an expansion of the domain of expression of the WUSCHEL homeobox transcription factor. These results support the importance of maintaining AP2 repression in the inflorescence to prevent reversion to the vegetative state, and underscore the role of AP2 in controlling the plasticity of SAM identity during the transition to flowering.

In the perennial species *Arabis alpina*, the life cycle consists of annual cycles of vegetative growth and flowering, which are regulated by the perennial-specific floral repressor *PERPETUAL FLOWERING 1* (*PEP1*)—an orthologue of Arabidopsis *FLOWERING LOCUS*

C. The *PEP1* gene encodes a MADS-domain transcription factor that represses flowering until the plant is exposed to vernalization, when *PEP1* transcription is repressed. The *pep1* mutant flowers continuously, or perpetually, without reverting to the vegetative state. The *SOC1* gene is a direct target of PEP1 and is strongly upregulated in the early-flowering *pep1* mutant; however, the *soc1 pep1* double mutant fails to flower. Nevertheless, when these plants are exposed to vernalization they do flower, showing the existence of a vernalization response in the absence of active *PEP1*. *MADS AFFECTING FLOWERING* (*MAF*) genes are homologues of *PEP1*, and using CRISPR-cas9 reverse genetics, I showed that these still function as floral repressors in the *pep1* mutant, but do not repress floral transition as strongly as *PEP1* and are unlikely to explain the vernalization response of *soc1 pep1* double mutants. However, in the absence of both *SOC1* and *FUL* activity in *soc1 ful1 ful2 pep1* quadruple mutants, flowering was prevented under long-day conditions or following vernalization. These results indicate that coordinated activation of *SOC1* and *FUL* is essential for floral transition of *A. alpina* during vernalization.

In conclusion, the work presented here in Arabidopsis provides a regulatory framework for understanding how *AP2* and *SOC1/FUL* coordinate the floral transition and maintain inflorescence development. In *A. alpina*, studies on *SOC1* have further revealed the interplay among *PEP1*, *MAFs*, *SOC1*, and *FUL* in establishing the vernalization requirement and regulating repeated flowering in perennials.