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Review

Is the role of the short-day solely to switch off the CONSTANS in *Arabidopsis*?

Martín Calviño, Hiroshi Kamada, Tsuyoshi Mizoguchi*

Institute of Biological Sciences, University of Tsukuba, Japan
*E-mail: mizoguchi@gene.tsukuba.ac.jp Tel: +81-298-53-6005 Fax: +81-298-53-7723

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Abstract Plants in the genus *Arabidopsis* are facultative LD plants that flower much earlier under LD conditions than SD regimens, with the photoperiod (or LD) pathway contributing to floral acceleration. *LHY* and *CCA1* genes, among other factors, have central roles in the circadian clock of *Arabidopsis*, which plays a key role in measuring day length. *GI* gene mediates the circadian clock and floral activator genes, *CO* and *FT*, to control photoperiodic flowering. *GI* is required to set the peak phase of *CO* expression at the end of the light period under LD conditions, so that the *CO* protein is stabilized and activated by light to increase *FT* expression. However, recent studies have demonstrated that the role of SDs is not solely to switch off CO activity. For example, GI interacts with SPY, a negative regulator of the GA signal. The flowering times of *gi* mutants were still significantly later under SD conditions than LD regimes, which suggests that *GI* has a potential role in accelerating the start of flowering, even under SDs. Over-expression of either *FT* or *TSF* genes caused early flowering, and the acceleration of flowering was enhanced under SDs, suggesting that SDs have an additional role to that in the *LHY*/*CCA1*-*GI*-*CO*-*FT* pathway. In this short review, we discuss the hidden roles of SDs in controlling flowering based on recent studies of the molecular genetics of flowering time in *Arabidopsis*.

Key words: Circadian clock, GIGANTEA (GI), LATE ELONGATED HYPOCOTYL (LHY), photoperiod, short days (SDs).

Flowering in the genus *Arabidopsis* occurs earlier under long-day (LD) conditions and later under short days (SDs; Yanovsky and Kay 2002, 2003). Flowering mutants that delay flowering under LDs, but have rather small effects on SDs, are thought to define a genetic pathway that specifically promotes flowering in response to long photoperiods (Koornneef et al. 1991, 1998). *GI*, *CO*, and *FT* are components of this floral-promoting pathway (Súarez-López et al. 2001; Mizoguchi et al. 2005). *GI* encodes a protein with an unknown biochemical function (Fowler et al. 1999; Park et al. 1999; Mizoguchi et al. 2005); *CO* is a zinc-finger transcription factor that is thought to play a critical role in the regulation of flowering time in response to photoperiod (Putterill et al. 1995; Samach et al. 2000); and *FT* encodes a Raf-kinase inhibitor-like protein that promotes flowering (Kardailsky et al. 1999; Kobayashi et al. 1999). The molecular mechanism responsible for the LD-specific promotion of flowering has been described. The expression of *CO* is regulated at both the transcriptional and protein levels. *CO* transcription is regulated by a circadian clock, with a peak in expression at the end of the day (Súarez-López et al. 2001). This peak in the *CO* mRNA level takes place during the light period under LDs, but during the night period under SDs. At the protein level, CO is regulated through light becoming stabilized by blue or far-red light; however, CO is unstable in darkness, where it is degraded by the proteasome (Valverde et al. 2004). Therefore, the CO protein accumulates only at the end of the light period under LDs, promoting *FT* expression. By contrast, under SDs, *CO* mRNA peaks in darkness, when the protein does not accumulate (Valverde et al. 2004). Coincidence of the circadian clock-controlled peak in *CO* mRNA abundance with the light-mediated activation of the CO protein is proposed to play a central role in the discrimination between LDs and SDs by *Arabidopsis* plants (Searle and Coupland 2004). Based on this model, is the role of SDs in the control of flowering restricted to switching off CO activity only? (Figure 1). Since the early flowering phenotype of *35S*:*FT* plants is enhanced under SD conditions (Kobayashi et al. 1999), mechanisms other than switching off CO activity must be involved in the control of flowering under SDs. In this review, we discuss the evidence supporting the other roles that SDs may have in the control of flowering time in *Arabidopsis*.

Abbreviations: CCA1, CIRCADIAN CLOCK ASSOCIATED 1; CO, CONSTANS; FT, FLOWERING LOCUS T; FLC, FLOWERING LOCUS C; GI, GIGANTEA; LHY, LATE ELONGATED HYPOCOTYL; LL, continuous light; LDs, long days; SDs, short days; SOC1, SUPPRESSOR OF OVEREXPRESSION OF CO 1; SPY, SPINDLY; TOC1, TIMING OF CAB EXPRESSION 1.

The SD pathway enhances the early flowering of plants that over-express either *FT* or *TSF*

CO accelerates flowering under LD conditions by up-regulating *FT* transcription directly (Samach et al. 2000; Súarez-López et al. 2001). Consistent with this finding, loss-of-function of the *CO* gene reduces *FT* expression and causes late flowering under LDs (Kardailsky et al. 1999; Kobayashi et al. 1999). Over-expression of either *CO* (*35S*:*CO*) or *FT* (*35S*:*FT*) causes photoperiod-independent early flowering (Kardailsky et al. 1999; Kobayashi et al. 1999; Onouchi et al. 2000).

Interestingly, the extremely early flowering phenotype of *35S:FT* plants is enhanced under SDs (Kobayashi et al. 1999). The *CO* loss-of-function mutant (*co-1*) also facilitates the early flowering of *35S:FT* plants under LDs. Based on these results, Araki's group suggested that either LDs or *CO* enhances the expression of genes antagonistic to *FT*, such as the *TERMINAL FLOWER 1 (TFL1)* gene (Figure 1; Kobayashi et al. 1999). Recently, a similar conclusion was reached after a study in which *FT* and *TSF* activation-tagged mutants flowered with fewer leaves under SDs than LDs (Michaelis et al. 2005; Table 1). Amasino's group proposed that a slower overall growth rate of the plants under SDs provides additional time for *FT* and *TSF* to act (Michaelis et al. 2005).

GI mediates the circadian clock and *CO-FT* to control photoperiodic flowering

The circadian clock modulates flowering time in response to photoperiod, and many mutations that affect clock components also affect flowering time through changes in the levels of *FT* expression (Figure 1; Hayama and Coupland 2003). Two genes that have been identified as essential to circadian clock function and flowering time regulation are *CCA1* and *LHY*. *CCA1* and *LHY* encode proteins that are highly homologous to MYB transcription factors. Over-expression of either gene caused late flowering under LD conditions (Schaffer et al. 1998; Wang and Tobin 1998), whereas the loss-of-function alleles *CCA1* and *LHY* caused early flowering under SDs (Green and Tobin 1999; Mizoguchi et al. 2002). Both *LHY* and *CCA1* are circadian clock-regulated genes that show a peak in expression soon after dawn, and their over-expression is associated with arrhythmia in leaf movement and expression of the clock-controlled genes (*CCG*s; Schaffer et al. 1998; Wang and Tobin 1998). A third gene essential for circadian clock functioning is the *TIMING OF CAB EXPRESSION 1* (*TOC1*), also called *ARABIDOPSIS PSEUDO RESPONSE REGULATOR 1* (*APRR1*), which encodes a pseudo-response regulator protein (Makino et al. 2000; Matsushika et al. 2000; Strayer et al. 2000). *TOC1* mRNA expression peaks in the evening and mutations of this gene are associated with a short-period phenotype (Millar et al. 1995; Somers et al. 1998; Strayer et al. 2000). These three genes are proposed to define a negative transcriptional feedback loop in which *LHY* and *CCA1* suppress *TOC1* expression during the day and *TOC1* activates *LHY* and *CCA1* expression at night (Alabadi et al. 2001; Mizoguchi et al. 2002).

Under SDs, plants harboring loss-of-function mutations in *LHY* and *CCA1—lhy cca* double mutants—flower earlier than wild-type plants (Table 1; Mizoguchi et al. 2002). The early flowering phenotype of the lhy cca1 double mutant under SDs is associated with an elevated level of *FT* expression (Mizoguchi et al. 2005). The up-regulation of *FT* expression is suppressed in gi mutants, consistent with the regulation of flowering through a *GI-CO-FT* cascade (Súarez-López et al. 2001; Mizoguchi et al. 2005). However, the extremely early flowering phenotype of the lhy cca1 double mutant under SDs is not suppressed completely by *co* or *ft*. Consistently, *lhy cca1* co and *lhy cca1 ft* triple mutants flower earlier than wild-type plants under SDs (Table 1). This suggests that the circadian clock mediates flowering independently of *CO* and *FT*, but *GI* appears to be a key factor in regulating the circadian clock and flowering time (Figure 1). In the *lhy-1* mutant, in which *LHY* is over-expressed, flowering is delayed under LDs. The delayed flowering of *lhy-1* is associated with low expression levels of the *GI*, *CO*, and *FT* genes (Fowler et al. 1999; Súarez-López et al. 2001). Interestingly, the *lhy-1* mutant flowers earlier than wild-type plants under SDs (Súarez-López et al. 2001; Mizoguchi et al. 2005; Table 1). The early flowering of the *lhy-1* mutant under SD conditions is puzzling and further studies are needed to elucidate the mechanisms responsible for this phenotype.

A potential role of *GI* in promoting flowering under SD conditions

Under SDs, *Arabidopsis* plants with strong loss-of-function *gi* alleles flower later than wild-types (Koornneef et al. 1991, 1998; Fowler et al. 1999). Moreover, we found that *35S*:*GI co* and *35S*:*GI ft* plants flower earlier than *co* and *ft* monogenic mutants, respectively, and earlier than the wild-type (Table 1). These results support the idea that *GI* also promotes flowering independently of *CO* and *FT* (Mizoguchi et al. 2005; Figure 1). How does *GI* mediate flowering independently of *CO* and *FT*? Recently, GI was found to interact with SPY, a negative regulator of the GA signal (Tseng et al. 2004). Therefore, a role of GI in the promotion of flowering under SDs may be to prevent SPY from inhibiting the GA signal. This view differs from the one proposed by Tseng et al. (2004), in which

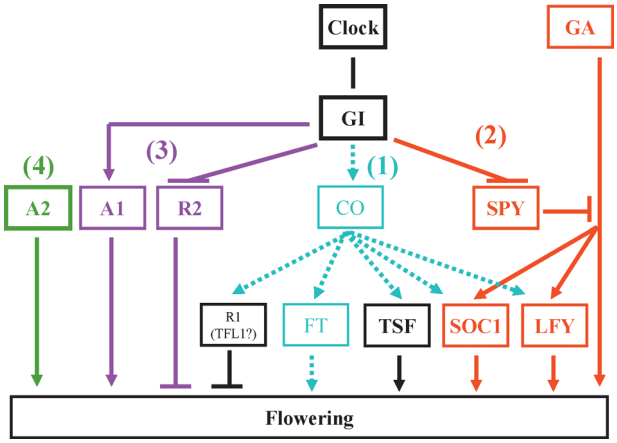

Figure 1. A hypothetical model on the hidden roles of SDs to control flowering. GI is proposed to play dual roles acting within the circadian clock to regulate period length and circadian phase, while also more directly promoting expression of a circadian clock output pathway that includes *CO* and *FT* and promotes flowering (Mizoguchi et al. 2005). CO positively regulates gene expression not only of *FT* but also *SOC1*, *LFY* and *TSF* (Michaelis et al. 2005; Yamaguchi et al. 2005). Pathways that are active under LDs but inactive under SDs are shown in blue (1). CO may up-regulate a floral repressor, indicated with R1 (repressor 1), in LDs but not in SDs. *TFL1* encodes a floral repressor (Bradley et al. 1997; Ohshima et al. 1997), is up-regulated by *CO-ox* (Simon et al. 1996) and was proposed as a candidate for R1 (Kobayashi et al. 1999; Fig. 1). The effect of GI on flowering probably includes other pathways, because *co* mutations only partially suppress the early flowering caused by over-expression of *GI* (Mizoguchi et al. 2005). The GA pathway plays important roles under SDs and SPY is a negative regulator in this pathway. *GI* has been shown to interact with SPY (Tseng et al. 2004). Loss-of-function of *GI* may cause an increase of SPY activity and therefore decrease of SOC1/LFY activities controlled by GA even under SDs (2: red). GI may repress another floral repressor, indicated with R1 (repressor 1), and/or activate another floral activator, indicated with A1 (activator 1) (3: purple). If these pathways shown in purple (3) promote flowering in response to SDs and are inactivated in LDs, *FT*-ox and *TSF*-ox should flower earlier in SDs than LDs. Alternatively, a floral activator, indicated with A2 (activator 2) that is only active under SDs and independent of GI may explain why the *FT-ox* and *TSF-ox* flower earlier under SDs (4: green).

Table 1. Effects of mutations in the photoperiod pathway on flowering time

Flowering phenotype [late>early]	References
gi>WT in SDs	Koornneef et al. 1991; 1998; Fowler et al. 1999
FT-ox in LDs>*FT-ox* in SDs	Kobayashi et al., 1999
FT-ox in LDs>*FT-ox* in SDs[a]	Michaels et al. 2005
TSF-ox in LDs>*TSF-ox* in SDs[a]	
ft>*CO-ox ft*>*CO-ox* in LDs	Onouchi et al. 2000
WT>*lhy or cca1*>*lhy cca1* in SDs	Mizoguchi et al. 2002
co>*GI-ox co*>*GI-ox* in SDs and LDs	Mizoguchi et al. 2005
ft>*GI-ox ft*>*GI-ox* in SDs and LDs	
co>*lhy cca1 co*>*lhy cca1* in SDs and LDs	
ft>*lhy cca1 ft*>*lhy cca1* in SDs and LDs	
gi>*lhy cca1 gi*>*lhy cca1* in SDs	
lhy-1>WT in LDs	Súarez-López et al. 2001;
WT>*lhy-1* in SDs	Mizoguchi et al. 2005

[a] *FT* and *TSF* activation-tagged mutants were used.

SPY was placed in the LD pathway acting upstream from *CO* and *FT*, rather than *GI* acting in GA signaling under SDs (Figure 1).

Strong mutant alleles of *ft* could only partially suppress the early flowering phenotype of the *35S*:*CO* mutant, suggesting that *CO* also regulates flowering via *FT*-independent pathways (Onouchi et al. 2000; An et al. 2004; Figure 1 and Table 1). *CO* regulated flowering through the activation of a second target gene, the *SUPPRESSOR OF OVEREXPRESSION OF CO 1* (*SOC1*)/*AGAMOUS LIKE 20* (*AGL20*; Borner et al. 2000; Lee et al. 2000; Onouchi et al. 2000). *SOC1/AGL20* is regulated by several pathways and is directly activated by *CO* and repressed by the *FLOWERING LOCUS C* (*FLC*) gene at the transcriptional level (Hepworth et al. 2002). *SOC1/AGL20* also mediates signals from the GA pathway, a major flowering pathway under SDs (Borner et al. 2000; Moon et al. 2003). Mutations that disrupt either GA biosynthesis or signaling display altered flowering time (Jacobsen and Olszewski 1993). GA biosynthesis was disrupted in the *Arabidopsis* mutant *ga1-3*, which failed to flower under SDs, and showed a slight delay in flowering under LDs (Wilson et al. 1992; Swarup et al. 1994). Over-expression of *SOC1/AGL20 (soc1-101D)* rescued the non-flowering phenotype of the *ga1-3* mutant under SDs. However, the double mutant *ga1-3 soc1-101D* flowered later than the *soc1-101D* single mutant (Moon et al. 2003). This suggests the presence of additional factors that are regulated by GA under SD conditions. *LFY* may be one of these factors because its expression is also regulated by GA (Blazquez et al. 1998). However, as in the case with *soc1-101D*, *35S*:*LFY* rescued the non-flowering phenotype of the *ga1-3* mutant under SDs, but the *ga1-3 35S*:*LFY* plants flowered later than *35S*:*LFY*, indicating that *LFY* alone could not completely mediate the GA flowering pathway (Moon et al. 2003).

Under SDs, the photoperiod pathway is not active and the GA pathway may still regulate flowering through additional factors other than *SOC1/AGL20* and *LFY*. This raises the possibility that other still unknown factors regulate floral transition independently of the floral integrators *FT*, *SOC1/AGL20*, and *LFY*, especially under SD conditions (Kotake et al. 2003).

Perspectives

Our view on the regulation of flowering time in *Arabidopsis* has changed considerably from the first model proposed at the beginning of the 1990 s (Koornneef et al. 1991). In this model, different floral-promoting pathways executed their action almost independently from each other. However, we are now learning about extremely complex interconnections

among these pathways (Mouradov et al. 2002; Simpson and Dean 2002). How is this interconnected system of floral signals integrated at the level of the *FT*, *SOC1/AGL20*, and *LFY* genes? It seems unlikely that these three floral integrators alone could account for the enormous variation in flowering response within various wild-type accessions of *Arabidopsis*. Do these accessions use the same discriminatory mechanism to differentiate LDs and SDs? Is light stabilization of the *CO* protein the only mechanism able to distinguish between LDs and SDs in all *Arabidopsis* accessions? Since *Arabidopsis* is a facultative LD plant, most of the research related to photoperiodic flowering has focused mainly on the role of the LD pathway rather than that of the SD. In this review, we have tried to highlight the possibility that SDs also have a role in the regulation of flowering. We also believe that *GI* has a role in promoting flowering under SDs. The biochemical function of the *GI* protein is unknown (Fowler et al. 1999; Park et al. 1999; Mizoguchi et al. 2005), but it has been shown recently that GI interacts with SPY, a negative regulator of GA signaling. The GA pathway plays a major role in the promotion of flowering under SDs. This suggests that *GI* plays a role in the acceleration of flowering even under SDs and that the possible repression of SPY by GI may be stronger under SDs than that under LDs (Figure 1). As discussed in the section 1, the *FT-ox* and *TSF-ox* plants flowered earlier in SDs than in LDs (Kobayashi et al., 1999; Michaelis et al. 2005). Loss-of function of *co* also enhanced the early flowering of the *FT-ox* plants under LDs (Kobayashi et al. 1999). It would be very interesting to test the flowering time of the *co-1 35S*:*FT* double mutant under SDs. We predict that SDs would enhance the early flowering of *co-1 35S*:*FT*, with respect to LDs.

One possible scenario is that GI may suppress floral repressor(s) that may have important roles in the delay of flowering under SDs, when the photoperiod pathway is not active (Fig. 1). Alternatively, GI may activate floral activator(s) that may promote flowering under SDs. We believe that the biochemical characterization of the *GI* protein is essential to further understand the promotion of flowering in both the LD and SD pathways.

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