FLOWERING NEWSLETTER REVIEW

Regulation and function of SOC1, a flowering pathway integrator

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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 determinacy (Liu et al., 2007; 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 MADS-Box gene which is involved in the transition to flowering in mustard (Borner et al., 2000). Subsequently, it has been shown that CO and FLC regulate SOC1 expression via separate regions of the SOC1 promoter (Heworth 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).

**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, ify 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

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**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.
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 SPLs is increased according to growth (Wang et al., 2009; Wu et al., 2009). The overexpression of SPLs 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

Gibberelin (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, API, 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-Rosasles 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 indeterminacy 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 myriad cold-inducible genes such as COR genes harbouring C-repeat-dehydration response elements (CRT/DRE) in their promoters and CRT/DRE binding factors (CBFs). 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 CBFs. By contrast, overexpression of CBFs 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 StMADSII-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 (Decrooq et al., 1999). Although SOCI in Arabidopsis is expressed predominantly in the meristem tissues, it is ubiquitously expressed in various tissues, including roots, leaves, shoots, inflorescences, and stems. Probably, SOCI/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 SOCI/TM3 also regulates floral transition or floral development. OsSOCI, one of two SOCI/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 SOCI (Tadege et al., 2003; Lee et al., 2004). ZmMADS3, a SOCI/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 SOCI. their expression patterns and conserved sequences suggest that SOCI/TM3-clade genes play conserved roles but have undergone gradual functional divergence among plant species.

SOCI 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, SOCI expressed in the leaves contributes to floral induction, but the molecular mechanism is not clear. It is possible that the SOCI protein moves to the shoot apex like FT, but that possibility has not been tested yet. SOCI interacts with many other MADS box proteins including flowering repressors (de Folter et al., 2005). It is likely that SOCI performs a variety of regulatory functions through combination with other MADS box genes. Understanding the protein networks including SOCI is necessary to get the full picture of SOCI 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. 2007030134011) 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.

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