Journal of Experimental Botany, Vol. 61, No. 9, pp. 2247–2254, 2010
doi:10.1093/jxb/erq098 Advance Access publication 22 April, 2010

Journal of Experimental Botany
www.jxb.oxfordjournals.org

FLOWERING NEWSLETTER REVIEW

Regulation and function of SOC1, a flowering pathway integrator

Jungeun Lee[1,2] and Ilha Lee[1,2,*]

[1] National Research Laboratory of Plant Developmental Genetics, School of Biological Sciences, Seoul National University, Seoul, 151-742, Korea

[2] Global Research Laboratory for Flowering at SNU and UW, Seoul 151-742, Korea

* To whom correspondence should be addressed: E-mail: ilhalee@snu.ac.kr

Received 21 January 2010; Revised 12 March 2010; Accepted 15 March 2010

Abstract

***SOC1*, encoding a MADS box transcription factor, integrates multiple flowering signals derived from photoperiod, temperature, hormone, and age-related signals. SOC1 is regulated by two antagonistic flowering regulators, *CONSTANS* (*CO*) and *FLOWERING LOCUS C* (*FLC*), which act as floral activator and repressor, respectively. *CO* activates *SOC1* mainly through *FT* but FLC represses *SOC1* by direct binding to the promoter. *SOC1* is also activated by an age-dependent mechanism in which *SPL9* and microRNA156 are involved. When *SOC1* is induced at the shoot apex, SOC1 together with AGL24 directly activates *LEAFY* (*LFY*), a floral meristem identity gene. *APETALA1* (*AP1*), activated mainly by *FT*, is also necessary to establish and maintain flower meristem identity. When *LFY* and *AP1* are established, flower development occurs at the anlagen of shoot apical meristem according to the ABC model. During early flower development, AP1 activates the A function and represses three redundantly functioning flowering time genes, *SOC1, AGL24*, and *SVP* to prevent floral reversion. During late flower development, such repression is also necessary to activate *SEPALATA3* (*SEP3*) which is a coactivator of B and C function genes with *LFY*, otherwise *SEP3* is suppressed by SOC1, AGL24, and SVP. Therefore, SOC1 is necessary to prevent premature differentiation of the floral meristem.**

Key words: Flower development, flowering, integrator, SOC1.

Introduction

The proper timing of flowering is the most critical aspect to ensure reproductive success. For this reason, plants have evolved sophisticated and elaborate regulatory mechanisms to bloom at the best time. Three decades of genetic analyses using *Arabidopsis* have revealed complex genetic networks for flowering that are mainly regulated by four genetic pathways, photoperiod, autonomous, vernalization, and gibberellin induced pathways (Simpson and Dean, 2002; Boss *et al.*, 2004; Sung and Amasino, 2004; Baurle and Dean, 2006). In *Arabidopsis*, the floral induction signals from these four major flowering pathways are transmitted to two central flowering regulators *CONSTANS* (*CO*) and *FLOWERING LOCUS C* (*FLC*) that antagonistically regulate flowering (Putterill *et al.*, 1995; Samach *et al.*, 2000). The *CO* gene encoding a zinc finger protein acts as a floral activator and mediates the photoperiod pathway, whereas the *FLC* gene encoding a MADS box protein acts as a floral repressor and mediates the autonomous and vernalization pathways. In turn, *CO* and *FLC* regulate the expression of downstream genes, the so-called flowering pathway integrators, *FT*, *SUPPRESSOR OF OVEREXPRESSION OF CO 1* (*SOC1*), and *LEAFY* (*LFY*). These three genes integrate signals from multiple flowering pathways and their expression levels eventually determine the exact flowering time (Simpson and Dean, 2002; Parcy, 2005).

SOC1 encodes a MADS box protein and is conserved among Angiosperms including both Monocotyledons and Dicotyledons (Lee *et al.*, 2000, 2004, 2008; Cseke *et al.*, 2003; Ferrario *et al.*, 2004; Nakamura *et al.*, 2005). Recent studies show that SOC1 is a multifunctional protein which

regulates not only flowering time but also floral patterning and floral meristem determinancy (Liu *et al.*, 2007, 2009; Melzer *et al.*, 2008). Such characteristics of SOC1 are also reported in other species beyond *Arabidopsis*. Therefore SOC1 is likely to play a role as a general regulator in organogenesis in plant development. In this review, the focus is on the regulation and function of *SOC1* as a floral activator and the newly identified functions of SOC1 are discussed based on the latest research (Fig. 1).

Identification of *SOC1*, a flowering time regulator

SOC1 has been identified by four independent approaches. It has been identified through a screening of suppressor mutants of overexpression of *CO*, which exhibits an extremely early flowering (Onouchi *et al.*, 2000). Loss of function of *SOC1* delays the early flowering of *35S::CO*. It has also been identified as a direct target of CO (Samach *et al.*, 2000). In *35S::CO:GR* transgenic plants, glucocorticoid treatment in the presence of cycloheximide induced the expression of *SOC1*, suggesting that *SOC1* is directly regulated by *CO*. *SOC1* has also been identified through the screening of a gain-of-function suppressor mutant from late flowering winter annual plants that have both *FRIGIDA* and *FLC* (Lee *et al.*, 2000). Overexpression of *SOC1* suppressed the late flowering phenotype caused by the high expression of *FLC* in winter annual plants, indicating that *SOC1* is a downstream target of *FLC*. It has also been identified by *Arabidopsis* homologue searching of the *MADSA* gene which is involved in the transition to flowering in mustard (Borner *et al.*, 2000). Subsequenctly, it has been shown that CO and FLC regulate *SOC1* expression via separate regions of the *SOC1* promoter (Hepworth *et al.*, 2002; Searle *et al.*, 2006). The loss-of-function and gain-of-function mutants of *soc1* exhibit late flowering and early flowering, respectively, and the mutants are able to respond to photoperiod. Expression analyses showed that *SOC1* is expressed mainly in developing leaves and meristems and the expression level is increased according to developmental age, which are characteristics suitable for a floral pathway integrator (Samach *et al.*, 2000).

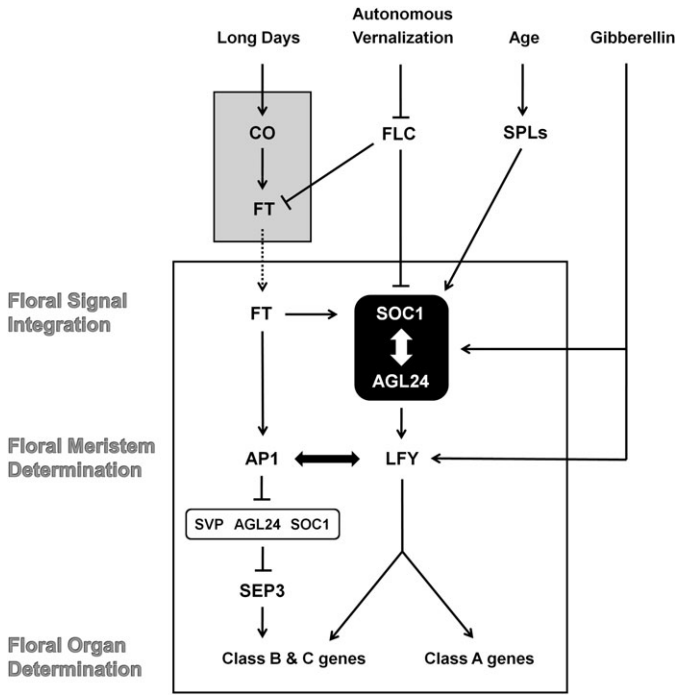

Fig. 1. SOC1 activity integrating multiple flowering signals and linking to flower development. SOC1 integrates multiple flowering signals from the long day, autonomous, and vernalization pathways. It also integrates flowering signals derived from plant age and gibberellin. SOC1 and AGL24 interact and positively regulate each other, thus providing a positive feedback loop (black box). The two genes expressed in the shoot apex activate *LFY*, a flower meristem identity gene. Subsequently, *LFY* initiates floral organ development by inducing a class A gene. In addition to the flowering time regulation, *SOC1* and *AGL24* are involved in the repression of precocious floral organ development through repression of *SEP3*, a gene required for activation of class B and C genes. In this way, *SOC1* and *AGL24* ensure floral induction and flower development occur in their proper time and space. The grey box indicates the vasculature of the leaf where CO-FT induction occurs, whereas the open rectangle indicates the shoot apical meristem where floral evocation occurs.

Upstream regulators of *SOC1*

Positive regulation of SOC1 *by the photoperiod pathway*

The *CO* gene plays a central role in the photoperiod pathway. Its mRNA levels show a circadian rhythm and the protein is stabilized by light, which is a key aspect of the measurement of the control of day length for flowering (Yanovsky and Kay, 2002; Valverde *et al.*, 2004). The expression of *FT*, *SOC1*, and *LFY,* the three flowering pathway integrators, are reduced in the *co* mutant but increased in *35S::CO* (Putterill *et al.*, 1995; Samach *et al.*, 2000; Yanovsky and Kay, 2002). Consistent with this, the overexpression of *SOC1*, *FT*, or *LFY* rescues the late flowering of *co*, whereas *soc1*, *ft*, *lfy* loss-of-function mutations delay the early flowering of *35S::CO*, suggesting that *FT*, *SOC1*, and *LFY* are downstream targets of *CO* (Moon *et al.*, 2005; Yoo *et al.*, 2005). However, later reports suggested that *FT* is the major output of CO and *SOC1* is regulated through *FT* (Wigge *et al.*, 2005; Yoo *et al.*, 2005). While the null mutation of *ft* was completely suppressed, the *soc1* mutation only partially suppressed the early flowering of *35S::CO* (Yoo *et al.*, 2005). In addition, an experiment treating a single long day showed that *FT* but not *SOC1* expression is increased depending on CO activity (Wigge *et al.*, 2005). The expression of *SOC1* is, rather, regulated by *FT* such that *SOC1* is increased by *35S::FT* and decreased by *ft* (Moon *et al.*, 2005; Yoo *et al.*, 2005). However, *SOC1* acts partially independently of *FT*. *ft soc1* double null mutants show an additive late flowering phenotype and the *SOC1* expression level is not much

reduced by *ft* compared to the mutants in the autonomous pathway (Lee *et al.*, 2000; Moon *et al.*, 2005; Yoo *et al.*, 2005), indicating that there is another factor(s) regulating *SOC1* expression.

The activation of *FT* by CO occurs specifically in the phloem that is not in the shoot apical meristem (SAM) (Takada and Goto, 2003; An *et al.*, 2004; Searle *et al.*, 2006), but the function of *FT* is required in the meristem for flowering, indicating that FT has to move to the SAM. Indeed, it has been revealed that the 20 kDa FT protein moves to the shoot apical meristem (SAM) (Takada and Goto, 2003; Searle *et al.*, 2006). Furthermore, FT interacts with a bZIP transcription factor, FD, which is expressed in the SAM, and regulates the downstream target genes such as *APETALA1, FRUITFUL*, and *SEPALATA3* (Abe *et al.*, 2005; Moon *et al.*, 2005; Wigge *et al.*, 2005; Corbesier *et al.*, 2007; Jaeger and Wigge, 2007; Mathieu *et al.*, 2007). An *in situ* hybridization assay has suggested that up-regulation of *SOC1* in the meristem is one of the earliest events in floral transition and the meristematic expression of *SOC1* is effective in promoting early flowering (Lee *et al.*, 2000; Samach *et al.*, 2000; Searle *et al.*, 2006). Since *SOC1* integrates the photoperiod pathway through FT, it is most likely that FT protein moves to the SAM and interacts with FD to up-regulate *SOC1*.

Age-dependent regulation of SOC1

As described above, FT is not the sole regulator of *SOC1*; the expression of *SOC1* increases according to developmental age and such an increase is independent of the FT/FD regulator and photoperiod (Moon *et al.*, 2003; Wang *et al.*, 2009). Recent reports suggested that *SPL* (*SQUAMOSA BINDING FACTOR-LIKE*) family transcription factors are involved in the age-related regulation of *SOC1* (Wang *et al.*, 2009). SPL transcription factors are known to influence a series of phase transitions in plants from juvenile to adult as well as vegetative to reproductive phase transitions (Schwab *et al.*, 2005; Wu and Poethig, 2006). SPLs are post-transcriptionally silenced by *microRNA156* (*miR156*) which is highly expressed in the juvenile phase and decreased as the plant ages; thus, the transcript level of *SPL*s is increased according to growth (Wang *et al.*, 2009; Wu *et al.*, 2009). The overexpression of *SPL*s accelerates, whereas a reduction of SPL activity through *miR156* overexpression delays phase transitions, and thus flowering too (Schwab *et al.*, 2005; Wu and Poethig, 2006; Schwarz *et al.*, 2008). Indeed, *SPL9,* which shows low expression at the early seedling stage but gradually increases afterwards independent of the photoperiod, binds to the first intron of SOC1, suggesting that SPL9 is an age-related positive regulator of SOC1 independent of FT/FD (Wang *et al.*, 2009).

Gibberellin-induced activation of SOC1

Gibberellin (GAs) is a plant hormone regulating a diverse range of plant growth and development. In *Arabidopsis*, GA signalling has a profound effect on flowering under non-inductive short days although it has a relatively minor influence under long days: the GA biosynthetic mutant, *ga1*-3, fails to flower under short days although flowering is only slightly delayed compared with the wild type under long days (Wilson *et al.*, 1992). *SOC1* integrates the GA pathway such that the *soc1* null mutant shows a reduced sensitivity to GA and overexpression of *SOC1* can rescue the non-flowering phenotype of *ga1-3* in short days. However, the molecular mechanism by which GA regulates *SOC1* expression is unknown. By contrast, it is known that gibberellins promote expression of *LFY* via distinct *cis*-elements on the promoter that can be bound by a GAMYB protein (Blazquez *et al.*, 1998; Gocal *et al.*, 1999, 2001). Considering that *SOC1* regulates *LFY* by direct binding to its promoter, gibberellins regulate *LFY* transcription by both *SOC1*-dependent and -independent pathways. Taken together, gibberellins influence the phase transition through the regulation of *SOC1* and *LFY* at the shoot apex.

Negative regulation of SOC1 *by repressor complex including* FLC *and* SVP

The signals from the vernalization and autonomous pathways converge on a strong repressor of flowering, *FLOWERING LOCUS C* (*FLC*). The autonomous and vernalization pathways promote flowering by repressing *FLC* expression and many genes involved in the vernalization and autonomous pathways control the epigenetic status of the *FLC* chromatin (Amasino, 2004; Baurle and Dean, 2006). FLC directly represses the expression of *FT, FD,* and *SOC1,* by binding to the promoters of *FD*, *SOC1,* and the first intron of *FT* (Searle *et al.*, 2006), thus preventing flowering until plants acquire the competency to flower. Consistent with this, *FLC* expressed in leaves delays flowering by repressing *FT* and *SOC1*, and *FLC* in the SAM delays flowering by repressing *SOC1* and *FD* (Searle *et al.*, 2006). Although how *SOC1* expressed in the leaves activates flowering is not known, the function of SOC1 and FT/FD in the SAM are well characterized. They activate floral meristem identity genes, *LFY, AP1*, and *FUL*, and thus initiate floral development in the shoot apex (Ruiz-Garcia *et al.*, 1997; Abe *et al.*, 2005; Wigge *et al.*, 2005).

SHORT VEGETATIVE PHASE (*SVP*), which encodes another MADS box transcription factor, is also a negative regulator of flowering in *Arabidopsis* (Hartmann *et al.*, 2000). The expression of *SVP* is mainly regulated by GA and the autonomous pathway but is not affected by the long day pathway or vernalization (Li *et al.*, 2008). In addition, *FRIGIDA,* which induces the higher expression of *FLC* in the winter annual *Arabidopsis*, does not affect the expression of *SVP* either. Thus, the regulatory mechanism of *SVP* is somewhat different from *FLC*. However, SVP interacts with FLC to form a floral repressor complex and directly binds to the promoters of *SOC1* and *FT* for transcriptional repression (Lee *et al.*, 2007; Li *et al.*, 2008). Consistent with the repressor function, *svp* loss-of-function mutation caused elevated expression of *SOC1* and *FT* whereas *35S::SVP* suppressed the expression of these genes.

It is noteworthy that the expression of *SOC1* is more strongly affected by the SVP-FLC repressor complex than by *FT* (Li *et al.*, 2008). These results suggest that SVP is another central flowering repressor and its interaction with FLC determines the expression of the floral pathway integrators in response to various endogenous and environmental signals. Size exclusion chromatography analysis shows that FLC is present in a high molecular weight complex around the size of 600–800 kDa, which is larger than the size expected for a heterodimer (50–60 kDa) or tetramer (100–120 kDa) of MADS box proteins (Helliwell *et al.*, 2006). Interestingly, the *SOC1* gene is widely associated with the repressive histone trimethylation mark at the transcriptional start site region (Adrian *et al.*, 2009), thus it is possible that FLC represses *SOC1* by forming a floral repressor complex inducing an inactive chromatin state of the target genes.

Positive feedback loop with AGL24

AGL24 is a close homologue of *SVP* encoding a MADS box transcription factor. However, AGL24 acts as a flowering activator similar to SOC1 (Yu *et al.*, 2002; Michaels *et al.*, 2003; Liu *et al.*, 2008). The loss-of-function mutant of *agl24* shows late flowering and the overexpression of *AGL24* causes early flowering. In addition, the expression of *AGL24* is affected by several flowering pathways including photoperiod, vernalization, and autonomous pathways, suggesting that *AGL24* is another flowering pathway integrator. *AGL24* is widely expressed in plant tissues such as leaves, shoot apices, roots, stems, and inflorescence; thus its spatial expression domain largely overlaps that of *SOC1* (Yu *et al.*, 2002; Michaels *et al.*, 2003). Interestingly, AGL24 and SOC1 are able to up-regulate each other's expression and such co-regulation is achieved by direct binding to the promoter of the other, indicating that a positive feedback loop between AGL24 and SOC1 integrates flowering signals (Michaels *et al.*, 2003; Liu *et al.*, 2008).

The SOC1 protein activity

A growing body of evidence indicates that FT mainly regulates *AP1,* and SOC1 mainly regulates *LFY* for floral initiation (Ruiz-Garcia *et al.*, 1997; Abe *et al.*, 2005; Wigge *et al.*, 2005; Lee *et al.*, 2008). AP1 and LFY are the two major determinant for flower meristem identity, thus are hub points linking floral induction and flower development (Mandel *et al.*, 1992; Gustafson-Brown *et al.*, 1994; Parcy *et al.*, 1998; Lohmann *et al.*, 2001). When the flower meristem identity genes such as *AP1* and *LFY* are mutated, plants produce shoot-like structures instead of flowers. LFY is a plant-specific transcription factor found in most of the plant kingdom from moss to angiosperms, and its sequence and function are conserved (Coen *et al.*, 1990; Weigel *et al.*, 1992; Mouradov *et al.*, 1998; Molinero-Rosales *et al.*, 1999; Champagne *et al.*, 2007). *SOC1* is known to induce *LFY* expression at the shoot apex. The *soc1* loss of function mutant exhibits decreased and gain of function mutant exhibits increased *LFY* expression, indicating that *SOC1* acts upstream of *LFY* (Lee *et al.*, 2000; Samach *et al.*, 2000; Moon *et al.*, 2003). Indeed, ChIP analysis showed that SOC1 directly binds to the modified CArG box in the *LFY* promoter (Lee *et al.*, 2008; Liu *et al.*, 2008).

The SOC1 protein is a member of the MIKC type MADS box proteins composed of 214 amino acids with the size of 24 kDa. Thus, it is composed of four characteristic domains, a MADS box (M), an intervening (I) region, a keratin (K) box, and a C-terminal domain from N-terminus to C-terminus. A recent study using intragenic suppressor mutants of overexpressor of *SOC1* and cellular localization analysis using a protoplast transient assay with *SOC1–GFP* fusion provided a clue to the biochemical function of each domain *in vivo* (Lee *et al.*, 2008). The missense mutation in Arg24, which is a highly conserved residue among MADS box proteins, completely eliminated the *SOC1* function as a flowering activator. X-ray crystallography analysis showed that the corresponding Arg residue in the MADS box of Serum Response Factor is a residue directly in contact with the phosphate group of DNA (Pellegrini *et al.*, 1995). Consistent with this, the missense mutation of Arg24 resulted in the loss of SOC1 binding to the *LFY* promoter (Lee *et al.*, 2008).

When the full-length SOC1 protein is expressed in protoplasts using a transient assay system, it is mainly localized in the cytoplasm. Such cytoplasmic localization was confirmed in *SOC1* overexpressor mutants *in vivo* such that SOC1 protein was not detected in the nuclear extracts (Lee *et al.*, 2008). For the nuclear trafficking of SOC1, the interaction with AGL24 is necessary and the MADS and I domains of SOC1 are required not only for nuclear localization but also for heterodimerization with AGL24 (Lee *et al.*, 2008).

SOC1 regulates floral meristem development

When a flowering signal(s) reaches the shoot apex, the identity of the SAM changes from the vegetative to the reproductive phase and the earliest event occurring is the rapid increase of *LFY* and *AP1* at the anlagen of the shoot apical meristem (Gustafson-Brown *et al.*, 1994; Lee *et al.*, 1997). In order to produce normal flower structures, the floral meristem identity must be actively maintained through a balance between indeterminancy and differentiation. Otherwise, floral reversion occurs which is the emerging floral meristems going backwards to produce inflorescence shoots. Such a floral reversion phenotype is observed in the mutants *lfy* and *ap1*, suggesting that floral meristem identity genes, *LFY* and *AP1,* promote the establishment and maintenance of floral identity in newly formed floral primordia (Weigel *et al.*, 1992; Wagner *et al.*, 1999; Parcy *et al.*, 2002). Recent reports suggest that the crosstalk between flowering time genes and floral meristem identity genes takes place to maintain floral identity (Yu *et al.*, 2004; Liu *et al.*, 2007, 2009). The ectopic expression

of *AGL24* caused an *ap1*-like phenotype, thus promoting partial transformation of flowers into inflorescences (Yu *et al.*, 2004). Consistent with this, the expression of *AGL24* is up-regulated by *AP1*. In addition, such a phenotype is enhanced by the ectopic expression of *SOC1* and *SVP*, thus, the floral meristems were converted to shoots (Yu *et al.*, 2004). It is likely that SOC1, AGL24, and SVP act redundantly to maintain shoot identity whereas AP1 acts to prevent the indeterminate growth of floral meristems by repressing these three flowering time genes. Indeed, it has been shown that AP1 binds to the promoters of *SOC1*, *AGL24*, and *SVP* genes by ChIP.

When flower meristem identity is established and maintained, floral organs are produced according to the ABC model (Coen and Meyerowitz, 1991). That is, the floral organ identity genes, A, B, and C, function to produce four floral organs, sepals, petals, stamens, and carpels by combination of the two genes or singly. The expression of the floral organ identity genes are under precise control in the context of timing and space to secure normal development of the floral anlagen into appropriate floral meristems that contain sufficient cells for the proper patterning of whorled organs. A recent report has revealed that the three flowering time genes, *SOC1*, *AGL24*, and *SVP* are required for the timely activation of B and C floral organ identity genes (Liu *et al.*, 2009). In *soc1 agl24 svp* triple mutants, *SEPALATA 3* (*SEP3*), a *LFY* co-regulator, is ectopically expressed and B and C genes are activated by the interaction of SEP3 and LFY in emerging floral meristems, thus causing defects in floral organ development (Gregis *et al.*, 2006; Liu *et al.*, 2009). It has been shown that SOC1, SVP, and AGL24 redundantly and directly repress *SEP3 in vivo* by interacting with chromatin regulators, TFL2/LHP1 (TERMINAL FLOWER2/LIKE HETEROCHROMATIN PROTEIN1) and SAP18, a member of the SIN3 histone deacetylase complex (Liu *et al.*, 2009). Therefore, it was proposed that these flowering time genes, *SOC1*, *AGL24*, and *SVP,* are required to prevent the precocious expression of B and C genes through the repression of *SEP3* in emerging floral meristems; however, as floral meristems develop, this negative regulation of *SEP3* is gradually derepressed because AP1, the repressor of these three genes, is expressed (Fig. 1).

Additional functions of SOC1

In addition to its role in the integration of multiple flowering signals, recent studies have uncovered other interesting functions of SOC1. For example, SOC1 controls the annual growth habit of *Arabidopsis* (Melzer *et al.*, 2008). Although the *soc1* single mutant shows only a late flowering phenotype, the *soc1 ful* double mutant shows perennial growth phenotypes such as extremely late flowering, formation of aerial rosettes, reiterating reversion to vegetative growth, and secondary growth of stems. Consistent with this, *SOC1* and *FUL* are expressed in procambial strands of the developing inflorescence. Thus, it is likely that *SOC1* and *FUL* act redundantly to suppress the perennial life cycle. Interestingly, *Populus tremuloides MADS-box5* (*PTM5*) gene, a member of the *SOC1* class of MADS box genes in poplar, shows a vascular tissue-specific expression (Cseke *et al.*, 2003). Temporal and spatial expression of *PTM5* suggests that it is seasonally expressed in differentiating primary and secondary vascular cambium. Therefore, the SOC1 class of MADS box genes may be involved in the evolutionary variations between annuals and perennials.

SOC1 also mediates crosstalk between cold sensing and flowering (Seo *et al.*, 2009). In general, flowering is delayed by cool temperatures and accelerated by warm temperatures. *SOC1* is involved in such a fine-tuning mechanism for flowering. A microarray analysis searching downstream targets of SOC1 identified myriads of cold-inducible genes such as *COR* genes harbouring C-repeat-dehydration response elements (CRT/DRE) in their promoters and *CRT/DRE binding factors* (*CBF*s). The ChIP analysis confirmed that SOC1 directly binds to the promoters of *CBF* genes *in vivo*, suggesting that SOC1 negatively regulates the cold response pathway through the direct repression of *CBF*s. By contrast, overexpression of *CBF*s increases the *FLC* transcript level and causes delayed flowering. Such findings reveal the presence of a feedback loop between cold response signalling and flowering regulation for adaptation to changing environments (Seo *et al.*, 2009).

Functional divergence of SOC1

MADS box proteins in Angiosperms have multiple functions regulating diverse developmental processes such as control of flowering time, floral meristem identity, floral organ development, and fruit development. The MADS-box gene family appears to have undergone gene duplication and functional divergence within various angiosperm lineages (Theissen *et al.*, 2000; Irish, 2003). Accumulating evidence suggests that SOC1 has also undergone such functional divergence during evolution. SOC1 is a member of the *SOC1/Tomato MADS-box gene 3* (*TM3*)-clade of MADS box genes and recent studies have identified members of this clade in various species (Decroocq *et al.*, 1999; Cseke *et al.*, 2003; Tadege *et al.*, 2003; Ferrario *et al.*, 2004; Nakamura *et al.*, 2005; Tan and Swain, 2007). *UNSHAVEN* (*UNS*), a *Petunia hybrida* MADS box gene sharing a sequence similarity with *SOC1*, is expressed in vegetative tissues, and down-regulated upon floral initiation and formation of floral meristems (Ferrario *et al.*, 2004). The constitutive expression of *UNS* results in early flowering, ectopic trichome formation on floral organs and the reversion of petals into organs with leaf-like features. Surprisingly, UNS is translocated to the nucleus by interacting with *StMADS11*-like gene which is homologous to AGL24 and SVP, suggesting that the biological function and molecular activity of SOC1 is conserved between *Arabidopsis* and petunia (Ferrario *et al.*, 2004).

One of three *SOC1/TM3*-like genes in *Eucalyptus globulus* ssp. *bicostata*, *ETL* (*Eucalyptus TM3 Like*), is expressed in

both vegetative and reproductive organs, including shoot meristems, roots, and floral organ primordia (Decroocq *et al.*, 1999). Although *SOC1* in *Arabidopsis* is expressed predominantly in the meristem tissues, it is ubiquitously expressed in various tissues, including roots, leaves, shoots, inflorescences, and stems. Probably, *SOC1*/*TM3*-like genes in dicots are widely expressed in various tissues and the regulatory functions of these genes may be more diversified.

In monocotyledons, a gene similar to *SOC1*/*TM3* also regulates floral transition or floral development. *OsSOC1*, one of two *SOC1*/*TM3*-like genes in rice (*Oryza sativa*), is expressed in vegetative tissues, and its expression is elevated at the time of floral initiation, exhibiting similar expression pattern to *Arabidopsis SOC1* (Tadege *et al.*, 2003; Lee *et al.*, 2004). *ZmMADS1*, a *SOC1*/*TM3*-like gene in maize, is co-expressed with *ZmMADS3,* which is a member of *SQUAMOSA* subfamily, in all ear spikelet organ primordia during floral development (Heuer *et al.*, 2001). *TrcMADS1* from *Trillium camtschatcense* (Trilliaceae) is expressed in both vegetative and reproductive organs (Nakamura *et al.*, 2005). Although further research is required to compare their function with that of *SOC1*, their expression patterns and conserved sequences suggest that SOC1/TM3-clade genes play conserved roles but have undergone gradual functional divergence among plant species.

SOC1 as an integrator of multiple flowering signals has been intensively studied for a decade, thus is well understood. However, there are still many more questions to be answered. For example, *SOC1* expressed in the leaves contributes to floral induction, but the molecular mechanism is not clear. It is possible that the SOC1 protein moves to the shoot apex like FT, but that possibility has not been tested yet. SOC1 interacts with many other MADS box proteins including flowering repressors (de Folter *et al.*, 2005). It is likely that SOC1 performs a variety of regulatory functions through combination with other MADS box genes. Understanding the protein networks including SOC1 is necessary to get the full picture of SOC1 function.

Acknowledgements

This work was supported partially by the Korea Ministry of Science and Technology under the National Research Laboratory Program (2006-01952), a grant from Global Research Laboratory Program (2006-03870), a grant (Code no. 20070301034011) from the BioGreen 21 program, Rural Development Administration. We are also grateful to J Yu for drawing the figure and to E Seo for formatting references.

References

Abe M, Kobayashi Y, Yamamoto S, Daimon Y, Yamaguchi A, Ikeda Y, Ichinoki H, Notaguchi M, Goto K, Araki T. 2005. FD, a bZIP protein mediating signals from the floral pathway integrator FT at the shoot apex. *Science* **309,** 1052–1056.

Adrian J, Torti S, Turck F. 2009. From decision to commitment: the molecular memory of flowering. *Molecular Plant* **2,** 628–642.

Amasino R. 2004. Vernalization, competence, and the epigenetic memory of winter. *The Plant Cell* **16,** 2553–2559.

An HL, Roussot C, Suarez-Lopez P, *et al*. 2004. CONSTANS acts in the phloem to regulate a systemic signal that induces photoperiodic flowering of Arabidopsis. *Development 1* **31,** 3615–3626.

Baurle I, Dean C. 2006. The timing of developmental transitions in plants. *Cell* **125,** 655–664.

Blazquez MA, Green R, Nilsson O, Sussman MR, Weigel D. 1998. Gibberellins promote flowering of Arabidopsis by activating the LEAFY promoter. *The Plant Cell* **10,** 791–800.

Borner R, Kampmann G, Chandler J, Gleissner R, Wisman E, Apel K, Melzer S. 2000. A MADS domain gene involved in the transition to flowering in Arabidopsis. *The Plant Journal* **24,** 591–599.

Boss PK, Bastow RM, Mylne JS, Dean C. 2004. Multiple pathways in the decision to flower: enabling, promoting, and resetting. *The Plant Cell* **16,** S18–S31.

Champagne CEM, Goliber TE, Wojciechowski MF, Mei RW, Townsley BT, Wang K, Paz MM, Geeta R, Sinha NR. 2007. Compound leaf development and evolution in the legumes. *The Plant Cell* **19,** 3369–3378.

Coen ES, Meyerowitz EM. 1991. The war of the whorls: genetic interactions controlling flower development. *Nature* **353,** 31–37.

Coen ES, Romero JM, Doyle S, Elliott R, Murphy G, Carpenter R. 1990. *floricaula*: a homeotic gene required for flower development in *Antirrhinum majus*. *Cell* **63,** 1311–1322.

Corbesier L, Vincent C, Jang SH, *et al*. 2007. FT protein movement contributes to long-distance signalling in floral induction of Arabidopsis. *Science* **316,** 1030–1033.

Cseke LJ, Zheng J, Podila GK. 2003. Characterization of PTM5 in aspen trees: a MADS-box gene expressed during woody vascular development. *Gene* **318,** 55–67.

de Folter S, Immink RGH, Kieffer M, *et al*. 2005. Comprehensive interaction map of the Arabidopsis MADS box transcription factors. *The Plant Cell* **17,** 1424–1433.

Decroocq V, Zhu XM, Kauffman M, Kyozuka J, Peacock WJ, Dennis ES, Llewellyn DJ. 1999. A TM3-like MADS-box gene from *Eucalyptus* expressed in both vegetative and reproductive tissues. *Gene* **228,** 155–160.

Ferrario S, Busscher J, Franken J, Gerats T, Vandenbussche M, Angenent GC, Immink RG. 2004. Ectopic expression of the petunia MADS box gene *UNSHAVEN* accelerates flowering and confers leaf-like characteristics to floral organs in a dominant-negative manner. *The Plant Cell* **16,** 1490–1505.

Gocal GFW, Poole AT, Gubler F, Watts RJ, Blundell C, King RW. 1999. Long-day up-regulation of a GAMYB gene during *Lolium temulentum* inflorescence formation. *Plant Physiology* **119,** 1271–1278.

Gocal GFW, Sheldon CC, Gubler F, *et al*. 2001. GAMYB-like genes, flowering, and gibberellin signalling in Arabidopsis. *Plant Physiology* **127,** 1682–1693.

Gregis V, Sessa A, Colombo L, Kater MM. 2006. *AGL24, SHORT VEGETATIVE PHASE*, and *APETALA1* redundantly control *AGAMOUS*

during early stages of flower development in *Arabidopsis*. *The Plant Cell* **18,** 1373–1382.

Gustafson-Brown C, Savidge B, Yanofsky MF. 1994. Regulation of the arabidopsis floral homeotic gene *APETALA1*. *Cell* **76,** 131–143.

Hartmann U, Hohmann S, Nettesheim K, Wisman E, Saedler H, Huijser P. 2000. Molecular cloning of SVP: a negative regulator of the floral transition in Arabidopsis. *The Plant Journal* **21,** 351–360.

Helliwell CA, Wood CC, Robertson M, Peacock WJ, Dennis ES. 2006. The Arabidopsis FLC protein interacts directly *in vivo* with SOC1 and FT chromatin and is part of a high-molecular-weight protein complex. *The Plant Journal* **46,** 183–192.

Hepworth SR, Valverde F, Ravenscroft D, Mouradov A, Coupland G. 2002. Antagonistic regulation of flowering-time gene *SOC1* by *CONSTANS* and *FLC* via separate promoter motifs. *EMBO Journal* **21,** 4327–4337.

Heuer S, Hansen S, Bantin J, Brettschneider R, Kranz E, Lorz H, Dresselhaus T. 2001. The maize MADS box gene *ZmMADS3* affects node number and spikelet development and is co-expressed with *ZmMADS1* during flower development, in egg cells, and early embryogenesis. *Plant Physiology* **127,** 33–45.

Irish VF. 2003. The evolution of floral homeotic gene function. *Bioessays* **25,** 637–646.

Jaeger KE, Wigge PA. 2007. FT protein acts as a long-range signal in Arabidopsis. *Current Biology* **17,** 1050–1054.

Lee H, Suh SS, Park E, Cho E, Ahn JH, Kim SG, Lee JS, Kwon YM, Lee I. 2000. The *AGAMOUS-LIKE 20* MADS domain protein integrates floral inductive pathways in Arabidopsis. *Genes and Development* **14,** 2366–2376.

Lee I, Blazquez MA, Soowal LN, Weigel D. 1997. LEAFY expression and flower initiation in Arabidopsis. *Development* **124,** 3835–3844.

Lee J, Oh M, Park H, Lee I. 2008. SOC1 translocated to the nucleus by interaction with AGL24 directly regulates LEAFY. *The Plant Journal* **55,** 832–843.

Lee JH, Yoo SJ, Park SH, Hwang I, Lee JS, Ahn JH. 2007. Role of SVP in the control of flowering time by ambient temperature in Arabidopsis. *Genes and Development* **21,** 397–402.

Lee S, Kim J, Han JJ, Han MJ, An G. 2004. Functional analyses of the flowering time gene *OsMADS50*, the putative *SUPPRESSOR OF OVEREXPRESSION OF CO 1/ AGAMOUS-LIKE 20* (*SOC1/ AGL20*) ortholog in rice. *The Plant Journal* **38,** 754–764.

Li D, Liu C, Shen L, Wu Y, Chen H, Robertson M, Helliwell CA, Ito T, Meyerowitz E, Yu H. 2008. A repressor complex governs the integration of flowering signals in Arabidopsis. *Developmental Cell* **15,** 110–120.

Liu C, Chen H, Er HL, Soo HM, Kumar PP, Han JH, Liou YC, Yu H. 2008. Direct interaction of AGL24 and SOC1 integrates flowering signals in Arabidopsis. *Development* **135,** 1481–1491.

Liu C, Xi WY, Shen LS, Tan CP, Yu H. 2009. Regulation of floral patterning by flowering time genes. *Development Cell* **16,** 711–722.

Liu C, Zhou J, Bracha-Drori K, Yalovsky S, Ito T, Yu H. 2007. Specification of Arabidopsis floral meristem identity by repression of flowering time genes. *Development* **134,** 1901–1910.

Lohmann JU, Hong RL, Hobe M, Busch MA, Parcy F, Simon R, Weigel D. 2001. A molecular link between stem cell regulation and floral patterning in Arabidopsis. *Cell* **105,** 793–803.

Mandel MA, Gustafson-Brown C, Savidge B, Yanofsky MF. 1992. Molecular characterization of the Arabidopsis floral homeotic gene *APETALA1*. *Nature* **360,** 273–277.

Mathieu J, Warthmann N, Kuttner F, Schmid M. 2007. Export of FT protein from phloem companion cells is sufficient for floral induction in Arabidopsis. *Current Biology* **17,** 1055–1060.

Melzer S, Lens F, Gennen J, Vanneste S, Rohde A, Beeckman T. 2008. Flowering-time genes modulate meristem determinacy and growth form in *Arabidopsis thaliana*. *Nature Genetics* **40,** 1489–1492.

Michaels SD, Ditta G, Gustafson-Brown C, Pelaz S, Yanofsky M, Amasino RM. 2003. AGL24 acts as a promoter of flowering in Arabidopsis and is positively regulated by vernalization. *The Plant Journal* **33,** 867–874.

Molinero-Rosales N, Jamilena M, Zurita S, Gomez P, Capel J, Lozano R. 1999. FALSIFLORA, the tomato orthologue of FLORICAULA and LEAFY, controls flowering time and floral meristem identity. *The Plant Journal* **20,** 685–693.

Moon J, Lee H, Kim M, Lee I. 2005. Analysis of flowering pathway integrators in Arabidopsis. *Plant and Cell Physiology* **46,** 292–299.

Moon J, Suh SS, Lee H, Choi KR, Hong CB, Paek NC, Kim SG, Lee I. 2003. The *SOC1* MADS-box gene integrates vernalization and gibberellin signals for flowering in Arabidopsis. *The Plant Journal* **35,** 613–623.

Mouradov A, Glassick T, Hamdorf B, Murphy L, Fowler B, Marla S, Teasdale RD. 1998. *NEEDLY*, a *Pinus radiata* ortholog of *FLORICAULA/LEAFY* genes, expressed in both reproductive and vegetative meristems. *Proceedings of the National Academy of Sciences, USA* **95,** 6537–6542.

Nakamura T, Song IJ, Fukuda T, Yokoyama J, Maki M, Ochiai T, Kameya T, Kanno A. 2005. Characterization of *TrcMADS1* gene of *Trillium camtschatcense* (Trilliaceae) reveals functional evolution of the *SOC1/TM3*-like gene family. *Journal of Plant Research* **118,** 229–234.

Onouchi H, Igeno MI, Perilleux C, Graves K, Coupland G. 2000. Mutagenesis of plants overexpressing *CONSTANS* demonstrates novel interactions among Arabidopsis flowering-time genes. *The Plant Cell* **12,** 885–900.

Parcy F. 2005. Flowering: a time for integration. *International Journal of Development Biology* **49,** 585–593.

Parcy F, Bomblies K, Weigel D. 2002. Interaction of *LEAFY*, *AGAMOUS* and *TERMINAL FLOWER1* in maintaining floral meristem identity in *Arabidopsis*. *Development* **129,** 2519–2527.

Parcy F, Nilsson O, Busch MA, Lee I, Weigel D. 1998. A genetic framework for floral patterning. *Nature* **395,** 561–566.

Pellegrini L, Song T, Richmond TJ. 1995. Structure of serum response factor core bound to DNA. *Nature* **376,** 490–498.

Putterill J, Robson F, Lee K, Simon R, Coupland G. 1995. The *CONSTANS* gene of Arabidopsis promotes flowering and encodes a protein showing similarities to zinc finger transcription factors. *Cell* **80,** 847–857.

Ruiz-Garcia LFM, Wilkinson M, Haughn G, Salinas J, Martinez-Zapater JM. 1997. Different roles of flowering-time genes in the

activation of floral initiation genes in Arabidopsis. *The Plant Cell* **9,** 1921–1934.

Samach A, Onouchi H, Gold SE, Ditta GS, Schwarz-Sommer Z, Yanofsky MF, Coupland G. 2000. Distinct roles of *CONSTANS* target genes in reproductive development of Arabidopsis. *Science* **288,** 1613–1616.

Schwab R, Palatnik JF, Riester M, Schommer C, Schmid M, Weigel D. 2005. Specific effects of MicroRNAs on the plant transcriptome. *Developmental Cell* **8,** 517–527.

Schwarz S, Grande AV, Bujdoso N, Saedler H, Huijser P. 2008. The microRNA regulated SBP-box genes SPL9 and SPL15 control shoot maturation in Arabidopsis. *Plant Molecular Biology* **67,** 183–195.

Searle I, He YH, Turck F, Vincent C, Fornara F, Krober S, Amasino RA, Coupland G. 2006. The transcription factor FLC confers a flowering response to vernalization by repressing meristem competence and systemic signalling in Arabidopsis. *Genes and Development* **20,** 898–912.

Seo E, Lee H, Jeon J, Park H, Kim J, Noh YS, Lee I. 2009. Crosstalk between cold response and flowering in Arabidopsis is mediated through the flowering-time gene *SOC1* and its upstream negative regulator. *FLC. The Plant Cell* **21,** 3185–3197.

Simpson GG, Dean C. 2002. Arabidopsis, the Rosetta stone of flowering time? *Science* **296,** 285–289.

Sung S, Amasino RM. 2004. Vernalization and epigenetics: how plants remember winter. *Current Opinion in Plant Biology* **7,** 4–10.

Tadege M, Sheldon CC, Helliwell CA, Upadhyaya NM, Dennis ES, Peacock WJ. 2003. Reciprocal control of flowering time by *OsSOC1* in transgenic Arabidopsis and by *FLC* in transgenic rice. *Plant Biotechnology Journal* **1,** 361–369.

Takada S, Goto K. 2003. *TERMINAL FLOWER2*, an Arabidopsis homolog of *HETEROCHROMATIN PROTEIN1*, counteracts the activation of *FLOWERING LOCUS T* by *CONSTANS* in the vascular tissues of leaves to regulate flowering time. *The Plant Cell* **15,** 2856–2865.

Tan FC, Swain SM. 2007. Functional characterization of AP3, SOC1 and WUS homologues from citrus (*Citrus sinensis*). *Physiologia Plantarum* **131,** 481–495.

Theissen G, Becker A, Di Rosa A, Kanno A, Kim JT, Munster T, Winter KU, Saedler H. 2000. A short history of MADS-box genes in plants. *Plant Molecular Biology* **42,** 115–149.

Valverde F, Mouradov A, Soppe W, Ravenscroft D, Samach A, Coupland G. 2004. Photoreceptor regulation of CONSTANS protein in photoperiodic flowering. *Science* **303,** 1003–1006.

Wagner D, Sablowski RWM, Meyerowitz EM. 1999. Transcriptional activation of *APETALA1* by LEAFY. *Science* **285,** 582–584.

Wang JW, Czech B, Weigel D. 2009. miR156-regulated SPL transcription factors define an endogenous flowering pathway in *Arabidopsis thaliana*. *Cell* **138,** 738–749.

Weigel D, Alvarez J, Smyth DR, Yanofsky MF, Meyerowitz EM. 1992. LEAFY controls floral meristem identity in Arabidopsis. *Cell* **69,** 843–859.

Wigge PA, Kim MC, Jaeger KE, Busch W, Schmid M, Lohmann JU, Weigel D. 2005. Integration of spatial and temporal information during floral induction in Arabidopsis. *Science* **309,** 1056–1059.

Wilson RN, Heckman JW, Somerville CR. 1992. Gibberellin is required for flowering in *Arabidopsis thaliana* under short days. *Plant Physiology* **100,** 403–408.

Wu G, Park MY, Conway SR, Wang JW, Weigel D, Poethig RS. 2009. The sequential action of miR156 and miR172 regulates developmental timing in Arabidopsis. *Cell* **138,** 750–759.

Wu G, Poethig RS. 2006. Temporal regulation of shoot development in *Arabidopsis thaliana* by miR156 and its target SPL3. *Development* **133,** 3539–3547.

Yanovsky MJ, Kay SA. 2002. Molecular basis of seasonal time measurement in Arabidopsis. *Nature* **419,** 308–312.

Yoo SK, Chung KS, Kim J, Lee JH, Hong SM, Yoo SJ, Yoo SY, Lee JS, Ahn JH. 2005. *CONSTANS* activates *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* through *FLOWERING LOCUS T* to promote flowering in *Arabidopsis*. *Plant Physiology* **139,** 770–778.

Yu H, Ito T, Wellmer F, Meyerowitz EM. 2004. Repression of AGAMOUS-LIKE 24 is a crucial step in promoting flower development. *Nature Genetics* **36,** 157–161.

Yu H, Xu Y, Tan EL, Kumar PP. 2002. AGAMOUS-LIKE 24, a dosage-dependent mediator of the flowering signals. *Proceedings of the National Academy of Sciences, USA* **99,** 16336–16341.