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FLOWERING NEWSLETTER REVIEW

The link between flowering time and stress tolerance

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Abstract

Evolutionary success in plants is largely dependent on the successful transition from vegetative to reproductive growth. In the lifetime of a plant, flowering is not only an essential part of the reproductive process but also a critical developmental stage that can be vulnerable to environmental stresses. Exposure to stress during this period can cause substantial yield losses in seed-producing plants. However, it is becoming increasingly evident that altering flowering time is an evolutionary strategy adopted by plants to maximize the chances of reproduction under diverse stress conditions, ranging from pathogen infection to heat, salinity, and drought. Here, recent studies that have revealed new insights into how biotic and abiotic stress signals can be integrated into floral pathways are reviewed. A better understanding of how complex environmental variables affect plant phenology is important for future genetic manipulation of crops to increase productivity under the changing climate.

Key words: Abiotic stress tolerance, *Arabidopsis*, drought, flowering time, GIGANTEA, heat, pathogen defence, plant hormones.

Introduction

Flowering is a critical life history trait that ensures seed production required for the survival of species. Over recent years, enormous scientific progress has been made to understand the molecular basis of this trait. Briefly, flowering, or, more broadly, the transition from vegetative to reproductive growth, requires genetic and epigenetic reprogramming and reallocation of metabolic and biochemical resources throughout the plant. As reviewed in detail elsewhere (Andrés and Coupland, 2012; Blümel *et al.*, 2014), flowering is regulated by an elaborate network of genetic pathways responsive to endogenous and environmental stimuli. This ensures that the transition to reproduction coincides with favourable conditions. In *Arabidopsis thaliana*, the vernalization, photoperiod, circadian clock, sugar budget, age, thermosensory, autonomous, and gibberellin (GA) pathways converge on a few floral integrator genes that promote flowering. *FLOWERING LOCUS T* (*FT*), a key floral integrator gene, encodes a component of the mobile signal 'florigen' that activates floral meristem identity genes. During long days, *FT* is up-regulated by the transcription factor CONSTANS (CO). The FT protein travels from the leaf to the meristem to initiate flowering via the activation of meristem identity genes such as *LEAFY* (*LFY*) and *APETALA1* (*AP1*), which then change the fate of the shoot apical meristem from vegetative to floral. The transcription factor FLOWERING LOCUS C (FLC) antagonizes the GA and photoperiod pathways by repressing floral promoters such as *FT* and *SUPPRESSOR OF CONSTANS* (*SOC1*). *FLC* transcription is epigenetically down-regulated via the vernalization or autonomous pathways at ambient temperatures, allowing flowering to occur during long days.

The research on *Arabidopsis* has greatly illuminated our current understanding of the processes involved in flowering. However, it is also becoming evident that plants have evolved diverse mechanisms to regulate flowering. For instance, FLC seems to be specific to *Brassica* species, and an FLC homologue does not seem to exist in cereals (Cockram *et al.*, 2007). Rather, the flowering activator VRN-1 is the key vernalization response gene in cereals (Greenup *et al.*, 2009; Diallo *et al.*, 2012). Varieties within crop species also have different photoperiod sensitivities that have arisen via adaptation to different growth environments or through breeding (Coles *et al.*, 2010; Gómez-Ariza *et al.*, 2015).

Stress-regulated flowering is not formally recognized as a floral transition pathway *per se*. However, as discussed in this review, a number of studies suggest that both biotic and abiotic stress factors play key roles in controlling the transition to flowering (Fig. 1). Here we will review the emerging evidence showing that diverse biotic and abiotic stresses alter flowering time in plants and examine the roles of flowering time regulators in the stress response. Genetic mechanisms underlying cross-talk between the stress response and flowering time and the co-evolution of these traits will also be considered. Finally, potential agricultural implications of the complex interplay between flowering and stress will be discussed.

Drought stress and flowering time

Drought is an abiotic stress factor that affects many regions of the world. Drought causes an early arrest of floral development and leads to sterility (Su *et al.*, 2013). To ensure survival during drought stress, plants often accelerate the flowering process, and this response is known as 'drought escape' (Sherrard and Maherali, 2006; Franks *et al.*, 2007; Bernal *et al.*, 2011; Franks, 2011). The related concept 'drought avoidance' refers to the condition where the plant reduces water loss to prevent dehydration (Kooyers, 2015). Emerging evidence suggests that floral pathways play key roles in modulating drought tolerance. In *Arabidopsis*, drought stress accelerates flowering under long days but delays flowering under short days. The photoperiodic flowering time gene GIGANTEA (GI), which promotes flowering via the photoperiod and circadian pathways, is a key regulator of the drought escape response (Fig. 2) (Riboni *et al.*, 2013). During long days, GI degrades the CYCLING DOF FACTOR (CDF) transcriptional repressors of the floral integrator genes CO and FT, thereby allowing activation of these integrators. GI can also directly activate *FT* by binding to its promoter (Sawa and Kay, 2011).

Under long days, drought stress triggers transcriptional induction of the floral promoters *FT* and *TWIN SISTER OF FT* (*TSF*) in a manner dependent on GI and the plant stress hormone abscisic acid (ABA). Under short days, drought and ABA are thought to activate floral repressors, inhibiting the transcription of *FT* and *TSF* (Riboni *et al.*, 2013).

Natural variability within a species for flowering time and stress responses can be utilized to dissect the association between these two plant processes. The relationship between flowering time and drought stress has been studied in near isogenic and recombinant inbred lines constructed between two *Arabidopsis* ecotypes that differ in flowering times and in their response to drought stress. When exposed to mild drought stress, early flowering lines suffered a greater fitness cost than late flowering lines, which were eventually able to recover (Schmalenbach *et al.*, 2014). In contrast, within 234 'summer' (non-vernalization-requiring) *Arabidopsis* ecotypes, early flowering correlated with higher biomass when plants were exposed to terminal drought stress, suggesting an adaptation towards the drought escape response in these ecotypes (Kenney *et al.*, 2014). Therefore, premature flowering, which ensures survival under severe terminal stress, may reduce plant yield under mild chronic stress conditions (Schmalenbach *et al.*, 2014). In addition, high water use efficiency is positively correlated with late flowering time in *Arabidopsis*, suggesting that ecotypes that favour the drought avoidance strategy can survive longer under drought (Kenney *et al.*, 2014). In rice, Grain Number, Plant Height and Heading Date 7 (Ghd7), a CCT domain CONSTANS-like protein that acts as a negative regulator of heading date under long days (Xue *et al.*, 2008), also regulates drought tolerance (Weng *et al.*, 2014).

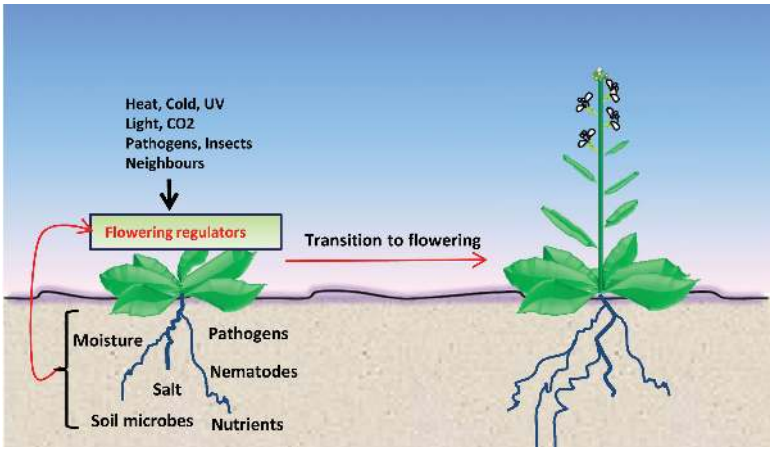

Fig. 1. Flowering under stress. Flowering time is altered by a number of biotic (e.g. pathogens, insects, soil microbes, and neighbouring plants) and abiotic stress factors (e.g. drought, cold, salt, and nutrients) that converge on endogenous floral regulators. See text for additional details.

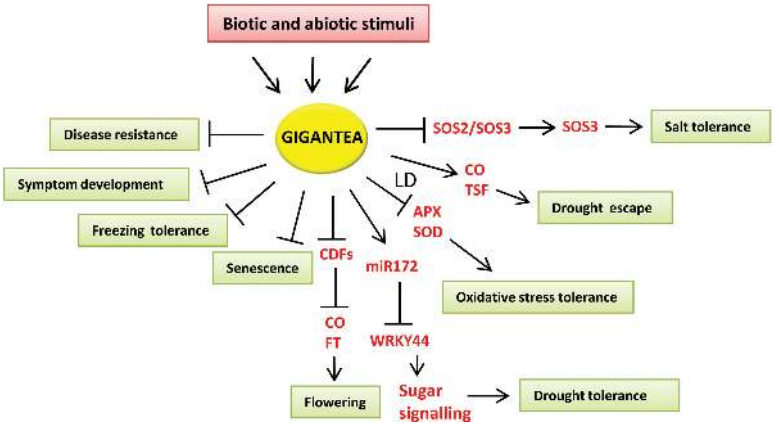

Fig. 2. Multiple functions of GIGANTEA in the regulation of biotic and abiotic stress tolerance. See text for additional details.

Salt stress and flowering time

Salinity substantially delays flowering time in *Arabidopsis* (Kim *et al.*, 2007), and several flowering regulators that mediate this response have been identified. Salt delays flowering in a process dependent on DELLA proteins acting as negative regulators of GA signalling and the plant hormone ethylene (Achard *et al.*, 2006). Salt stress suppresses the expression of *CO* and *FT*, contributing to the delay in flowering (Kim *et al.*, 2007; Li *et al.*, 2007). In addition, the salinity-induced delay in flowering time appears to be dependent on the floral repressor BROTHER OF FT AND TFL1 (BFT), as the delay observed in wild-type plants was not evident in *bft* mutants (Ryu *et al.*, 2011). BFT interacts with FD, a bZIP transcription factor that positively regulates flowering. The BFT–FD interaction most probably interferes with the interaction known to occur between FT and FD, leading to the delay in flowering under saline conditions (Ryu *et al.*, 2014). More recently, the *Arabidopsis* CYCLIN-DEPENDENT KINASE G2 (CDKG2) has been shown to be a regulator of both salinity stress and flowering time (Ma *et al.*, 2015).

GI is also a pivotal component of salt stress tolerance in plants (Fig. 2). *gi* mutants show enhanced salt tolerance, whereas GI-overexpressing plants show increased salt sensitivity, suggesting that GI negatively regulates salinity tolerance. The mechanism of GI action in salt tolerance has recently been elucidated in *Arabidopsis*. Under normal growth conditions, GI interacts with SALT OVERLY SENSITIVE 2 (SOS2), a protein kinase that activates the Na^+/H^+ antiporter SOS1 to promote Na^{2+} export and salt tolerance. The GI–SOS2 interaction prevents the interaction between SOS2 and SOS1 that is required for the activation of salt stress responses. During salt stress, GI is degraded by the proteasome and this enables SOS2 to interact with SOS3 and to form a protein kinase complex that phosphorylates and activates SOS1 (Kim *et al.*, 2013).

PHYTOCHROME AND FLOWERING TIME 1/MEDIATOR 25 (PFT1/MED25), a subunit of the plant Mediator complex that acts to regulate flowering time independently of photoperiod (Cerdán and Chory, 2003), also regulates the response to several abiotic and biotic stresses, including salt stress. *med25* loss-of-function mutants are not only late flowering (Cerdán and Chory, 2003) but also show increased sensitivity to salt stress, and increased drought and disease tolerance (Kidd *et al.*, 2009; Elfving *et al.*, 2011). PFT1/MED25 interacts with DREB2A (drought responsIVE element binding protein 2A), an AP2/ERF transcription factor that regulates stress-responsive gene expression as well as flowering (Elfving *et al.*, 2011). Interactions between PFT1/MED25 and AP2/ERFs (e.g. ERF1) and basic helix–loop–helix (bHLH) transcription factors (e.g. MYC2) that may be critical for the function of PFT1/MED25 in disease tolerance, have also been identified (Çevik *et al.*, 2012). Similarly another Mediator subunit, MED18, regulates both flowering time (Zheng *et al.*, 2013) and plant defence against pathogens (Lai *et al.*, 2014), suggesting that the multiple subunits of the plant Mediator complex act to integrate developmental and stress signals.

Heat stress and flowering time

Heat is another abiotic stress factor that can have a dramatic effect on flowering time. Heat stress is expected to be increasingly problematic in the face of climate change. In *Arabidopsis* (e.g. the ecotype Col-0), elevated ambient temperatures accelerate flowering while cooler temperatures delay flowering. Key modulators of temperature-regulated flowering are *FLOWERING LOCUS M* (*FLM*) and *SHORT VEGETATIVE PHASE* (*SVP*). Under cooler ambient temperatures, flowering activators are repressed by a complex containing SVP and FLM. At warmer temperatures, SVP stability is decreased and inactive forms of the complex accumulate due to alternative splicing of *FLM*, leading to derepression of floral activators (Lee *et al.*, 2013; Posé *et al.*, 2013). In *Arabidopsis*, a negative correlation was found between heat response and flowering time (Bac-Molenaar *et al.*, 2015). Heat stress delays flowering in chrysanthemum, a short-day plant. Heat stress-induced delayed flowering in this species correlates with reduced expression of *FLOWERING LOCUS T-like 3* (*FTL3*), an FT homologue (Nakano *et al.*, 2013). It should also be noted that the effect of heat stress can be dependent on not only the exact temperature but also the duration of heat stress and whether the heat stress occurs

gradually or suddenly (Yeh *et al.*, 2012). The effect of heat stress that coincides with flowering due to climate change will be discussed below.

In economically important cereal crops such as wheat and barley, high temperatures promote flowering under long days, but delay inflorescence development under short days. In barley, short days and high temperatures are accompanied by elevated expression of the MADS box flowering repressor HvODDSOC2, which may be related to the FLC gene family (Hemming *et al.*, 2012). However, the mechanism underlying accelerated flowering under long days and high temperatures is unclear. In rice, a short-day plant, heat stress occurring during flowering can cause sterility. A quantitative trait locus (QTL) conferring early flower opening during the day in the wild rice *Oryza officinalis* significantly contributes to escape from heat stress and has the potential to be utilized in rice breeding (Hirabayashi *et al.*, 2014).

Cold stress and flowering time

Cold acclimation is a process whereby plants increase their freezing tolerance after exposure to a short period of low but non-freezing cold temperatures. Whereas cold acclimation can be induced at 12 °C, vernalization requires lower temperatures, suggesting that the cold acclimation and vernalization pathways are independent (Bond *et al.*, 2011). Exposure to long-term cold promotes flowering in vernalization-sensitive *Arabidopsis* plants via the vernalization pathway. In contrast, exposure to short-term cold or overexpression of cold-responsive genes delays flowering by activating *FLC* (Seo *et al.*, 2009; Jung *et al.*, 2012, 2013).

Similarly to its effect on drought and salt stress tolerance, the flowering gene GI regulates responses to cold in *Arabidopsis*. *gi* mutants exhibit increased freezing tolerance accompanied by up-regulation of cold-responsive genes (Fig. 2). Epistasis analyses revealed that the freezing tolerance phenotype in the *gi* mutants is dependent on CDF transcriptional repressors. GI is also thought to regulate oxidative stress and hypocotyl growth responses via CDF, as mutation of *CDF* in the *gi* background (*gi cdf*) rescued these phenotypes (Fornara *et al.*, 2015). Interestingly, an earlier study reported that an independent loss-of-function *gi* mutant had increased sensitivity to freezing tolerance (Cao *et al.*, 2005), suggesting that differences may exist between mutants or ecotype backgrounds.

Flowering time genes HIGH EXPRESSION OF OSMOTICALLY RESPONSIVE GENE 1 (HOS1) and FVE negatively regulate cold acclimation via a GI-independent pathway. *fve* and *hos1* mutants show enhanced freezing tolerance and altered flowering time phenotypes. FVE is a member of the autonomous pathway that encodes a homologue of the human histone-binding protein RETINOBLASTOMA-ASSOCIATED PROTEIN 46. FVE forms histone-repressive complexes with HISTONE DEACETYLASE 6 (HDA6) and other chromatin modifiers, and directly binds to *FLC* chromatin, repressing *FLC* transcription. FVE also targets cold-responsive genes for epigenetic repression (Gu *et al.*, 2011; Jeon and Kim, 2011). HOS1 is an E3 ubiquitin ligase that targets the cold regulator INDUCER OF CBF EXPRESSION 1 (ICE1) for degradation. Under cold stress, HOS1 targets two flowering pathways to delay the transition to flowering. First, HOS1 interferes with the FLC–HDA6 interaction in an FVE-dependent manner, inhibiting *FLC* repression. Secondly, HOS1 promotes degradation of CO, inhibiting CO-mediated activation of *FT* (Jung *et al.*, 2012, 2013; Jung and Park, 2013).

A cross-repressive interaction between SOC1 and the GATA transcription factors GNC and GNL has recently been shown to account for cross-talk between flowering and the cold response. Increased cold tolerance of *soc1* mutants is dependent on GNL and GNC, positive regulators of cold tolerance. Interestingly, GNC and GNL also directly repress *SOC1* transcription, delaying flowering (Richter *et al.*, 2013).

In winter cereals such as wheat and barley that require vernalization (long exposure to non-freezing temperatures), the vernalization pathway is mainly mediated via the flowering activator VRN-1 (Diallo *et al.*, 2012). Vernalization triggers epigenetic modifications at FLC in *Arabidopsis* or at VRN-1 in cereals to promote flowering. An epigenetic 'memory' of cold is imprinted on these loci so that they can promote flowering when favourable conditions (longer days and warmer temperatures) are experienced (Sheldon *et al.*, 2009; Diallo *et al.*, 2012). However, although winter cereals display freezing tolerance during vegatative development, they can still be sensitive to cold temperatures during flowering. Interestingly, a role for VRN-1 in suppressing the expression of genes encoding C-REPEAT BINDING ELEMENT FACTORS (CBEFs) and their targets *COLD REGULATED* (*COR*) genes has been shown under long days in the diploid wheat *Triticum monococcum*, leading to the proposal that non-essential expression of the *COR* regulon during spring can be deterimental to plant growth (Dhillon *et al.*, 2010).

Currently, relatively little is known about the extent to which the plant's epigenetic 'memory' of winter affects stress responses on a global scale. Expression studies in cereals suggest that a small number of stress-related genes may acquire a memory of vernalization. While many genes differentially regulated during vernalization return to basal levels upon transfer to ambient growth conditions, several stress-related genes remain up- or down-regulated for a week after a return to ambient temperatures (Greenup *et al.*, 2011; Huan *et al.*, 2013). A correlation between the length of vernalization and defence gene expression and disease resistance has also been demonstrated in cereal crops (White and Jenkyn, 1995; Gaudet *et al.*, 2011), suggesting that prolonged exposure to cold plays developmental as well as defensive roles. Together, these examples indicate the multiplicity and complexity of molecular events associated with stress tolerance and flowering in plants.

Nutrient stress, sugar budget, and flowering time

The excessive presence or absence of certain nutrients can trigger a stress response that promotes flowering (Shinozaki

et al., 1988; Tanaka *et al.*, 1991; Kolár and Senková, 2008; Wada and Takeno, 2010; Miyazaki *et al.*, 2014). In *Arabidopsis*, accelerated flowering is triggered by low nitrate levels (Marin *et al.*, 2010; Liu *et al.*, 2013). Nitric oxide is thought to regulate expression of flowering time genes in the photoperiod and autonomous pathways (He *et al.*, 2004).

The sugar budget of the plant, which can be modulated by biotic and abiotic stresses, is a signal that plays a complex regulatory role in flowering (Bolouri Moghaddam and Van den Ende, 2013*a*). In *Arabidopsis*, low sucrose concentrations (e.g. 1%) promote flowering while higher concentrations (e.g. 5%) delay flowering (Ohto *et al.*, 2001). Sucrose affects flowering timing by regulating the expression of floral meristem genes such as *LFY* (Ohto *et al.*, 2001). Mutants compromised in sugar metabolism such as *idd8* deficient for the INDETERMINATE DOMAIN transcription factor IDD8 also show altered flowering time (Seo *et al.*, 2011). New evidence has also shown that AKIN10, a subunit of the SUCROSE NONFERMENTING-1-RELATED PROTEIN KINASE 1 (SnRK1) involved in the regulation of cellular energy metabolism, delays flowering by interacting with IDD8 and inhibiting its activity (Jeong *et al.*, 2015). Trehalose-6-phosphate levels increase during the floral transition and promote flowering by trans-activating *FT* (Wahl *et al.*, 2013). Sugar is also a signal for plant biotic and abiotic stress responses (Bolouri Moghaddam and Van den Ende, 2013*b*; Tauzin and Giardina, 2014; Li *et al.*, 2015), suggesting that sugar signalling is at the intersection of plant stress and flowering.

Biotic stress and flowering time

Infection by fungal, viral, and bacterial pathogens

Biotic stress factors such as attack by pests and pathogens can have a significant effect on plant development including flowering. In *Arabidopsis*, pathogen infection alters flowering time in response to infection with the vascular wilt fungal pathogen *Fusarium oxysporum* (Lyons *et al.*, 2015), and the bacterial pathogen *Pseudomonas syringae* (Korves and Bergelson, 2003). Infection by *Verticillium* spp., which cause vascular wilt disease, delays flowering in some *A. thaliana* ecotypes but accelerates flowering in others. Age-related resistance (ARR), a phenomenon whereby plants become resistant to certain pathogens as they mature, has been associated with the transition to flowering. However, use of flowering time mutants has demonstrated that the transition to flowering is not the developmental switch required for ARR (Wilson *et al.*, 2013). Further studies are required to dissect the link between flowering time and ARR.

QTL analyses conducted on a number of crop plants have identified significant associations between disease resistance and flowering time (e.g. Pinson *et al.*, 2010; Van Inghelandt *et al.*, 2012; Mizobuchi *et al.*, 2013). However, the molecular bases of these associations are often not clear, and the possibility of pleiotropy cannot be ruled out. In *Arabidopsis* natural ecotypes, early flowering time is positively associated with increased susceptibility to *Verticillium* spp. (Veronese *et al.*, 2003) while late flowering is associated with resistance to *F. oxysporum* (Lyons *et al.*, 2015). These associations can be at least partly explained by responses such as senescence that coincide with flower development and promote disease development by hemibiotrophic pathogens (Wingler *et al.*, 2010; Lyons *et al.*, 2015).

Mutant analyses in *Arabidopsis* have identified a number of genes that affect both flowering and plant defence and disease resistance (Kidd *et al.*, 2009; Lai *et al.*, 2014). Autonomous pathway proteins promote flowering by down-regulating *FLC* at ambient temperatures independently of vernalization (Andres and Coupland, 2012). Loss-of-function autonomous pathway mutants have late flowering phenotypes which can be rescued by vernalization or loss of function of FLC (Simpson, 2004). Several autonomous proteins are global regulators of epigenetic modifications or RNA processing, and pleiotrophic defensive phenotypes have been shown for some of these proteins. For example, FLD and FPA promote susceptibility to *P. syringae* (Lyons *et al.*, 2013; Singh *et al.*, 2013). FCA negatively regulates *Cauliflower mosaic virus* (CaMV) symptom development (Cecchini *et al.*, 2002) and FPA and FVE promote susceptibility to *F. oxysporum* (Lyons *et al.*, 2015). Interestingly, enhanced resistance phenotypes of *fpa*, *fld*, and *fve* mutants can be uncoupled from flowering time by epistasis (e.g. double mutant) analysis with *flc* (Singh *et al.*, 2014; Lyons *et al.*, 2015). These findings suggest that these proteins have evolved to play dual roles in defence and flowering. Indeed, transcriptional analyses revealed that defence-associated genes are misregulated in *fve-3* and other flowering time mutants (Wilson *et al.*, 2005; Lyons *et al.*, 2015). Furthermore, genome-wide binding studies conducted for some of the key flowering genes such as FLC and LFY (Deng *et al.*, 2011) identified targets involved in diverse processes, suggesting that these genes may have roles in other plant processes as well.

Similarly to their roles in abiotic stress tolerance, photoperiodic pathway regulators also seem to regulate defence responses. For instance, *Arabidopsis gi* mutants show altered responses to *F. oxysporum* (Lyons *et al.*, 2015) (Fig. 2) and CaMV (Cecchini *et al.*, 2002), while the Mediator complex subunit mutants MED25 and MED8 promote flowering and susceptibility to *F. oxysporum* (Kidd *et al.*, 2009). The meristem identity gene LFY reduces flg22-triggered defence responses by directly binding to and repressing defence genes including *FLS2* and *PEN3*. Consistent with this, the *lfy* mutant shows increased resistance against *P. syringae*, suggesting that LFY diverts resources to flower development at the expense of plant defence (Winter *et al.*, 2011).

Herbivory

Similarly to pathogen infection, herbivory alters flowering time. For instance, herbivory by the green peach aphid *Myzus persicae* or the African cotton leafworm *Spodoptera littoralis* delays flowering in *Arabidopsis* (Züst *et al.*, 2011) and *Brassica rapa* (Schiestl *et al.*, 2014), respectively. Defence compounds such as glucosinolates produced in response to herbivory are thought to contribute to the delay in flowering (Schiestl

et al., 2014). The mechanism by which pathogens influence flowering time is often elusive. In a few cases, pathogen effectors responsible for alterations of plant development, including flowering time, have been identified. For example, when expressed in *Arabidopsis*, the 8D05 effector protein from the nematode *Meloidogyne incognita* accelerates flowering, suggesting that altering the development of the host plant is a strategy used by these parasites (Xue *et al.*, 2013).

Altering flowering timing is also a strategy used by some plants to avoid insect attack. For example, evening primrose plants avoid predation by the moth *Mompha brevivittella* by delaying flowering (Agrawal *et al.*, 2013), and *Lobelia siphilitica* plants that show late flowering show decreased herbivory by the weevil *Cleopmiarus hispidulus* (Parachnowitsch and Caruso, 2008). Finally, similarly to herbivory, wounding/mechanical damage accelerates flowering (Hanley and Fegan, 2007), suggesting that at least some herbivory-induced effects could be attributed to wounding.

Endophytes, soil microbes, and competition from nearby plants

Endophytic relationships also alter flowering time, as evidenced by the plant growth-promoting bacterial endophyte *Burkholderia phytofirmans*, which accelerates flowering of *Arabidopsis* (Poupin *et al.*, 2013). Recently, distinct roles for non-pathogenic soil microbes in flowering time have been discovered (Lau and Lennon, 2012; Panke-Buisse *et al.*, 2014; Wagner *et al.*, 2014). Soil microbes that cause late flowering also promote plant biomass in *B. rapa* (Lau and Lennon, 2011). Together, these findings are consistent with the view that the plant's biotic environment has an overwhelming effect on plant phenology and indicate that additional tools may be available to manipulate flowering time in crop plants.

Stress imposed by neighbouring plants competing for available resources such as light, water, and nutrients can dramatically alter flowering time (Vermeulen, 2015). In particular, altered red:far-red (R:FR) light spectra in the lower canopy can lead to rapid stem elongation and altered flowering time in species that are not tolerant to shade by neighbouring plants, a phenomenon known as the shade avoidance syndrome (Pierik and Testerink, 2014).

Plant hormones and flowering time

Given that plant hormones regulate diverse plant processes, including responses to biotic and abiotic stress responses, it is perhaps not surprising that associations between flowering time and plant hormones (cytokinins, salicylic acid, jasmonic acid, GA, ABA, auxin, ethylene, and brassinosteroids) have been observed (reviewed by Davis, 2009; Diezel *et al.*, 2011; Wasternack *et al.*, 2013) (Fig. 3). Here, we briefly discuss the roles of plant hormones involved in development and stress in regulating transition to flowering in response to biotic and abiotic stress factors. The role of the GA pathway in flowering has been reviewed elsewhere (e.g. Mutasa-Göttgens and Hedden, 2009).

Salicylic acid

Salicylic acid (SA) is a plant hormone with major regulatory roles in plant defence, but it also regulates flowering time in several plant species (Khurana and Cleland, 1992; Hatayama and Takeno 2003; Wada and Takeno, 2010; Wada *et al.*, 2010; Shimakawa *et al.*, 2012; Yamada and Takeno, 2014). The relationship between SA signalling and flowering time is summarized in Fig. 4. As early as 1974, a positive regulatory effect of SA on flowering time was proposed when SA contained in aphid-produced honeydew was shown to induce flowering in duckweed (Cleland and Ajami, 1974). More recently, *Arabidopsis* SA-deficient mutants were shown to have delayed flowering, suggesting that SA accelerates flowering time (Martinez *et al.*, 2004; Villajuana-Bonequi *et al.*, 2014). PATHOGEN AND CIRCADIAN CONTROLLED 1 (PCC1) is required for SA-triggered flowering but seems to act independently of key photoperiodic pathway regulators such as CO and FT (Segarra *et al.*, 2010; Mir and Leon, 2014). A correlation between flowering time and systemic acquired resistance (SAR), which is regulated by SA, has also been proposed in *Arabidopsis* (Singh *et al.*, 2013; Banday and Nandi, 2015). SA-mediated defence regulators SUMO (small ubiquitin related modified), E3 ligase SIZ1 (Jin *et al.*, 2008), PLANT U-BOX 13 (PUB13), (Liu *et al.*, 2012), MYB30 (Liu *et al.*, 2014), GLYCINE RICH RNA-BINDING PROTEIN 7 (GRP7) (Streitner *et al.*, 2008), and SA signalling components

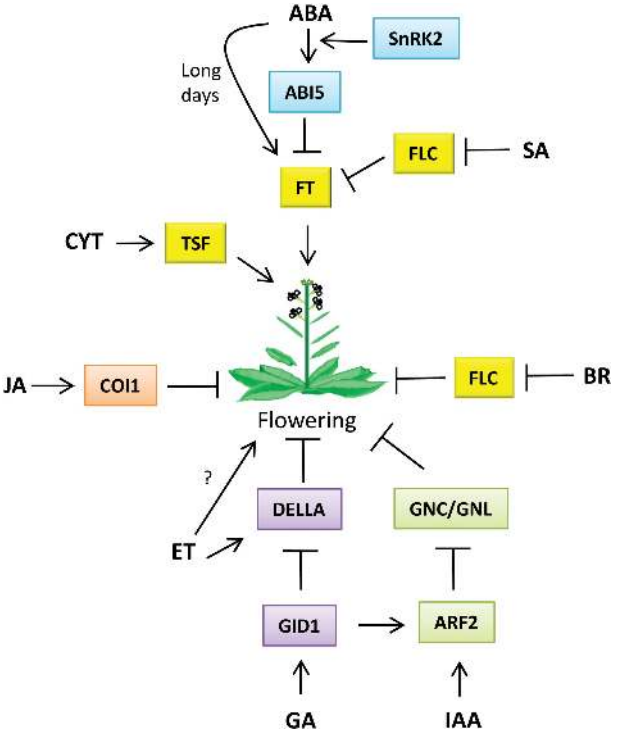

Fig. 3. Flowering time in plants is regulated by multiple hormones. In general, JA (jasmonate) delays flowering while SA (salicylic acid), BR (brassinosteroids), IAA (auxin), CYT (cytokinin), and GA (gibberellins) accelerate flowering in *Arabidopsis*. The role of ABA (abscisic) and ET (ethylene) in flowering time regulation requires further investigation as these hormones have been shown both to promote and to delay flowering in *Arabidopsis.* See text for further details.

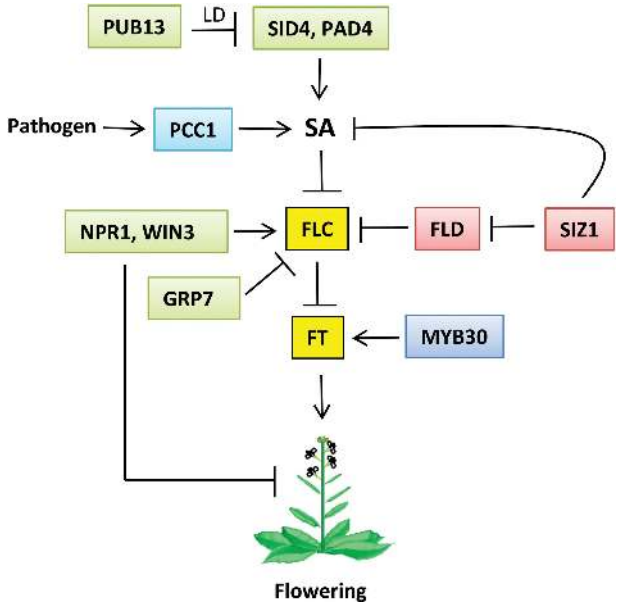

Fig. 4. The role of the salicylic acid (SA) pathway, a regulator of biotic and abiotic stress tolerance, in flowering. See text for details.

Nonexpresser of PR genes 1 (NPR1) and HOPW1-1-INTERACTING3 (WIN3) have been implicated in flowering time regulation (Wang *et al.*, 2011) (Fig. 4).

Jasmonic acid

Jasmonates are plant hormones with diverse roles in regulating biotic and abiotic stress tolerance as well as development (Kazan, 2015). The interactions between jasmonic acid (JA) and light, phytochrome, and circadian pathways are also known (Kazan and Manners, 2011, 2013). Therefore, it is probably unsurprising that links between JAs and flowering processes have also been uncovered. In *Arabidopsis*, JA seems to delay flowering. The JA receptor mutant *coi1-1* is early flowering (Song *et al.*, 2013), and plants that are touched repeatedly show a delay in flowering, which is dependent on JA signalling (Chehab *et al.*, 2012). A subgroup of bHLH transcription factors that negatively regulate JA-mediated defence responses are thought to promote flowering. Mutant plants defective for this subgroup of bHLH factors showed altered resistance to several pathogens and pests, and were late flowering, while overexpression lines were early flowering (Song *et al.*, 2013). In wheat, *maintained vegetative phase 1* (*mpv1*) mutants that lack the vernalization gene VRN-1 never flower and produce enhanced levels of methyl jasmonate (MeJA). MeJA levels increase during vernalization, and exogenous MeJA application delays flowering, suggesting that JA modulates both vernalization and flowering in wheat (Diallo *et al.*, 2014). This may have evolved as a mechanism to protect wheat plants from biotic and abiotic stresses during the vernalization period.

Ethylene

In contrast to JA, ethylene, a plant hormone that regulates biotic and abiotic stress tolerance in plants (Kazan, 2015), seems to delay flowering time in *Arabidopsis* (Achard *et al.*, 2007), although further research is required to dissect the role of this plant hormone in flowering processes. *Arabidopsis* mutants compromised in ethylene biosynthesis are early flowering (Tsuchisaka *et al.*, 2009), while the ethylene receptor gain-of-function mutant *etr1-1* is late flowering (Ogawara *et al.*, 2003). *constitutive triple response1* (*ctr1*) mutants, which exhibit constitutive ethylene signalling, are late flowering under short days. In the *ctr1* background, ethylene is thought to delay flowering by promoting the accumulation of DELLA proteins, thereby interfering with the GA flowering promotion pathway (Achard *et al.*, 2007). In contrast, ethylene-insensitive mutants are late flowering, suggesting that ethylene signalling may accelerate flowering (Ogawara *et al.*, 2003). In rice, the ethylene receptor mutant *etr2* is early flowering while the ETR2 overexpressor is late flowering (Wuriyanghan *et al.*, 2009), suggesting that ethylene signalling delays flowering in both rice and *Arabidopsis*.

Abscisic acid

ABA is a plant hormone that primarily regulates plant abiotic stress tolerance. The role of this hormone in flowering seems to be complex. On one hand, it appears that ABA delays flowering in *Arabidopsis* since ABA-insensitive/deficient mutants such as *aba1* and *aba2* are early flowering (Barrero *et al.*, 2005; Domagalska *et al.*, 2010) and ABA treatment delays flowering (Wang *et al.*, 2013). However, other evidence suggests that endogenous ABA has no effect on flowering (Domagalska *et al.*, 2010) or accelerates flowering via promoting expression of *FT* and related genes (Riboni *et al.*, 2013, 2014). ABA-deficient mutants *aba2-1* and *aba1-6* are late flowering and show reduced *FT* expression (Riboni *et al.*, 2013).

The contradictory findings of the role of ABA in flowering may be explained by differences in *Arabidopsis* mutant ecotypes, differences in the ABA concentrations applied (Domagalska *et al.*, 2010), or by ABA differentially modulating flowering in short and long days (Riboni *et al.*, 2013). Readers interested in finding out more about the role of this hormone in flowering responses should consult the recent review by Conti *et al.* (2014).

Brassinosteroids and cytokinins

Brassinosteroids (BRs) regulate both stress and flowering responses in plants. BR-deficient mutants *det2*, *dwf4*, and *cpd* and the BR-insensitive mutant *bri1* are late flowering, suggesting that BR signalling promotes flowering (Domagalska *et al.*, 2007; Li *et al.*, 2010). Similarly, cytokinin application to roots promotes flowering in short days in *Arabidopsis* through the activation *TSF* and *FD* (D'Aloia *et al.*, 2011).

Epigenetic factors regulating flowering time and stress responses

As reviewed previously (Yaish *et al.*, 2011), remarkable progress has been made in recent years to understand

how epigenetic modifications such as DNA methylation and histone marks affect flowering (Zhang *et al.*, 2011). The *Arabidopsis* EMBRYONIC FLOWER1 (EMF1) and ULTRAPETALA1 (ULT1) implicated in mediating histone depositions were found to regulate both salt stress and flowering responses (Pu *et al.*, 2013). Remarkably, stress experienced by the parents can be transmitted to the offspring to accelerate flowering (Suter and Widmer, 2013). Drought stress experienced by the plant can have an effect on flowering in its offspring. Plants of *B. rapa* grown from seeds collected after natural drought stress flowered earlier than those collected before the drought, suggesting that stressful memories can be epigenetically transmitted and that plants can rapidly evolve to select for drought escape (Franks, 2011). Similarly, an interesting function of FT in temperature sensing has recently been described. It appears that temperatures experienced by the mother plant are transduced by FT to alter seed protoanthocyanin content which controls progeny seed dormancy (Chen *et al.*, 2014).

Regulation of flowering time by stress-associated microRNAs

A number of stress-inducible miRNAs are involved in the regulation of flowering time in plants (reviewed in Spanudakis and Jackson, 2014; Hong and Jackson, 2015; Teotia and Tang, 2015). The characteriaztion of these miRNAs is providing new insights into the processes involved in stress-mediated flowering time regulation. Overexpression of miR169, which is cold, drought, and salt inducible, results in an early flowering phenotype. The transcription factor NF-YA2 activates *FLC*, delaying flowering. miR169 targets NF-YA2 for degradation, accelerating flowering by derepressing *FLC* (Xu *et al.*, 2014). miR156, another stress-inducible miRNA, delays flowering in *Arabidopsis* by targeting the SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) family of transcription factors for repression. miR156 disrupts the cleavage of *SPL3*, which activates floral promoters *FRUITFULL* (*FUL*), *LFY*, and *AP1* (Yamaguchi *et al.*, 2009) and also represses the expression of *SPL9*, which accelerates flowering by activating floral promoters including *FUL*, *SOC1*, *LFY*, and *AP1* (Fig. 5). Under abiotic stress conditions, plants overexpressing miR156 are late flowering and more tolerant to stress, whereas plants that have a silenced copy of miR156 show accelerated flowering and increased sensitivity to abiotic stress (Cui *et al.*, 2014). The role of miR156 in controlling flowering seems to be conserved in tobacco since miR156-overexpressing plants show delayed flowering (Zhang *et al.*, 2015). As a potential fine-tuning feedback mechanism, miR156 expression is also induced by the floral repressors AGL15 and AGL18 (Serivichyaswat *et al.*, 2015).

Another miRNA, miR172, promotes flowering in *Arabidopsis* by targeting AP2-like floral repressors including *TOE2* and *SMZ* (Hong and Jackson, 2015). Several flowering time genes including *GI*, *SVP*, and *FCA* regulate the expression of miR172, which is drought stress inducible (Han *et al.*, 2013). *gi* mutants also show altered drought escape responses. It was proposed that the down-regulation of WRKY44 by miR172, which is regulated by GI, contributes to this phenomenon (Han *et al.*, 2013) (Fig. 2). In rice, OsmiR393 down-regulates the expression from putative auxin receptors *OsTIR1* and *OsAFB2*; this leads to early flowering but reduced salt and drought tolerance (Xia *et al.*, 2012).

Flowering time and climate change

Global temperatures are expected to rise significantly in the future in parallel with the increases in CO_2 levels. It is expected that one of the major effects of climate change will be on flowering time (Craufurd and Wheeler, 2009; Ellwood *et al.*, 2013; Hänel and Tielbörger, 2015). Indeed, a study of >400 plant species revealed a trend towards earlier flowering in response to climate change (Parmesan, 2006). The frequency and intensity of drought, heat stress, disease epidemics, and plant growth are also expected to be affected by global warming. Therefore, a better understanding of how complex environmental variables affect plant phenology is important for future genetic manipulation of crops to increase productivity.

Many crops, including rice and barley, show accelerated flowering when grown under elevated CO_2. Natural variation in response to altered CO_2 is evident in *A. thaliana*, where different ecotypes show accelerated, delayed, or unaltered flowering time in response to high CO_2, suggesting localized adaptations to environmental variables (Springer and Ward, 2007). Interestingly, high CO_2 and elevated temperatures differentially regulate the expression of miR156/157 and miR172 (Fig. 5). High CO_2 accelerates flowering by repressing miR156/157 and activating miR172 (May *et al.*, 2013).

Global warming is predicted to accelerate flowering in many *A. thaliana* ecotypes (Li *et al.*, 2014). Allelic variation in QTLs underlying altered flowering time responses to increased temperatures has been shown, suggesting that *A. thaliana* has the potential to adapt to a changing climate (Li *et al.*, 2014). In recent years, elevated temperatures have also accelerated the heading date of wheat grown in Europe by nearly 2 weeks. At first sight, early flowering appears to be a useful agronomic trait for wheat, given that this crop is sensitive to heat stress that may occur late during flowering (Stratonovitch and Semenov, 2015). However, it is currently unknown if early heading can positively contribute to yield as heat stress experienced during grain filling could still have a negative effect on yield by shortening the period between heading and full maturity. Early flowering could also increase the possibility of flowering coinciding with the onset of late frosts which can cause sterility and reduce yield (Fuller *et al.*, 2007).

GIGANTEA (GI) is a master regulator of stress tolerance and flowering time

It is evident from the studies reviewed here that GI, which promotes flowering through photoperiod and circadian pathways, is emerging as a master regulator of biotic and

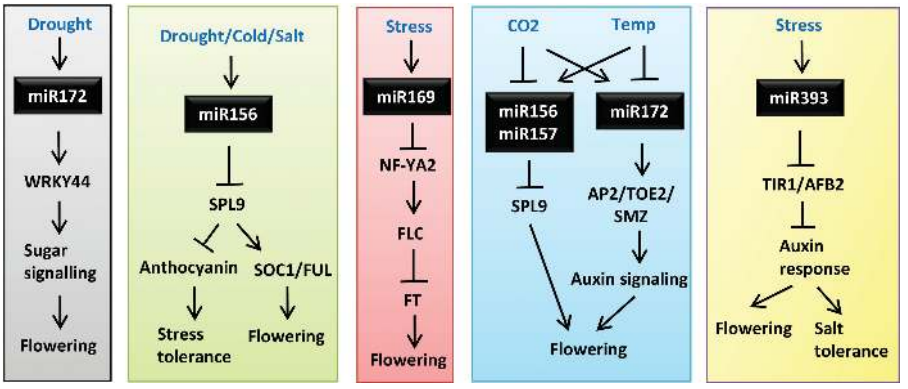

Fig. 5. Regulation of flowering by stress-associated miRNAs in *Arabidopsis*. See text for further details.

abiotic stress responses (reviewed by Mishra and Panigrahi, 2015). As discussed above, *gi* mutants exhibit altered freezing tolerance/cold acclimation (Cao *et al.*, 2005, 2007; Fornara *et al.*, 2015), hypocotyl growth (Fornara *et al.*, 2015), drought escape response (Riboni *et al.*, 2013), salt tolerance (Park *et al.*, 2013; Kim *et al.*, 2013), oxidative stress response (Kurepa *et al.*, 1998*a*), sucrose metabolism (Kurepa *et al.*, 1998*b*), and increased longevity (Kurepa *et al.*, 1998*a*) phenotypes (Fig. 2). Given the associations described between stress tolerance and flowering time, one may wonder if alterations in one trait may have an unintended consequence on the other. Interestingly, a recent study showed that of the two *GI* homoealleles found in *B. rapa*, an allopolyploid plant species, only one *GI* allele could rescue the increased abiotic stress tolerance phenotype of the *Arabidopsis gi* mutant. In contrast, both *GI* alleles could rescue the flowering time defects of the *gi* mutant (Xie *et al.*, 2015). These findings suggest that the effect of GI on phenology and stress tolerance may be uncoupled, and this provides an opportunity for modifying stress tolerance without affecting flowering time.

Flowering time and adaptation of plants to marginal environments

The ability to alter flowering time genetically allows the plant to reproduce in an environment that is not normally conducive to flowering. One particularly interesting example that illustrates this point is the barley *Ppd-H1* gene that promotes early flowering under long-day conditions. Barley accessions from the Middle East, where domestication of this species occurred, contain *Ppd-H1* alleles that facilitate completion of their life cycle before the onset of summer drought. In contrast, barley accessions adapted to the milder environments of northern Europe flower late and contain mutant alleles of *Ppd-H1* (reviewed by Turner *et al.*, 2005; Andrés and Coupland, 2012). Similarly, barley accessions adapted to environments with relatively short growing seasons contain mutant alleles (*eam8*) of the circadian clock gene *EARLY MATURITY8*, a homologue of the *Arabidopsis* EARLY FLOWERING3 gene. The *eam8* mutation accelerates reproductive development (Faure *et al.*, 2012).

In contrast to winter cereals, flowering in rice is promoted by short days through the actions of floral promoters including EARLY HEADING DATE 1 (EHD1) (Itoh *et al.*, 2010) Under long days such as those observed in northern latitudes, floral repressors target EHD1 to suppress flowering (Lee *et al.*, 2010). Recent research has shown that mutations in long-day floral repressors have played a major role in enabling rice to adapt to northern latitudes of Europe (Gómez-Ariza *et al.*, 2015). Therefore, an increased understanding of the natural adaptations used by plants to grow in different environments can be harnessed to expand the growing area of crops.

Stress-induced flowering: a dilemma faced by agriculture

As is evident from this review, mechanisms underlying the control of the flowering transition derive overwhelmingly from the model species *Arabidopsis*. The impact of stress and environment on flowering timing is species and variable dependent (Jung and Müller, 2009), and it is likely that genetic pathways underlying the trade-off between flowering and stress have diverged somewhat between *Arabidopsis* and crops. Therefore, more work is required to understand how economically important species such as rice, maize, and wheat, which provide 60% of global human food consumption (Food and Agriculture Organization of the United Nations, 1995) integrate stress responses and flowering time.

It can be speculated that plants have the ability to sense the strength of stress factors and respond appropriately. It also appears that the effect of stress-induced flowering, which can be influenced by day length, is dependent on the timing of the stress and the plant species. Under a gradual and mild stress, flowering is delayed so that reproductive processes can resume at a later time. However, under a terminal stress where physical damage to tissue is expected to occur, flowering is accelerated to ensure reproduction, even if this comes at the expense of yield. Indeed, in crop plants, the correct timing of flowering is critical for adaptation to specific environments and closely correlates with grain yield (Gao *et al.*, 2014). If flowering occurs prematurely under stressful environments, seed-set and grain filling may be compromised. If flowering is delayed, the plant risks succumbing to terminal stress

before producing any seed. The association between stress and flowering time can lead to undesirable consequences. For instance, heterologous expression of a MYB transcription factor from chrysanthemum in *Arabidopsis* enhances drought and salinity tolerance but can also lead to delays in flowering (Shan *et al.*, 2012). Therefore, enhanced understanding of the links between these processes is essential for engineering of stress tolerance in crops. A challenge for future agriculture in a changing climate is to predict which combination of traits to breed into crops in order to maximize development while avoiding stress-induced yield penalties.

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