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SI EDITOR'S CHOICE

The control of flowering time by environmental factors

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SUMMARY

The timing of flowering is determined by endogenous genetic components as well as various environmental factors, such as day length, temperature, and stress. The genetic elements and molecular mechanisms that rule this process have been examined in the long-day-flowering plant *Arabidopsis thaliana* and short-day-flowering rice (*Oryza sativa*). However, reviews of research on the role of those factors are limited. Here, we focused on how flowering time is influenced by nutrients, ambient temperature, drought, salinity, exogenously applied hormones and chemicals, and pathogenic microbes. In response to such stresses or stimuli, plants either begin flowering to produce seeds for the next generation or else delay flowering by slowing their metabolism. These responses vary depending upon the dose of the stimulus, the plant developmental stage, or even the cultivar that is used. Our review provides insight into how crops might be managed to increase productivity under various environmental challenges.

Keywords: flowering time, environmental factors, nutrient, ambient temperature, drought, salinity, exogenous hormones, biotic stresses, flowering time.

INTRODUCTION

Studies of the molecular mechanisms involved in controlling flowering time have been extensively reviewed for *Arabidopsis thaliana*, a model long-day (LD) plant (Amasino, 2010; Song *et al.*, 2013b) and for the short-day (SD) model plant rice (*Oryza sativa*) (Jeong *et al.*, 2015; Lee and An, 2015b).

The leaf phloem produces florigen molecule(s) that induce the transition to reproductive development when transferred to the shoot apical meristem (SAM). The first identified molecule was FLOWERING LOCUS T (FT), from Arabidopsis (Corbesier *et al.*, 2007; Jaeger and Wigge, 2007). This molecule is highly conserved in the plant kingdom (for review, Wigge, 2011). In Arabidopsis, *FT* expression is activated by CONSTANS (CO), a zinc finger protein (Putterill *et al.*, 1995). Expression of *CO* is affected by GIGANTEA (GI), which is involved in circadian clock functions (Fowler *et al.*, 1999; Park *et al.*, 1999). The GI-CO-FT module is the main photoperiod pathway in Arabidopsis. This pathway is also widely used in other plants, such as for photoperiod-dependent induction of dormancy in *Populus deltoides* (Bohlenius *et al.*, 2006).

Vernalization, i.e., prolonged exposure to cold, induces flowering in many plant species including Arabidopsis (for review, Song *et al.*, 2012a). This process promotes flowering in the SAM by suppressing expression of *FLOWERING LOCUS C* (*FLC*), which encodes a MADS-box protein (Michaels and Amasino, 1999). As a major inhibitor of flowering, FLC suppresses the expression of transcription factors (TFs) needed for the SAM floral transition.

The autonomous pathway is independent of the photoperiod pathways. Mutants in that pathway alter flowering time irrespective of day length. Several genes in the autonomous pathway have been identified and all regulate *FLC* mostly through RNA binding/processing or chromatin remodeling (for review, Kim *et al.*, 2009).

Gibberellic acid (GA) plays a major role in promoting flowering under non-inductive photoperiod conditions (for review, Davis, 2009). Mutations in GA biosynthesis and signaling affect this timing in Arabidopsis.

MicroRNAs miR156 and miR172 also control the timing of the transitions from vegetative growth to the reproductive phase (for review, Wang, 2014). Whereas miR156 is abundant during young developmental stages but gradually decreases as plants mature, the opposite pattern is exhibited by miR172 (Fornara and Coupland, 2009; Wang *et al.*, 2009). AP2-like TFs that delay flowering are targets of miR172 (Mathieu *et al.*, 2009).

Most crop plants bloom after a lengthy period of vegetative growth because several regulators preferentially inhibit flowering under LD conditions in Summer. Therefore, mechanisms that control this timing in most SD crops are different from those that function in Arabidopsis, an LD plant. For example, in rice, HEADING DATE 3a (Hd3a) and Rice FT 1 (RFT1) are major florigen proteins with important roles in promoting flowering (Tamaki *et al.*, 2007; Komiya *et al.*, 2009). The former acts preferentially under SD whereas the latter operates mainly under LD (Komiya *et al.*, 2009).

In rice, the Arabidopsis CO ortholog Heading date 1 (Hd1) functions upstream of the florigen genes as a positive regulator under SD (Yano *et al.*, 2000). However, the protein delays flowering by suppressing florigen production under non-permissive LD conditions. Early heading date 1 (Ehd1) is another upstream regulatory element that promotes florigen expression (Doi *et al.*, 2004). Homodimerization, as promoted by phosphorylation, is necessary for that function, and an A-type responsive element RR1 binds to Ehd1 to interfere with homodimerization (Cho *et al.*, 2016).

A major LD-preferential inhibitor gene is *Grain number, plant height, and heading date 7* (*Ghd7*), which delays flowering by inhibiting *Ehd1* expression (Xue *et al.*, 2008). The CO-like protein Ghd7 is phosphorylated by Heading date 16 (Hd16), a casein kinase I protein (Hori *et al.*, 2013). Functional *Ghd7* alleles are present in cultivars at low latitude, while non-functional alleles are found in high-latitude cultivars (Xue *et al.*, 2008). Phytochromes and GI control expression of several genes that determine flowering time in rice (Takano *et al.*, 2005; Lee and An, 2015a; Lee *et al.*, 2016).

Although several reviews have been written about research on the control of flowering time (Amasino, 2010; Song *et al.*, 2015), most have focused on molecular genetic mechanisms that are regulated by photoperiod and vernalization. Here, we review the regulation of that process by other environmental factors, such as nutrients, ambient temperature, drought, salinity, exogenous substances, and biotic stresses.

EXTERNAL NUTRIENT STATUS

Excessive amounts or a deficiency of certain elements in the soil can alter flowering time (Robinson and Jones, 1972; Bernier *et al.*, 1993; Wada and Takeno, 2010). In general, poor nutrition promotes flowering. When *Pharbitis nil*, an SD plant, is grown under LD, flowering is induced when nutrients are limited but not when plants are supplied with normal nutrient solutions (Shinozaki *et al.*, 1988; Wada and Takeno, 2010). Although *PnFT1* and *PnFT2*, both orthologs of *FT*, are induced under SD, only expression of the latter is elevated under poor conditions (Wada and Takeno, 2010). These results imply that the induction mechanism by nutrient deficiency differs between SD and LD conditions. Although the effect varies and is ecotype-dependent in Arabidopsis, nutrient contents in those plants also affect flowering time (Zhang and Lechowicz, 1994; Pigliucci *et al.*, 1995). Landsberg *erecta* (L*er*) bloom later when plants are grown with a 1 or 5% Hoagland solution rather than a 25% solution (van Tienderen *et al.*, 1996). In contrast, ecotype L*er* and Colombia (Col) plants flower early when transferred from a high-nutrient to low-nutrient solution (Kolář and Seňková, 2008). This induction of flowering is more significant under SD, and the effect is heightened in younger plants.

Nitrogen regulates a broad range of biological processes (Vidal *et al.*, 2014). In Arabidopsis, nitrate-limiting conditions promote flowering under neutral (12 h/12 h) or SD (8 h/16 h, day/night) conditions (Marín *et al.*, 2011; Liu *et al.*, 2013). This response is independent from the vernalization and autonomous pathways (Marín *et al.*, 2011; Liu *et al.*, 2013). However, transcript levels of GA biosynthesis genes and GA_3 concentrations are increased when nitrate is limited (Liu *et al.*, 2013). Expression of *CO* and *SOC1* is also significantly increased under those conditions, suggesting that lower nitrate levels induce flowering by at least two independent pathways. The blue-light receptor cryptochrome 1 (CRY1) and ferredoxin-$NADP^+$-oxidoreductase have been identified as N-regulated flowering genes by a modified suppression-subtractive hybridization method (Yuan *et al.*, 2016). Mutants in those genes are insensitive to altered N contents, suggesting that this element functions as a signal for modulating blue-light perception and the central circadian clock to interfere with flowering.

Few studies have focused on how other nutrients alter this timing. Flowering in *Trifolium subterraneum* is delayed by a phosphorus (P) deficiency (Rossiter, 1978), but is unaffected by that deficiency if plants are shaded. In the alpine *Gnaphalium supinum*, flowering is advanced by a greater P supply, while the amount of N has no effect (Petraglia *et al.*, 2014). In Arabidopsis, flowering is delayed under low-P conditions but flowering is not altered when plants are given high P (Kant *et al.*, 2011). Transcript levels of *FT*, *LFY*, and *AP1* are reduced and *FLC* expression is increased in plants grown under low P. Because P and GA levels are positively correlated (Jiang *et al.*, 2007), GA appears to play a major role when P is limited.

INTERNAL METABOLIC STATUS

Sucrose promotes flowering in several species (Bernier *et al.*, 1993). In white mustard (*Sinapis alba*), sucrose concentrations in the phloem near the SAM increase rapidly after floral induction (Pryke and Bernier, 1978). Similarly, in Arabidopsis, the rate of leaf sucrose export increases when flowering is induced (Corbesier *et al.*, 1998). Sucrose contents also rise rapidly in Arabidopsis apices under

inductive conditions (King *et al.*, 2008). Analyses of phloem sap from Arabidopsis and white mustard have indicated that the organic C:N ratio increases markedly early during flowering induction (Corbesier *et al.*, 2002). Exogenous application of a low concentration of sucrose partially rescues the late-flowering phenotypes of Arabidopsis mutants, e.g., *co*, *fca*, and *gi* (Roldán *et al.*, 1997), but not *ft* (King *et al.*, 2008). This demonstrates that sucrose functions positively in the floral transition by partially increasing *FT* expression. Arabidopsis *IDD8*, which regulates sucrose synthase expression, also functions in *FT*-dependent flowering (Seo *et al.*, 2011).

In contrast, a high concentration (5–6%) of exogenous sucrose delays flowering in WT Arabidopsis as well as in early- and late-flowering mutants (Zhou *et al.*, 1998; Ohto *et al.*, 2001). This inhibition is most effective during the late vegetative phase when activation of *LFY* expression is delayed. These results indicate that exogenous sugar acts as an inductive signal for flowering at low concentrations, but inhibits flowering by an independent mechanism when concentrations are high.

Starch-metabolism mutants of *A. thaliana,* such as *cam1*, *pgm* (AT5G51820), *adg1* (AT5G48300), and *sex1* (AT1G10760), have higher sugar concentrations in their leaves and exhibit late-flowering phenotypes (Lin *et al.*, 1988; Caspar *et al.*, 1991; Eimert *et al.*, 1995). Trehalose-6-phosphate (T6P) levels are increased significantly during the floral transition in the meristem of wild-type (WT) Arabidopsis and loss of *TREHALOSE-6-PHOSPHATE SYNTHASE 1* (*TPS1*) leads to extremely late flowering (Wahl *et al.*, 2013).

The contents of starch and soluble sugars in Arabidopsis are affected by photoperiod and developmental stage. Mutations of *GRANULE BOUND STARCH SYNTHASE* (*GBSS*) reduce amylose levels and plants exhibit a late-flowering phenotype under LD (Ortiz-Marchena *et al.*, 2014). This indicates that the distribution of sugar and starch is linked to the floral transition.

Lipids accelerate flowering time in *A. thaliana* and *Brassica napus.* Increasing the level of phosphatidylcholine (PC) in the Arabidopsis SAM hastens flowering while a reduction in PC delays that process. The FT protein, homologous to the phosphatidylethanolamine-binding protein in mammalian cells, binds to PC (Nakamura *et al.*, 2014). Wrinkled 1, which is a central regulator of oil synthesis, accelerates flowering in *B. napus* when it is overexpressed (Li *et al.*, 2015).

AMBIENT TEMPERATURE

Ambient temperature influences flowering time by affecting the rate of growth and development throughout the plant life cycle (Jagadish *et al.*, 2016). Analyses of the relationship between historical dates and temperature perturbations have revealed that most plants tend to flower early in response to warmer temperatures (Jagadish *et al.*, 2016). For example, average bloom-dates of *Syringa vulgaris*, *Lonicera tatarica*, *and L. korolkowii* in the western United States have been advanced by 2.0 to 3.8 days per decade, while the mean spring temperature has risen 0.36°C per decade (Cayan *et al.*, 2001). Between 1985 and 2009, this timing in annual crops such as *Avena sativa*, wheat (*Triticum aestivum*), and maize (*Zea mays*), advanced by 1–3 weeks in northern Europe (Olesen *et al.*, 2012).

In Arabidopsis, a rise from 23°C to 27°C upregulates *FT* expression and induces flowering even under non-inductive SD conditions (Balasubramanian *et al.*, 2006; Sanchez-Bermejo *et al.*, 2015). This is also observed in *gi* and *co* mutants, suggesting that *GI* and *CO* are not involved in this thermal acceleration of flowering (Balasubramanian *et al.*, 2006). Instead, a basic helix-loop-helix (bHLH) TF, PHYTOCROME INTERACTING FACTOR4 (PIF4), induces *FT* expression through direct binding to the *FT* promoter in a temperature-dependent manner (Kumar *et al.*, 2012). *Cryptochrome2* (*CRY2*) is also involved in thermosensory responses (Sanchez-Bermejo *et al.*, 2015). The Cape Verde Islands (Cvi-0) ecotype of *A. thaliana*, which carries the hyperactive gain-of-function *CRY2* allele, does not flower early at higher temperatures. However, knock-down of *CRY2* in the Cvi-0 background restores temperature sensitivity, suggesting that CRY2 overrides this thermosensory response (Sanchez-Bermejo *et al.*, 2015). Defects in *FLOWERING LOCUS M* (*FLM*), a MADS-box TF, reduces thermal sensitivity of flowering (Balasubramanian *et al.*, 2006). This gene has at least four different splicing variants (Scortecci *et al.*, 2001). The two main variants – *FLM*-β and *FLM*-δ – are preferentially abundant at a lower (16°C) and higher (27°C) ambient temperature, respectively, and their translated proteins compete for interaction with the floral repressor SHORT VEGETATIVE GROWTH (SVP) to determine flowering time (Posé *et al.*, 2013). Whereas the SVP-FLM-β complex represses flowering at low temperatures, at higher temperatures, the SVP-FLM-δ complex induces flowering via dominant-negative action (Figure 1). Therefore, the ratio between *FLM*-β and *FLM*-δ is a critical factor in regulating this timing in *A. thaliana* (Lutz *et al.*, 2015). Moreover, *FLC* is involved in temperature sensitivity, lengthening the period of the circadian clock and delaying flowering at higher temperatures (Edwards *et al.*, 2006). Wild accessions of Arabidopsis vary tremendously in their response to elevated temperatures, partly because *FLC* has a suppressive effect on flowering (Balasubramanian *et al.*, 2006).

MicroRNA *miR156* and *miR172* are involved in temperature-dependent regulation of flowering (Lee *et al.*, 2010; Kim *et al.*, 2012). Expression of *miR172* is greater at 27°C than at lower temperatures (Cho *et al.*, 2012). The microRNA induces flowering by reducing mRNA levels of

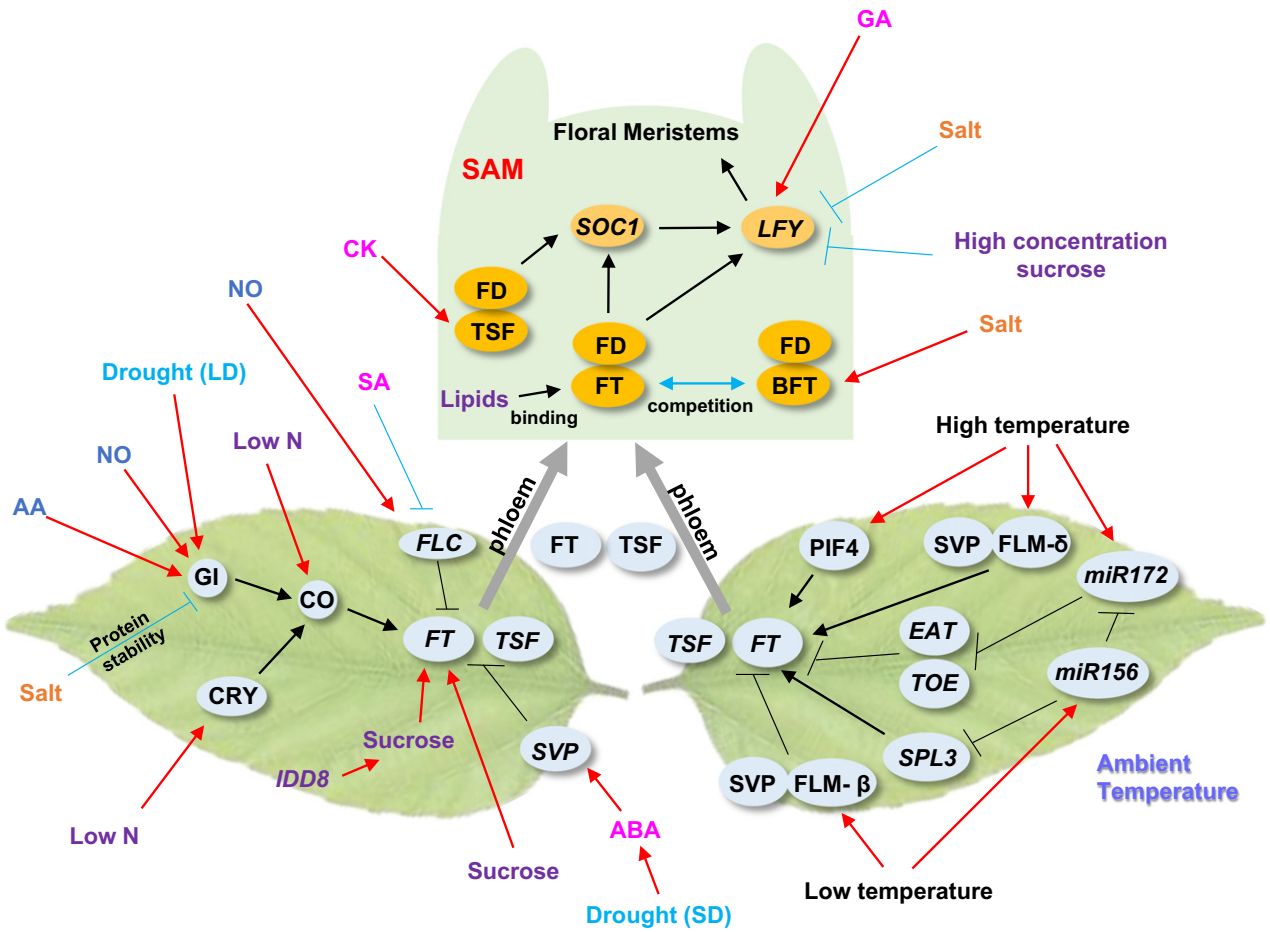

Figure 1. Overview of flowering-time regulation by environmental factors in Arabidopsis.

APETALA2 (*AP2*)-like genes *EAT* and *TOE1*, which act as repressors (Aukerman and Sakai, 2003; Cho *et al.*, 2012). In contrast, miR156 inhibits flowering by suppressing **SPL3**, which is a floral activator at a low temperature (16°C) (Kim *et al.*, 2012) (Figure 1).

Under LD conditions, flowering of WT Arabidopsis and most of its late-flowering mutants is delayed at 16°C. However, mutants defective in *FCA* or *FVE* flower at the same time regardless of temperature (Blázquez *et al.*, 2003). Mutations in *FLC* that are critical to vernalization do not affect the response to ambient temperature, suggesting that the thermosensory pathway is independent of the vernalization pathway (Lee *et al.*, 2008). Another thermosensory gene, *SVP*, acts downstream of *FCA* and *FVE* (Lee *et al.*, 2007). The gene product preferentially binds to the *FT* promoter and suppresses *FT* expression.

The optimum temperature for growing rice is between 25°C and 35°C. When treated at a constant temperature under either SD or LD conditions, those plants bloom later at 23°C than at 27°C (Luan *et al.*, 2009; Song *et al.*, 2012b). Transcript levels of *Ehd1*, *Hd3a*, and *RFT1* are decreased in response to lower temperatures under both day lengths, while those of *Ghd7*, an LD-preferential floral repressor, are increased at lower temperatures only under LD conditions (Luan *et al.*, 2009; Song *et al.*, 2012b) (Figure 2). This implies that *Ghd7* is a major temperature-responsive element when days are long. However, under SD, it is unclear whether *Ehd1* is an immediate responsive gene or if it is a stress-responsive upstream element that controls *Ehd1* expression. The effects of high temperatures on flowering time have not yet been elucidated in rice.

Flowering of WT diploid chrysanthemums, e.g., the SD *Chrysanthemum morifolium* and *C. seticuspe*, occurs later at 30°C than at 20°C (Nakano *et al.*, 2013). This heat-induced delay is accompanied by a reduction in the expression of *FTL3*, a floral inducer that is produced in leaves after SD stimulus and then transported to the shoot tip. Grafting experiments have indicated that the decline in FTL3 transport from leaf to shoot tip at high temperatures is the main reason for this lag in chrysanthemum flowering.

DROUGHT STRESS

Elevating global surface temperatures are causing changes in regional precipitation patterns and levels of atmospheric

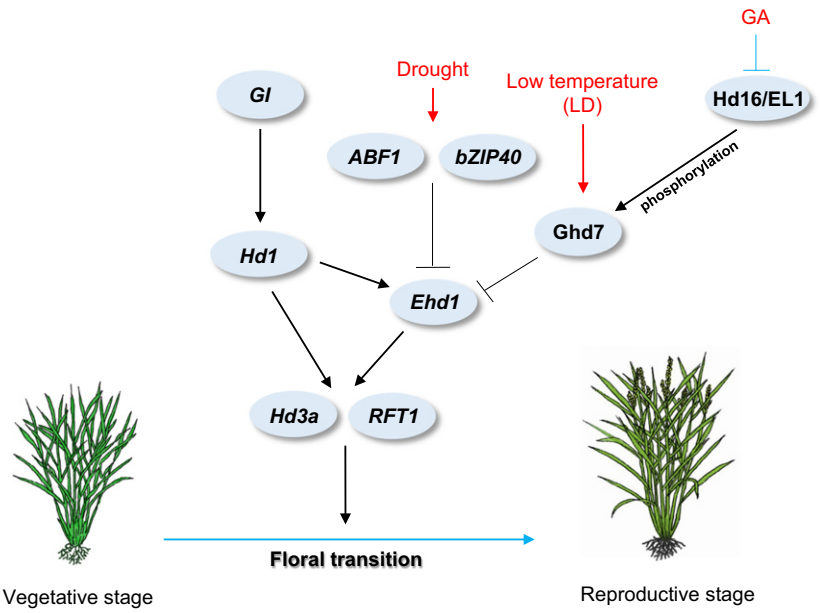

Figure 2. Overview of flowering-time regulation by environmental factors in rice.

moisture (Dore, 2005). These perturbations lead to asymmetric water distributions in plant ecosystems and trigger drought stress that alters aspects of growth and development, including flowering time. One related adaptation is that flowers generally produce seeds before the effects of stress become lethal. For example, drought causes *Brassica rapa* to bloom earlier (Franks *et al.*, 2007) and slightly advances flowering in *Mimulus guttatus* (Jordan *et al.*, 2015).

In Arabidopsis, water deficits promote flowering under LD but delay it under SD, suggesting that this drought-mediated regulation is associated with the photoperiodic flowering pathway. The early-flowering response disappears in *gi* mutants while transcript levels of *GI* are upregulated by drought (Han *et al.*, 2013; Riboni *et al.*, 2013). This indicates that GI plays an important role in the response. Premature flowering is also diminished in double mutants of the florigen genes *ft* and *tsf.* Riboni *et al.* (2013) showed that the response remains in mutants defective in *CO*, which functions downstream of *GI* and is an upstream activator of *FT* and *TSF.* However, Han *et al.* (2013) have reported that, although transcript levels of *CO* are not significantly increased by water stress, the response is impaired in *co* mutants, which do not flower during periods of drought. These conflicting results suggest that displaying a drought response depends on the intensity and duration of the stress as well as the plant developmental stage. In these studies, Riboni *et al.* (2013) used 3-day-old seedlings and maintained a 30% relative soil water content during treatment while Han *et al.* (2013) used mature plants and halted irrigation entirely.

The abscisic acid (ABA)-biosynthesis Arabidopsis mutant *aba deficient1* blooms later than the WT under normal moisture conditions, indicating that ABA promotes flowering. Under drought stress, the *aba* mutant shows a reduced flowering response, implying a role for this hormone. Mutations in the ABA negative regulator Protein Phosphatase 2C (PP2C) gene family, which cause hypersensitized ABA signaling, induce significantly earlier flowering and increased expression of *FT*, *TSF*, and *SOC1* under normal growing conditions (Riboni *et al.*, 2013). Under drought stress, this early-flowering phenotype is nearly absent. These results are evidence that ABA signaling is needed for drought-induced early flowering. Under non-inductive SD conditions, water stress and ABA activate repressors such as *SHORT VEGETATIVE PHASE* to restrict transcription of florigen genes (Riboni *et al.*, 2013) (Figure 1).

The GA biosynthesis mutant *ga1* exhibits delayed flowering under both normal and drought conditions when compared with WT Arabidopsis (Riboni *et al.*, 2013). This suggests that the GA-dependent pathway may not be involved in drought-responsive flowering. *Sapium sebiferum*, a tree species, normally blooms 3–5 years after seeding but will flower within the first year or two if plants are exposed to drought (Yang *et al.*, 2015). During such stress, expression of *GA1* is significantly induced, possibly indicating a role for GA in drought-induced flowering in that species.

In rice, moisture stress delays flowering by most cultivars but can also accelerate flowering in other cultivars (Lilley and Fukai, 1994; Lafitte *et al.*, 2006). This contradictory phenomenon among cultivars is probably due to differences in genetic backgrounds in genes that are part of flowering-time control.

Exposure to drought abolishes the expression of rice florigen genes *Hd3a* and *RFT1* as well as *Ehd1* under both SD and LD (Galbiati *et al.*, 2016). Moreover, the cycling

amplitude of *Hd1* is reduced while that of *Gl* is not significantly affected. Research with mutants has shown that *Hd3a* and *RFT1* expression levels are similar to that of the WT in *hd1* and *gi* mutants during drought periods, but that flowering occurs later in the mutants (Galbiati *et al.*, 2016). These analyses indicate that *Gl* and *Hd1* are not involved in the drought response and that the regulatory pathways differ between Arabidopsis and rice.

A bZIP TF, *Oryza sativa* ABA-responsive element binding factor 1 (OsABF1), suppresses the floral transition by inhibiting *Ehd1* under both SD and LD conditions (Zhang *et al.*, 2016a) (Figure 2). While overexpression of *Ehd1* suppresses the delay in flowering caused by *OsABF1* overexpression, RNAi suppression of *Ehd1* diminishes that alteration in timing associated with *OsABF1.* This indicates that *Ehd1* acts downstream of *OsABF1.* The induction of *OsABF1* by drought and its downregulation, along with that of its closest homologue, *OsbZIP40*, attenuates the drought-related delay in flowering. The OsABF1 factor directly upregulates *OsWRKY104* and overexpression of the latter delays flowering (Zhang *et al.*, 2016a). Because OsWRKY104 acts as a transcriptional activator, a repressor that functions upstream of *Ehd1* is likely a target of the WRKY TF. However, expression analyses of *Ehd1*-upstream repressors, including *Ghd7*, *OsLFL1*, *DTH8*, and *COL4*, have revealed that none is significantly affected by *OsABF1* (Zhang *et al.*, 2016a). Thus other repressors, e.g., *SNB*, *OsIDS1*, or *OsCO3*, could be targets. Alternatively, an unknown repressor might play a critical role during drought periods to delay floral induction in rice.

SALT STRESS

High soil salinity, like many other environmental stresses, affects plant growth and developmental processes, including flowering. In a salt-tolerant halophytic C_4 grass, *Sporobolus virginicus*, salinity can also reduce the number of inflorescences produced (Blits and Gallagher, 1991). Flowering is also delayed in salt-sensitive plants, e.g., wild mustard (*Sinapis arvensis*) and Arabidopsis (Stanton *et al.*, 2000; Achard *et al.*, 2006). This is generally a dose-dependent effect (Li *et al.*, 2007). Whereas mild salt stresses (25 or 50 mM NaCl) delay flowering only slightly, without any significant accompanying growth defect, higher concentrations (≥100 mM) can significantly delay flowering or totally inhibit the floral transition in Arabidopsis.

Salinity-induced floral repression depends mainly on the GA signaling pathway in Arabidopsis. Quadruple-DELLA mutants that lack *GA-INSENSITIVE* (*GAI*), *REPRESSOR OF GA1-3* (*RGA*), *RGA-LIKE 1* (*RGL1*), and *RGL2* bloom earlier than WT plants under salt stress (Achard *et al.*, 2006). Furthermore, such stress decreases transcript levels of *LFY* in the WT while having no effect in those mutant plants.

As a component of the photoperiodic flowering pathway, the Gl-CO-FT module is a critical regulator of flowering time under salt stress. While transcript levels of *CO* and *FT* can be reduced by salinity, the loss-of-function *co-2* mutant does not exhibit a delayed-flowering phenotype under moderate salt stress (Li *et al.*, 2007). The same is true for Arabidopsis *gi* mutants (Kim *et al.*, 2013). Moreover, the salt-induced delay of flowering is completely diminished in *Gl*-overexpression plants while expression of *CO* and *FT* in the transgenics is not reduced in response to stress. Stability of Gl protein is regulated by the 26S proteasome pathway under salt stress, conferring resistance and delaying flowering (Kim *et al.*, 2013).

A member of the NAM, ATAF1/2, CUC2 (NAC) TF family – *NAC WITH TRANSMEMBRANE MOTIF 1-LIKE 8* (*NTL8*) – is induced by salt stress; overexpression of a truncated form that lacks the transmembrane domain leads to delayed flowering due to repression of *FT* (Kim *et al.*, 2007). Salt-induced disassociation of NTL8 from the membrane is considered critical for repressing *FT* expression under salinity (Kim and Park, 2007).

The floral repressor *BROTHER OF FT AND TFL1* (*BFT*) is another important component of salt-induced floral repression. This gene is highly induced by such stress but flowering time of the *bft* mutant is unaffected by the stress (Ryu *et al.*, 2011). The BFT protein binds to FD, a bZIP TF that promotes flowering after forming FT-FD complex. Therefore, salt-induced BFT competes with FT for interactions with FD, interfering with the formation of FT-FD complex, which ultimately delays flowering (Ryu *et al.*, 2014) (Figure 1).

Flowering of rice is delayed by salinity, even at low NaCl concentrations (20–50 mM) (Lutts *et al.*, 1995). This delay is more significant in salt-sensitive varieties. However, the underlying molecular mechanism by which this response occurs in rice is mostly unknown.

EXOGENEOUS SUBSTANCES

Exogenous hormones

Exogenously applied hormones have distinctive roles in controlling plant growth and development, including the flowering process (Davis, 2009). Such treatments mimic the natural influence of endogenous hormones. However, consequences vary according to developmental stage, hormone concentration, and site of application (Kinet *et al.*, 1993).

Flowering is often induced when GA is exogenously applied to non-cold-treated biennial plants or to LD plants grown under SD conditions (Lang, 1957). However, although this occurs in some species, flowering can be delayed in others, indicating that the role of GA in this response is species-specific (Davis, 2009).

In *A. thaliana*, exogenous GA advances flowering while a defect of the GA biosynthesis gene *ga1-3* causes a never-flowering phenotype under SD conditions (Wilson *et al.*,

1992). Plants of white mustard treated with GA also bloom early under non-inductive SD conditions (Bonhomme *et al.*, 2000). An ortholog of *SOC1*, *SaMADS A*, appears to be involved in that response.

Flowering by fruit tree species such as *Citrus sinensis*, *Prunus persica*, *Malus domestica*, and *P. avium* is delayed or inhibited by GA applications (Lord and Eckard, 1987; García-Pallas *et al.*, 2001; Lenahan *et al.*, 2006; Zhang *et al.*, 2016b), possibly because this treatment stimulates vegetative growth and changes endogenous hormone contents (Zhang *et al.*, 2016b). However, whereas the GA biosynthesis inhibitor paclobutrazol suppresses flowering of *Lolium temulentum* and *A. thaliana* (King *et al.*, 2006; Jung *et al.*, 2012), it advances flowering in *Mangifera indica* (Kulkarni, 1988).

In rice, exogenous GA suppresses expression of *Heading date 16* (*Hd16*)/*Early flowering 1* (*EL1*), which encodes a casein kinase I protein (Dai and Xue, 2010) that specifically phosphorylates the DELLA protein SLR1, a repressor of GA signaling (Sasaki *et al.*, 2003). This kinase protein can also activate other floral repressors, e.g., Ghd7 and OsPRR37, via phosphorylation (Hori *et al.*, 2013; Kwon *et al.*, 2015) (Figure 2). However, the effect of GA treatment on flowering time in rice has not been reported.

Exogenous auxin influences the flowering of various species, and has a dual function in regulating that process in the SD *Xanthium pensylvanicum* (Salisbury, 1955). Although this hormone can inhibit flowering if applied before floral induction, it increases the rate of bud development if applied after floral induction. The specific auxin concentration is also a critical factor. For example, flowering of *Ananas comosus* is advanced in response to a low level of α-naphthaleneacetic acid (NAA), such as 0.001–0.006%, but is delayed at higher NAA concentrations, i.e., 0.0–0.1% (Clark and Kerns, 1942).

In *A. thaliana*, exogenous auxin induces flowering (Shimada *et al.*, 2005). However, defects in *AUXIN RESPONSE FACTOR 2* (*ARF2*), an auxin-mediated TF, delay flowering (Okushima *et al.*, 2005). This gene decreases transcript levels of two paralogous GATA TFs – *NITRATE-INDUCIBLE, CARBON-METABOLISM INVOLVED* (*GNC*) and *GNC-LIKE* (*GNL*) – that act as floral repressors (Richter *et al.*, 2013; Behringer and Schwechheimer, 2015).

Exogenously applied salicylic acid (SA) induces flowering in *Lemma gibba* under a non-inductive photoperiod (Cleland and Ajami, 1974). In Arabidopsis, SA-deficient mutants bloom late under both LD and SD due to increased expression of *FLC* (Martínez *et al.*, 2004). Whereas exogenous application of SA accelerates flowering by WT Arabidopsis, this treatment does not affect flowering time of *nahG* plants that are defective in SA accumulation (Martínez *et al.*, 2004).

Application of methyl jasmonate (MeJA) to vernalization-insensitive *Triticum aestivum* delays flowering in conjunction with a significant downregulation of *TaFT1* expression, which suggests that MeJA has a role in modulating flowering time in that crop (Diallo *et al.*, 2014). In Arabidopsis, jasmonic acid (JA) also appears to delay flowering. Mutants in bHLH TF genes that repress JA signaling exhibit late flowering whereas the JA receptor mutant *coi1* blooms early (Song *et al.*, 2013a).

Exogenous applications of ABA alter flowering time in several plant species (for review, Conti *et al.*, 2014). Because this hormone is accumulated during periods of drought, leading to early flowering, ABA signaling might underpin that response. Arabidopsis mutants defective in ABA biosynthesis bloom late due to reduced expression of *FT* and *TSF*, supporting a positive role for ABA in flowering (Riboni *et al.*, 2013). However, it is regarded as an inhibitor of flowering when exogenously applied to Arabidopsis (Wang *et al.*, 2013). These reported contradictions may be due to differences in ABA concentrations or experimental methods.

A single 8-h application of 5 or 50 μM benzylaminopurine (BAP) through the roots promotes flowering of 7-week-old Arabidopsis plants grown under non-inductive SD (D'Aloia *et al.*, 2011). Five-week-old plants are less sensitive. Such treatment activates critical flowering-time genes, *TWIN SISTER OF FT* (*TSF*) and *SOC1* downstream, within the first 32 h, demonstrating that the florigenic effect of BAP is rapid. Because mutants defective in either gene do not respond to BAP, it appears that both are necessary for initiating flowering in response to the application. Arabidopsis mutants with an increased amount of endogenous cytokinin show an early-flowering phenotype (He and Loh, 2002). In contrast, flowering is delayed in plants overexpressing CYTOKININ OXIDASE/DEHYDROGENASE (CKX), which reduces levels of active cytokinin (Werner *et al.*, 2003). All of these results demonstrate that cytokinin induces flowering in Arabidopsis.

Exogenous chemicals

Nitric oxide (NO) production is increased by various stimuli, such as high salt, drought, and pathogens (Lamattina *et al.*, 2003). Exogenous application of NO represses the floral transition in Arabidopsis (He *et al.*, 2004). Whereas a mutant defective in NO production flowers early, NO-overexpression plants flower late. Expression of *CO* and *GI* is reduced by NO, while that of *FLC* is increased, indicating that NO regulates the photoperiod and autonomous pathways (Figure 1).

Application of nitrogen dioxide (NO_2) accelerates flowering without changing the number of rosette leaves in Arabidopsis because this treatment also increases the rate at which those leaves appear (Takahashi and Morikawa, 2014). Being able to achieve a maximum number of leaves sooner in the growing season may contribute to extending crop range.

Elevated levels of atmospheric carbon dioxide (CO_2) reduce transpiration and enhance photosynthesis, resulting in higher concentrations of sugars and carbohydrates in leaves of Arabidopsis and woody species (Curtis and Wang, 1998; Teng *et al.*, 2006). This increase often changes the timing of flowering and the size of a plant at which flowering occurs (Springer and Ward, 2007). For example, elevated concentrations accelerate flowering in rice, *Hordeum vulgare*, *Petunia hybrida*, and *Solanum tuberosum*, but delay it in *Sorghum bicolor.* However, experiments with Arabidopsis, maize, and *Glycine max* have not shown consistent responses, probably due to differences in growing conditions and choice of cultivars. Therefore, further studies are needed to understand whether the effect of CO_2 on flowering time is mediated mainly by altering sugar concentration or due to additional mechanisms that operate under elevated CO_2 conditions.

Heavy metal contamination by atmospheric particulates is of global concern because large amounts of metals can be rapidly transported long distances and cause adverse environmental consequences (for review, Csavina *et al.*, 2012). For example, the frequency of dust storms has increased significantly in recent decades throughout many parts of the world. Dust emitted from mining operations may also mobilize high levels of heavy metals into the environment. Their effects on flowering time vary among species. For *Senecio vulgaris*, *Andryala integrifolia*, *and Hypochoeris radicata*, high concentrations of Cu in the soil delay flowering by one to 10 weeks (Brun *et al.*, 2003). However, such exposure does not significantly affect this timing in *Poa annua.*

Ascorbic acid (AA, or vitamin C) is a co-factor for many enzymes and it mediates detoxification of reactive oxygen species (ROS) (Smirnoff and Wheeler, 2000). These ROS are generated by various stresses such as heavy metals (Michalak, 2006). Early studies proposed that AA has a role in controlling flowering time (Bharti and Garg, 1970). Exogenous application of L-galactono-γ-lactone (GalL), a precursor of AA, to *B. rapa* delays flowering under LD but promotes it under SD (Daniela and De Tullio, 2007). In *A. thaliana*, flowering is deferred when GalL is sprayed under LD conditions (Attolico and De Tullio, 2006). Mutants of Arabidopsis defective in AA biosynthesis bloom earlier than the WT under both SD and LD, indicating that AA inhibits the floral transition regardless of day length (Kotchoni *et al.*, 2009). Under either SD or LD, an AA deficiency also promotes the expression of major genes in the photoperiodic flowering pathway, including *GI*, *CO*, and *FT* (Kotchoni *et al.*, 2009). Ascorbic acid is also crucial to the biosynthesis of GA, ABA, and ethylene (Barth *et al.*, 2006). Therefore, AA and these plant hormones orchestrate flowering time in plants.

Another antioxidant, glutathione (GSH), also affects flowering time. Exogenous treatment of Arabidopsis with L-buthionine sulfoximine, a specific inhibitor of GSH biosynthesis, under LD promotes flowering, but this effect is diminished by exogenous GSH treatment (Ogawa *et al.*, 2001).

BIOTIC STRESSES

Diseases linked to microbial pathogens such as fungi, bacteria, and viruses affect many aspects of plant growth and development (for review, Banday and Nandi, 2015). Infections by the bacteria *Pseudomonas syringae* and *Xanthomonas campestris* accelerate flowering in Arabidopsis (Korves and Bergelson, 2003), but plants treated with a low dose of *P. syringae* bloom earlier than those exposed to a medium or high dose. Analyses of 83 different *A. thaliana* ecotypes have revealed that those susceptible to the fungal pathogen *Fusarium oxysporum* flower comparatively early while resistant ecotypes bloom relatively late (Lyons *et al.*, 2015). The effects of *Verticillium* spp. infection on flowering time are also dependent on *A. thaliana* ecotypes. There, the susceptible ecotype 'Col' shows early flowering while the tolerant 'C-24' flowers late upon infection (Veronese *et al.*, 2003).

Transcription of *FLD* is activated by inoculation with *P. syringae*; defects in that gene lead to a failure to induce systemic acquired resistance, or SAR (Singh *et al.*, 2013). Because *FLD* stimulates flowering by repressing *FLC* expression (He *et al.*, 2003), pathogen-induced promotion of flowering also appears to be mediated by *FLD* (Banday and Nandi, 2015). A mutant defective in SA biosynthesis blooms early under LD (Wang *et al.*, 2011). Expression of the related gene is upregulated by pathogen infection or exogenous SA treatment, both of which provide long-term protection against subsequent infections due to SAR (Jagadeeswaran *et al.*, 2007). Although these results show that SAR can inhibit flowering, further studies are required to understand the relationship between timing of that event and pathogen infections.

Natural soil microbes also affect flowering (Wagner *et al.*, 2014). Panke-Buisse *et al.* (2015) have isolated soil microbiomes that cause early- or late-flowering of *B. rapa* and *A. thaliana* grown under poor nutrient conditions. A high level of reproducibility of microbes alters flowering time because it improves the soil composition. Endophytes also affect timing in Arabidopsis (Poupin *et al.*, 2013). For example, inoculation with *Burkholderia phytofirmans*, a plant growth-promoting rhizobacteria, advances flowering by increasing *AP1* expression.

Some plants depend on insects or other animals for pollination and successful reproduction. For them, pollinator availability often alters flowering time. Blooming of *Fouquieria splendens* is synchronized with the abundance of pollinators such as hummingbirds and bees (Waser, 1979). In the perennial *Arabidopsis lyrata*, timing is also affected by pollinator-mediated selection (Sandring and Ågren,

2009). Recent global warming has coordinately accelerated the dates of first appearance by pollinators as well as the flowering time of their hosts (Hegland *et al.*, 2009).

Herbivores act as a selective pressure. For *Tripolium vulgare* (Riber Albrectsen, 2000) and *Vaccinium hirtum* (Mahoro, 2002), their early flowering is advantageous because it enables those plants to avoid predators. By contrast, late flowering benefits plants such as *Helianthus annuus* (Pilson, 2000) and *Ferocactus* species (McIntosh, 2002) because that timing lets them avoid their predators.

CONCLUSIONS

In higher plants, flowering time is tightly manipulated by interactions among several internal elements and diverse external factors. To maximize reproductive success, plants must form flowers under the most suitable environmental conditions. The timing of flowering affects total yields as well as fruit quality. When conditions are unfavorable, plants tend to alter this timing to ensure that seed is produced for the next generation. Flowering time can be influenced by various environmental factors such as photoperiod, temperature, availability of water and nutrients in the soil, exogenous chemical compounds, microbes, and pollinator availability. Understanding the regulatory mechanisms by which these factors modulate flowering time is very helpful when developing management strategies for plant production.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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