Journal of Experimental Botany, Vol. 66, No. 3 pp. 719–730, 2015
doi:10.1093/jxb/eru441 Advance Access publication 4 November, 2014

Journal of Experimental Botany
www.jxb.oxfordjournals.org

FLOWERING NEWSLETTER REVIEW

Time to flower: interplay between photoperiod and the circadian clock

Mikael Johansson and Dorothee Staiger*

Molecular Cell Physiology, Faculty for Biology, Bielefeld University, Germany

* To whom correspondence should be addressed. E-mail: dorothee.staiger@uni-bielefeld.de

Received 21 August 2014; Revised 6 October 2014; Accepted 6 October 2014

Abstract

Plants precisely time the onset of flowering to ensure reproductive success. A major factor in seasonal control of flowering time is the photoperiod. The length of the daily light period is measured by the circadian clock in leaves, and a signal is conveyed to the shoot apex to initiate floral transition accordingly. In the last two decades, the molecular players in the photoperiodic pathway have been identified in *Arabidopsis thaliana.* Moreover, the intricate connections between the circadian clockwork and components of the photoperiodic pathway have been unravelled. In particular, the molecular basis of time-of-day-dependent sensitivity to floral stimuli, as predicted by Bünning and Pittendrigh, has been elucidated. This review covers recent insights into the molecular mechanisms underlying clock regulation of photoperiodic responses and the integration of the photoperiodic pathway into the flowering time network in *Arabidopsis*. Furthermore, examples of conservation and divergence in photoperiodic flower induction in other plant species are discussed.

Key words: *Arabidopsis*, circadian clock, CO, flowering, FT, photoperiod.

Introduction

Plants carefully time the onset of flowering to the appropriate season of the year to ensure reproductive success. Premature flowering limits vegetative growth and thus the accumulation of sufficient resources, while flowering too late puts the developing seeds at risk of harmful environmental conditions in the autumn, such as frost. A major determinant of floral transition is the relative duration of light and dark, as the change of daylength over the year is a reliable indicator of seasonal progression (Srikanth and Schmid, 2011; Andres and Coupland, 2012). Plants keep track of daylength, the photoperiod, with the help of an endogenous timekeeper. This clock synchronizes physiological and molecular processes to the day–night cycle, enabling plants to anticipate upcoming conditions (Green *et al.*, 2002; Dodd *et al.*, 2005). The endogenous clock has for some time been implicated in photoperiodic flowering time control (Bünning, 1936). It was also known that the photoperiod is perceived in the leaves and thus a signal has to travel to the shoot apex to initiate flowering. This led to the concept of 'florigen'—hormone-like substances transmitting the command to flower within the plant (Chailakhyan, 1936). Additionally, the idea of graft-transmissible floral repressors, 'antiflorigens', was put forward (Lang and Melchers, 1943).

Elucidation of the molecular events underlying photoperiodic time measurement and relaying this information to the site of flower initiation gained momentum with *Arabidopsis thaliana* emerging as a model organism for flowering time control (Redei, 1962; Koornneef *et al.*, 1991). Twenty years ago, the central player of the photoperiodic pathway was cloned from the *constans* mutant that flowers irrespective of daylength (Putterill *et al.*, 1995). Around the same time, the first clock components were cloned, providing an entry to dissect the connection between circadian timing and photoperiodic response (Schaffer *et al.*, 1998; Wang and Tobin, 1998; Fowler *et al.*, 1999). Identification of an ever-increasing array of clock components and insights into their molecular properties progressed in parallel with in-depth characterization of flowering time mutants and targeted biochemical and cell biology approaches to unravel molecular players of the

© The Author 2014. Published by Oxford University Press on behalf of the Society for Experimental Biology. All rights reserved.
For permissions, please email: journals.permissions@oup.com

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

external and internal coincidence mechanism and resolve the identity of florigens and antiflorigens. In addition, mathematical modelling predicted new interactions within the photoperiodic pathway that were confirmed experimentally (Salazar *et al.*, 2009). The importance of the circadian clock in flowering time control was further emphasized by a field study combining genome-wide association and quantitative trait loci mapping, where clock-related genes, not previously connected to flowering in greenhouse conditions, were identified (Brachi *et al.*, 2010).

Here we review recent insights into the molecular mechanisms underlying clock regulation of photoperiodic responses and the integration of the photoperiodic pathway into the flowering time network in *Arabidopsis*. Furthermore, we point out emerging conservation and divergence in photoperiodic flower induction in plant species where the information is less comprehensive.

The circadian timing system in *Arabidopsis thaliana*

The core clockwork of the 'circadian' (meaning about a day) timing system operates at the level of single cells. It comprises molecular feedback loops through which clock proteins generate their own 24 h rhythm. The blueprint of the *Arabidopsis* clock serves as a reference for clocks in other plants (McClung, 2013; Staiger *et al.*, 2013). Of note is that while the principle of timekeeping is conserved in eukaryotes, the molecular components of plant clocks are distinctly different from those of mammals and insects, which share a high degree of homology (Young and Kay, 2001).

Initially, the core clock in *Arabidopsis* was viewed as a series of feedback circuits through which clock components reciprocally or sequentially repress each other. Two related Myb transcription factors LATE ELONGATED HYPOCOTYL (LHY) and CIRCADIAN CLOCK ASSOCIATED1 (CCA1) whose expression peaks at dawn repress *TIMING OF CAB EXPRESSION1* (*TOC1*)/*PSEUDO-RESPONSE REGULATOR1* (*PRR1*), peaking at dusk (Schaffer *et al.*, 1998; Wang and Tobin, 1998; Alabadi *et al.*, 2001) (Fig. 1). Inhibition of *TOC1* transcription by CCA1 and LHY additionally involves a co-repressor complex of CONSTITUTIVE PHOTOMORPHOGENIC10 (COP10), DE-ETIOLATED1, and DDB1 (Lau *et al.*, 2011). TOC1/PRR1 is the founding member of a family of PRRs that peak in a serial manner over the day, starting with *PRR9* in the morning, followed by *PRR7*, *PRR5*, and then *TOC1* peaking around dusk (Matsushika *et al.*, 2000). The PRRs including TOC1 sequentially repress *CCA1* and *LHY* in the course of the day (Nakamichi *et al.*, 2010; Gendron *et al.*, 2012; Huang *et al.*, 2012; Carre and Veflingstad, 2013). Recently it was found that *CCA1* and *LHY* repression by PRR9, PRR7, and PRR5 relies on co-repressors of the TOPLESS/TOPLESS-RELATED (TPR) family (Wang *et al.*, 2013).

Another important regulatory element is the evening complex (EC) comprising *EARLY FLOWERING 3* (*ELF 3*), *ELF4*, and the DNA-binding factor *LUX ARRHYTHMO* (*LUX*), all peaking at the end of the day (Dixon *et al.*, 2011; Herrero *et al.*, 2012; Nusinow *et al.*, 2011). The EC is under negative control by CCA1 and LHY (Lu *et al.*, 2012). The EC in turn represses *PRR9*, *PRR7*, *PRR5*, and *TOC1*, and as a consequence repression of *LHY* and *CCA1* is alleviated.

Post-translational processes contribute to adjust the period of clock protein oscillations to 24 h (Schöning and Staiger, 2005; Mas, 2008). ZEITLUPE (ZTL), an F-box protein subunit of an SCF ubiquitin ligase complex with a light-regulated LOV (LIGHT, OXYGEN, VOLTAGE) protein interaction

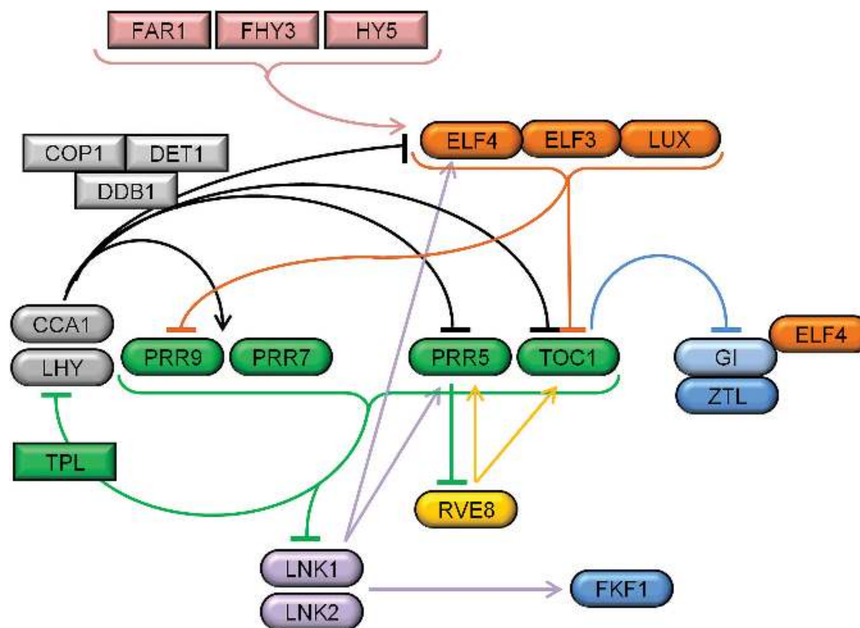

Fig. 1. Conceptual model of the Arabidopsis circadian clockwork. The interaction of clock components (ovals) is shown. Rectangles represent accessory factors; arrows indicate activation; arrows with blunt ends represent repression; adjoining ovals/rectangles indicate protein interaction. See text for details. (This figure is available in colour at *JXB* online.)

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

domain, interacts with the clock protein GIGANTEA (GI). During the day, blue light enhances the interaction with GI so that ZTL is stabilized at specific times of the day, resulting in ZTL protein oscillations (Kim *et al.*, 2007; J. Kim *et al.*, 2013). ZTL also interacts with PRR5 and TOC1 to target them for proteosomal degradation in the cytoplasm (Mas *et al.*, 2003). Interaction between PRR5 and TOC1 enhances TOC1 accumulation in the nucleus and thus shields TOC1 from degradation (Wang *et al.*, 2010).

While the components of these clock feedback loops all interact by reciprocal or sequential repression, positive elements have been unveiled more recently. FAR-RED ELONGATED HYPOCOTYL3 (FHY3), FAR-RED IMPAIRED RESPONSE1 (FAR1), and HY5 activate *ELF4* during the day (Li *et al.*, 2011), and CCA1 and LHY repress *ELF4* at dawn through direct interaction with these activators. The REVEILLE (RVE) family with homologues of CCA1 and LHY comprises members that positively regulate clock gene expression (Farinas and Mas, 2011). RVE8 promotes *PRR5* and *TOC1* expression. PRR5 in turn binds to the *RVE8* promoter and presumably represses *RVE8*, thus giving rise to yet another feedback loop (Rawat *et al.*, 2011; Hsu *et al.*, 2013).

LIGHT-REGULATED WD1 (LWD1) and (LWD2) are activators of *PRR9*, *PRR7*. and *PRR5* (Wang *et al.*, 2011). In addition, the morning-phased and red light-activated NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED1 (LNK1) and LNK2 promote expression of a subset of early evening genes, among them *ELF4* and *PRR5*, while they are directly repressed by PRRs, creating another negative feedback loop (Rugnone *et al.*, 2013). Furthermore, they interact with CCA1, LHY, RVE4, and RVE8, acting as co-activators of *PRR5* and *ELF4* transcription (Xie *et al.*, 2014). In addition, LNK1 and LNK2 activate FLAVIN BINDING, KELCH REPEAT, F-BOX1 (FKF1), an important regulator of photoperiodic flowering. Overall, the increasing number of reciprocal interactions suggests that the clockwork is best represented by an intricate web of connections (Fogelmark and Troein, 2014).

To attain synchrony with day and night, the clock is entrained via the red/far-red-absorbing PHYTOCHROMES (PHYA–PHYE), the blue light-absorbing CRYPTOCHROMES (CRY1 and CRY2), and the LOV domain proteins ZTL, FKF1, and LOV KELCH PROTEIN 2 (LKP2) (Devlin, 2002; Fankhauser and Staiger, 2002). Clock protein oscillations in turn control biological processes, the clock outputs, so that photosynthetic activity or growth take place at the optimal time of day for maximal performance and so that seasonal processes including photoperiodic flowering occur at the right time of the year.

Photoperiodic flowering

In many plants, the length of the light phase, the photoperiod, determines flowering time (Garner and Allard, 1920). Long-day (LD) plants including *Arabidopsis* and the crop plant *Hordeum vulgare* flower in response to increasing daylength. Short-day (SD) plants include *Oryza sativa*, sorghum, and chrysanthemum. Erwin Bünning was the first to implicate an endogenous rhythm of alternating light-sensitive (photophile) and dark-sensitive (scotophile) phases in photoperiodic timekeeping (Bünning, 1936). This rhythm is generated by a biological clock itself entrained by the day–night cycle, and plants distinguish LDs and SDs based on whether ambient light coincides with the photophile or scotophile phase. This was further specified in the external coincidence model of an endogenous rhythm that needs to interact productively with light to trigger the photoperiodic response (Pittendrigh and Minis, 1964). It was also taken into consideration that multicellular organisms harbour populations of oscillators with defined phase relationships. According to the internal coincidence model, the photoperiodic response would then result from the interaction of two endogenous rhythms falling into phase. Thus, the function of light would be to control the phase relationship of these rhythms (Pittendrigh, 1972).

Experimental proof for the involvement of the clock in photoperiodic flowering came from mutants affected in both circadian and photoperiodic timekeeping (Hicks *et al.*, 1996; Schaffer *et al.*, 1998; Wang and Tobin, 1998; Fowler *et al.*, 1999). Subsequently it was found that the circadian clock and light signalling control the activity of CONSTANS (CO), the key component of the photoperiodic pathway, in the leaves. CO belongs to the B-Box Zinc-Finger Family (BBX), with the B-box B1 and B2 protein interaction domains at the N-terminus. The C-terminal domain can bind DNA and is known as the CCT (CO, CO-like, and TOC1) domain. CO activates transcription of *FLOWERING LOCUS T* (*FT*) and the more weakly expressed *TWIN SISTERS OF FT* (*TSF*) in leaf phloem companion cells (Samach *et al.*, 2000; Yamaguchi *et al.*, 2005). The FT protein then moves through the phloem to the shoot apical meristem (Corbesier *et al.*, 2007; Jaeger and Wigge, 2007; Mathieu *et al.*, 2007). Interaction with the bZIP transcription factors FD and FD PARALOGUE (Abe *et al.*, 2005) activates floral integrator genes including *SUPPRESSOR OF CONSTANS OVEREXPRESSION1* and *APETALA1*, entailing induction of a cascade of downstream genes, leading to flowering.

Temporal control of *CO* transcription

An intricate network of factors shapes CO expression at the transcriptional and post-translational level to confine *FT* transcription to LDs (Fig. 2). In non-inductive SDs, the *CO* mRNA undergoes circadian oscillations with a peak at zt8 (Zeitgeber time 8, i.e. 8 h after dawn) to zt10 (Suarez-Lopez *et al.*, 2001). The CO protein is degraded in darkness, however, and does not accumulate. *CO* transcription is repressed in the morning by the CYCLING DOF FACTOR (CDF) proteins (Fornara *et al.*, 2009). The CDF1 binding site, CTTT, is part of a 7 bp sequence CTTTACA that occurs in multiple repeats in the *CO* promoter (Imaizumi *et al.*, 2005). Natural variation in the number of these repeats caused by a 7 bp insertion/deletion polymorphism correlates with flowering time (Rosas *et al.*, 2014). CDF1 and CDF2 levels are regulated by

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

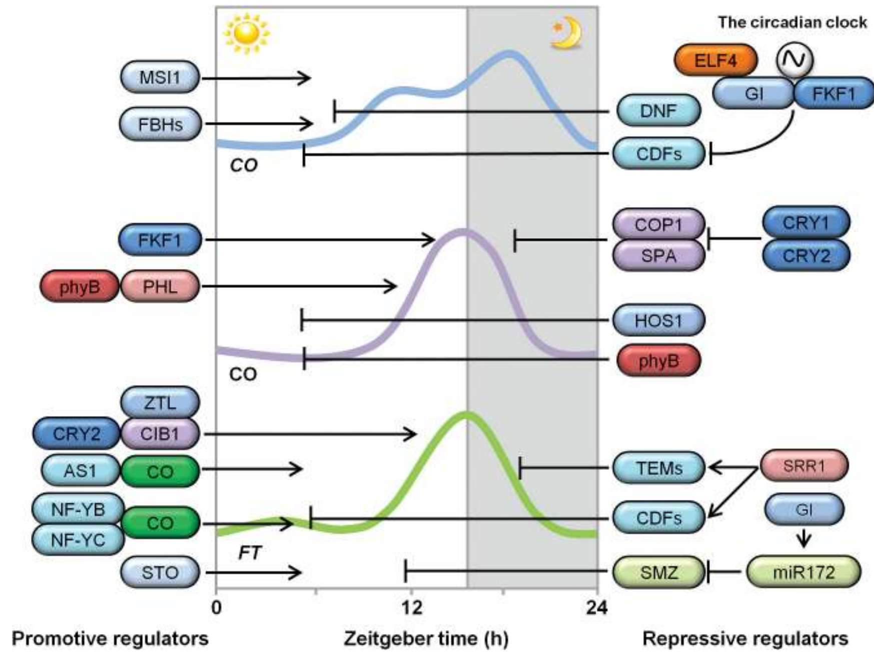

Fig. 2. Photoperiodic regulation of CO and FT in inductive LDs. The patterns of *CO* transcript (top), CO protein (middle), and *FT* transcript (bottom) during the day are displayed with promotive regulators indicated on the left and repressive regulators on the right. Activators of *CO* transcription include MSI1 and FBH1-4; repressors include the CDFs and DNF. Activators of CO protein accumulation include FKF1 and PHL; repressors include the COP1–SPA complex, phyB, and HOS1. Activators of *FT* transcription include CO, NF-YB and NF-YC, AS1, CIB1, and STO; repressors include TEM1 and TEM2, CDFs, and SMZ. The end of the arrows indicates the time of day when the factors are active. (This figure is available in colour at *JXB* online.)

proteolytic degradation via a light-dependent complex of the clock proteins GI and FKF1. In SDs, *GI* peaks at zt7 and *FKF1* peaks at zt10 (Fornara *et al.*, 2009). Thus, both proteins are expressed during darkness and do not interact so that the CDFs remain active (Sawa *et al.*, 2007). In LDs, the *FKF1* and *GI* rhythms become synchronized, peaking around zt13, and the proteins undergo a blue light-dependent complex formation which stabilizes FKF1 (Sawa *et al.*, 2007). The FKF1–GI complex then targets CDF1 and CDF2 to the proteasome, and the relief from repression allows *CO* expression with a peak around zt12–zt16. This allows CO protein to accumulate in the light and stimulate *FT* transcription. The light-dependent interaction of FKF1 and GI is similar to the interaction of ZTL with GI during the circadian cycle. To date, a role for ZTL in photoperiodic flowering has not been described.

Recently, additional factors have been identified that control *CO* expression. ELF4 regulates the access of GI to chromatin by sequestering GI from the nucleoplasm into subnuclear bodies preferentially during the night, thus restricting its ability to bind to the *CO* promoter (Y. Kim *et al.*, 2013). DAY NEUTRAL FLOWERING (DNF) is expressed between zt4 and zt6 in SDs and acts to repress *CO* around this time (Morris *et al.*, 2010). This occurs independently of the GI–FKF1–CDF1 module. As DNF encodes a membrane-bound E3 ligase, it may affect CO stability. The FLOWERING BHLH (basic helix–loop–helix) proteins FBH1, FBH2, FBH3, and FBH4 act as transcriptional activators of *CO* (Ito *et al.*, 2012). Binding of FBH1 to the *CO* promoter occurs throughout the day, suggesting that any timed activity would require post-translational regulation. FBH1 is also connected to the circadian clock through binding and repression of the *CCA1* promoter (Pruneda-Paz *et al.*, 2014).

PHYTOCHROME AND FLOWERING TIME (PFT1)/MED25, a subunit of the Mediator complex that bridges transcription factors with RNA polymerase II, promotes *CO* expression and *FT* expression in a CO-independent manner (Inigo *et al.*, 2011). Recently, the function of PFT1 was shown to depend on the photoperiod and on the length of a short tandem repeat region encoding a 90 amino acid region enriched in Q (Rival *et al.*, 2014). PFT1 complemented *pft1-2* late flowering in LDs when the tandem repeat region was shorter or longer than in the wild-type protein, whereas it complemented *pft1-2* early flowering in SDs only with the authentic length of the Q-rich region. As proteosomal degradation is crucial for PFT1 function, it has been suggested that reduced degradation in SDs could convert PFT1 from an activator to a repressor.

Regulators of *CO* on the chromatin level have also been described. The histone-binding protein MULTICOPY SUPPRESSOR OF IRA 1 (MSI1) is part of chromatin-modifying complexes and is needed for efficient activation of *CO* during photoperiodic flowering induction (Steinbach and Hennig, 2014).

Regulation of CO protein

In addition to the elaborate regulation of *CO* transcription, CO protein accumulation is restricted in non-inductive conditions until light exposure acts to overcome this inhibition (Fig. 2). CO protein is targeted for proteosomal degradation by a complex of the RING motif-containing E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC1

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

(COP1) and SUPPRESSOR OF PHYTOCHROME A-105 1 (SPA1) in darkness (Laubinger *et al.*, 2006; Jang *et al.*, 2008). Thus, the SPA1–COP1 complex prevents accumulation of CO in the second half of the day and throughout the night in SDs. Additionally, phyB activated by red light targets CO for degradation via an unknown ubiquitin ligase in the morning, and another RING finger E3 ligase, HIGH EXPRESSION OF OSMOTICALLY RESPONSIVE GENE1 (HOS1), binds to CO to destabilize it (Valverde *et al.*, 2004; Lazaro *et al.*, 2012).

In LDs, photoactivated CRYs reduce the activity of the COP1–SPA1 complex. CRY1 binds to the SPA1 C-terminus to prevent SPA1 from binding to COP1 (Lian *et al.*, 2011; Liu *et al.*, 2011), whereas CRY2 binds to the SPA1 N-terminus, promoting SPA1 binding to COP1 but repressing the SPA1–COP1 activity (Zuo *et al.*, 2011). In addition to CRY1 and CRY2, the far-red-absorbing PHYA contributes to CO stabilization (Valverde *et al.*, 2004). An interaction of COP1 with MIDGET, a regulator of a topoisomerase complex, is necessary for COP1/SPA1-controlled repression of flowering in SDs by an unknown mechanism (Schrader and Uhrig, 2013). Furthermore, FKF1 binds to CO in response to blue light, helping to stabilize CO in the afternoon (Song *et al.*, 2012*b*). Furthermore, PHYTOCHROME-DEPENDENT LATE-FLOWERING (PHL) accelerates flowering through red light-dependent interaction with phyB and CO, thus antagonizing the inhibitory effect of phyB on CO protein (Endo *et al.*, 2013).

Collectively, the appropriate phasing of *CO* relative to light and dark and the light-dependent stabilization of the CO protein in LDs show features of both the internal and the external coincidence models: synchronization of the endogenous *GI* and *FKF1* rhythms in LDs allows degradation of CDFs in LDs to create the *CO* transcript pattern, and regulation of FKF1 activity by blue light through its LOV domain and of COP1/SPA1 activity through CRYs at the peak of *CO* oscillations allows CO protein accumulation.

The importance of an endogenous rhythm matching the environment for proper flowering has also been shown by using light–dark cycles that are longer or shorter than 24 h and alter transcript oscillations relative to dawn and dusk (Roden *et al.*, 2002). This showed that the perception of daylength relies on adjustments of the phase angle of circadian rhythms relative to the light–dark cycle.

Co-ordination of *FT* transcription

A number of proteins collectively ensure expression of *FT* at the right time of the day (Fig. 3). An upstream region of 5.7 kb is required to drive sufficient *FT* expression to complement the *ft-10* late flowering mutant (Adrian *et al.*, 2010). CO binds to two so-called CO-responsive elements (COREs) in the promoter-proximal part via its CCT domain and activates *FT* transcription, presumably via a glutamic acid-rich region (Tiwari *et al.*, 2010). Additionally, CCAAT boxes at around –5.3 kb that are binding sites for trimeric NUCLEAR FACTOR Y (NF-Y) complexes enhance CO-mediated *FT* activation (Tiwari *et al.*, 2010). This effect is due to interaction of CO with the NF-YB and NF-YC subunits (Wenkel *et al.*, 2006; Cai *et al.*, 2007; Kumimoto *et al.*, 2008). Long-distance chromatin loops have been detected at the *FT* promoter that bring the distal enhancer elements close to the proximal promoter bound by CO (Cao *et al.*, 2014). These loops are preferentially formed at the end of the light period when CO activity and *FT* transcription peak. NF-Y complexes assembled at the distal CCAAT boxes thus may play a role in recruiting CO to the COREs.

The CDF proteins which are crucial repressors of *CO* also bind to the *FT* promoter to repress it in the morning (Song *et al.*, 2012*b*). Furthermore, FKF1 and GI also bind directly to the *FT* promoter (Sawa and Kay, 2011), and FKF1 may remove CDF1 to promote *FT* expression (Song *et al.*, 2012*b*). Thus, transcriptional timing of *CO* and of *FT* shares the same logic. Additionally, CRYPTOCHROME-INTERACTING BASIC-HELIX–LOOP–HELIX1 (CIB1) is activated by interaction with CRY2 and induces *FT* expression at the end of the day (Liu *et al.*, 2008). Thus, in addition to an indirect influence on *FT* activation via entrainment of the circadian clock and via blue light-dependent stabilization of GI and FKF1, CRY2 also directly regulates *FT*. Notably, two other blue light receptors, ZTL and LKP2, counteract proteosomal degradation of CIB1 in the absence of blue light (Liu *et al.*, 2013) (Fig. 4).

Furthermore, several factors have been identified that regulate *FT* chromatin. At the end of LDs, CO enables the

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

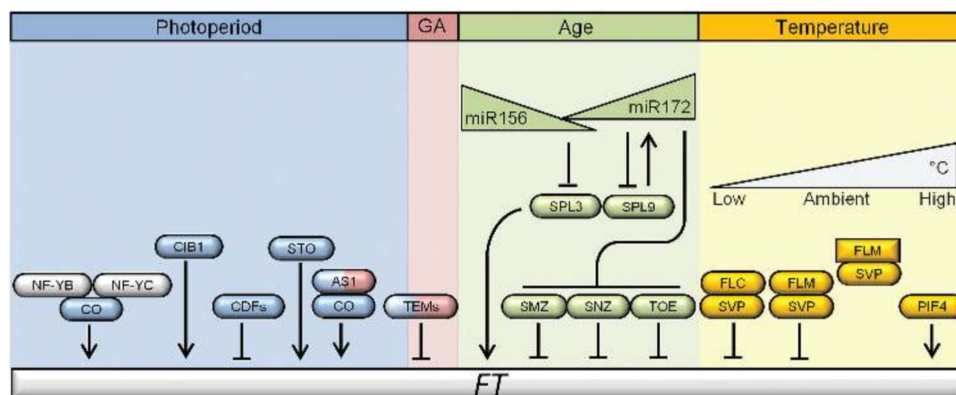

Fig. 3. Internal and exogenous cues converging on *FT*. Factors responding to photoperiod, gibberellin level, age, and temperature, respectively, that modulate *FT* transcription in the leaves are indicated. (This figure is available in colour at *JXB* online.)

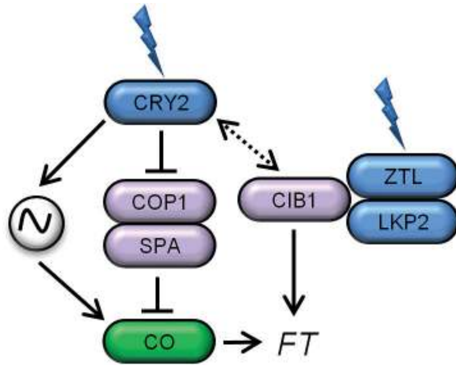

Fig. 4. Three modes of action of CRY2 in photoperiodic flower induction. CRY2 entrains the circadian clock that in turn mediates *CO* oscillations. CRY2 interacts with CIB1 to regulate FT transcription. CRY2 inhibits the activity of the COP1–SPA1 complex to prevent CO degradation in LDs. Arrows indicate activation; arrows with blunt ends represent repression; the broken arrow indicates protein interaction. (This figure is available in colour at *JXB* online.)

recruitment of histone deacetylases to *FT*, resulting in down-regulation of *FT* expression (Gu *et al.*, 2013). Thus, *CO* regulation by the photoperiodic pathway not only sets in motion *FT* expression but also leads to subsequent dampening, presumably to fine-tune *FT* levels. A Polycomb group complex directly represses *FT* during the night to prevent photoperiod-independent flowering (Y. Wang *et al.*, 2014). At dusk, its binding to *FT* chromatin is disrupted by the photoperiodic pathway, enabling *FT* activation.

The TEMPRANILLO (TEM1) and TEM2 transcription factors compete with CO for binding to the *FT* promoter and thus antagonize CO activity (Castillejo and Pelaz, 2008). TEM1 and TEM2 also interact with GI, suggesting that GI contributes to *FT* regulation also via regulation of the stability of *FT* repressors (Sawa and Kay, 2011). Moreover, TEM1 and TEM2 repress *GA3OXIDASE1* (*GA3OX1*) and *GA3OX2* encoding gibberellic acid (GA) biosynthetic enzymes and thus also repress flowering through the GA-dependent flowering pathway (Osnato *et al.*, 2012). GAs have been assigned a role in response to inductive LDs through activation of *FT* transcription in leaves and of the *SQUAMOSA PROMOTER BINDING PROTEIN LIKE* (*SPL*) genes in the shoot apical meristem (Porri *et al.*, 2012). Furthermore, ASYMMETRIC LEAVES 1 (AS1) binds to the *FT* promoter and interacts with CO to regulate *FT* expression (Song *et al.*, 2012*a*). AS1 also positively regulates *GA20OXIDASE* and thus may also affect *FT* indirectly via increased GA levels and derepression of *SPL3*.

Expression of *TEM1* and *TEM2* is promoted by SENSITIVITY TO RED LIGHT REDUCED1 (SRR1) (Johansson and Staiger, 2014). SRR1, a pioneer protein, was identified based on its role in red light signalling and clock regulation (Staiger *et al.*, 2003). Furthermore, its importance for flowering time control was noticed in natural conditions (Brachi *et al.*, 2010). SRR1 inhibits flowering in non-inductive conditions by promoting the expression of direct *FT* repressors, among them also *CDF1* and the major repressor of flowering, the MADS box transcription factor *FLOWERING LOCUS C* (*FLC*), and thus SRR1 influences flowering through photoperiod-dependent and -independent pathways.

Repression by FLC is also alleviated by exposure to cold temperatures during winter, enabling flowering upon increasing daylength in spring. This vernalization process involves epigenetic silencing of *FLC* and has been reviewed recently (Ream *et al.*, 2012; Song *et al.*, 2013). *FLC* is also down-regulated by endogenous regulators collectively referred to as the autonomous pathway, among them the chromatin modification factors FVE and FLOWERING LOCUS D, as well as the RNA-binding proteins FCA, FPA, and FLK (Rataj and Simpson, 2014). FLC and several components of the autonomous pathway have been shown to affect the period of the clock (Edwards *et al.*, 2006; Salathia *et al.*, 2006). A glycine-rich RNA-binding protein that affects flowering time through regulating *FLC* and which is rhythmically expressed, in turn regulates circadian transcripts, pointing to cross-talk between the circadian system and the floral-promoting network beyond photoperiodic timekeeping (Streitner *et al.*, 2008, 2010; Schmal *et al.*, 2013; Löhr *et al.*, 2014). Among other members of the B-box family that have been connected to photoperiodic regulation is SALT TOLERANT (STO) or BBX24 that has B1 and B2 B-boxes but no CCT domain. It is under circadian control and activates *FT* expression by competing with FLC (Li *et al.*, 2014). Recently, BBX19 that also has B1 and B2 B-boxes but no CCT domain and oscillates in antiphase to *CO* has been identified as a negative regulator of flowering in LDs (C.-Q. Wang *et al.*, 2014). Physical interaction of BBX19 with CO leads to reduced *FT* expression, suggesting that BBX19 acts through a novel mechanism, sequestering CO to prevent premature *FT* activation.

As well as low temperature, ambient temperature also has a decisive role in flowering time. SHORT VEGETATIVE PHASE (SVP) represses *FT* via direct binding to the promoter. Another MADS-box protein, FLOWERING LOCUS M, occurs as two temperature-dependent alternative splice variants that differentially interact with SVP to repress *FT* preferentially at lower temperatures (Pose *et al.*, 2013). PHYTOCHROME-INTERACTING FACTOR 4 (PIF4) mediates early flowering at high temperatures by binding to the *FT* promoter (Kumar *et al.*, 2012). Depending on the time of day of warm temperature exposure, flowering is regulated via FT-dependent and FT-independent pathways (Thines *et al.*, 2014).

FT expression during development

Among components that regulate *FT* expression during plant development are microRNAs (miRNAs). An age-dependent pathway of flower induction is defined by miRNA156 (Wang *et al.*, 2009). While early in development, during the juvenile vegetative phase, *Arabidopsis* is refractory to flowering cues, it becomes responsive during the adult vegetative phase. A high miR156 level in young plants leads to cleavage of the *SPL* transcripts. miR156 decreases with development as a result of sugars accumulating through photosynthesis (Yang *et al.*, 2013; Yu *et al.*, 2013). Declining miR156 levels then allow SPL3 to accumulate and bind to the *FT* promoter to

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

activate transcription. Another miR156 target, SPL9, activates *MIR172* transcription. Thus, miR172 levels increase with age, with a concomitant decrease in its targets, the *FT* repressors SCHLAFMUETZE, SCHNARCHZAPFEN, and TARGET OF EAT1, 2, and 3 (Mathieu *et al.*, 2009). GI also stimulates increased miR172 levels in LDs, defining a CO-independent branch of the photoperiodic pathway (Jung *et al.*, 2007). Furthermore, *TEM1* levels inversely correlate with *FT* during early development, and TEM1 represses *FT* during the juvenile phase (Sgamma *et al.*, 2014), and levels of *FLC* and *SVP* also decrease with leaf age (Li *et al.*, 2008).

FT protein function

FT and TSF are members of a gene family with similarity to phosphatidylethanolamine-binding proteins in mammals. A large body of work demonstrated that FT protein is the mobile signal conveying the output of the photoperiodic pathway in the leaf to the site of flower initiation (Turck *et al.*, 2008). While FT is small enough to move passively through plasmodesmata, a first regulator of FT trafficking has been described. FT-INTERACTING PROTEIN1 (FTIP1) is an endoplasmic reticulum (ER) membrane protein that regulates FT transport from phloem companion cells into sieve elements, and genetic evidence suggests that FTIP1 may also act to transport TSF (Liu *et al.*, 2012). Mutation of D^{17} and V^{18} impaired the ability of FT to induce flowering when expressed specifically in companion cells, suggesting that these residues control the interaction with a regulator of transport (Ho and Weigel, 2014).

FT shares ~60% identity with TERMINAL FLOWER1 (TFL1) that represses flowering, raising the question about the difference between the antagonistic proteins. Notably, mutation of a single residue, Y^{85}, in FT to H conferred a weak TFL1 activity, whereas the reciprocal mutation of H^{88} in TFL1 to Y conferred a weak FT-like activity (Hanzawa *et al.*, 2005). Subsequently, the difference between the two proteins was largely attributed to an external loop (Ahn *et al.*, 2006). Evidence for the critical role of the loop comes from *Beta vulgaris* that harbours two FT orthologues, BvFT1 with repressing function and BvFT2 with activating function. Swapping the loop domains with three amino acid differences converted BvFT1 into an activator, whereas the converse mutations convert BvFT2 into a repressor (Pin *et al.*, 2010).

Similarly, the obligate SD plant *Chrysanthemum seticuspe* harbours FT-like proteins with antagonistic functions. *CsFTL3* is activated in SDs and promotes flowering (Oda *et al.*, 2012). *Chrysanthemum seticuspe ANTI-FLORIGENIC FT* (*CsAFT*) is activated in non-inductive LDs and represses flowering (Higuchi *et al.*, 2013). The antagonism of CsAFT and CsFTL3 occurs through competing for CsFDL1, an FD homologue. Importantly, CsAFT acts systemically and thus represents an 'antiflorigen' (Fig. 5).

Recently, the Weigel laboratory undertook a large-scale functional screen of mutagenized FT proteins (Ho and Weigel, 2014). Mutations that transformed FT into a TFL1 mimic altered the surface charge. Whereas this did not impair interactions with FD, FD PARALOGUE, and 14-3-3, the interaction with several TEOSINTE BRANCHED, CYCLOIDEA, PCF (TCP) transcription factors was affected. Previously, FT has been shown to interact with BRANCHED1/TCP18 that represses differentiation of axillary meristems and delays flowering (Niwa *et al.*, 2013).

Based on the homology of FT to phosphatidylethanolamine-binding proteins, a putative phospholipid binding activity was monitored, unravelling binding of FT to diurnally oscillating phosphatidylcholine but not to phosphatidylethanolamine *in vitro* (Nakamura *et al.*, 2014). An increased ratio of phosphatidylcholine to phosphatidylethanolamine causes early flowering at least partially dependent on FT and TSF, providing a first hint that FT phospholipid binding may be relevant for its *in vivo* function.

Photoperiodic regulation in crop species

Understanding seasonal control of flowering beyond *Arabidopsis* is of major interest, as it may ultimately allow the breeding of crop varieties that cope better with changing climate conditions (Jung and Müller, 2009). Through mutant analysis and whole-genome sequencing, homologues of *Arabidopsis* photoperiodic regulators have been identified with similar but also divergent functions, and additional components absent in *Arabidopsis* contribute to photoperiodic flowering time control (Andres and Coupland, 2012).

The LD plant *Pisum sativum* has been used for some time as a model to study the control of flowering time and mobile flowering signals (Weller *et al.*, 1997). Several of the genetic loci associated with the photoperiodic response encode orthologues of *Arabidopsis* clock genes. LATE BLOOMER1 corresponds to GI, DIE NEUTRALIS corresponds to ELF4, HIGH RESPONSE TO PHOTOPERIOD corresponds to ELF3, and STERILE NODES corresponds to LUX (Hecht *et al.*, 2007; Liew *et al.*, 2009, 2014; Weller *et al.*, 2012). Furthermore, the characterization of several *FT* family members provided evidence for a more complex scenario of flower

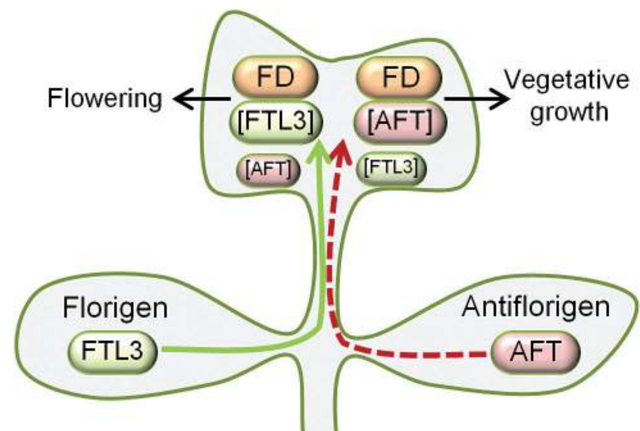

Fig. 5. Antagonistic function of CsFTL3 with florigen activity and CsAFT with antiflorigen activity. CsFTL3 is produced in leaves in inductive SDs and moves systemically to the shoot apex to interact with CsFDL to promote flowering. CsAFT is synthesized in leaves in non-inductive LDs and moves systemically to the shoot apex to compete for binding to CsFDL and inhibit flowering (Higuchi *et al.*, 2013). (This figure is available in colour at *JXB* online.)

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

induction in pea compared with *Arabidopsis*, relying on cross-regulation among different *FT* genes with distinct expression patterns and functions, including at least two mobile signals (Hecht *et al.*, 2011).

In the LD plant *Hordeum vulgare*, the closest orthologue of CO, HvCO1, causes up-regulation of *HvFT1* (Campoli *et al.*, 2011). However, HvCO1 promotes flowering in both SDs and LDs and thus does not mediate the response to photoperiod. Rather, photoperiodic flowering is controlled by the *PHOTOPERIOD1* (*Ppd-H1*) gene, which is homologous to the *Arabidopsis* clock gene *PRR7* (Turner *et al.*, 2005). Ppd-H1 promotes expression of *HvFT1* independently of HvCO1 but is at the same time also regulated by HvCO. In contrast to PRR7, Ppd-H1 does not have a prominent effect on clock gene expression (Campoli *et al.*, 2012).

Loss of function of the barley ELF3 homologue results in rapid flowering in both SDs and LDs (Faure *et al.*, 2012). HvELF3 is necessary to maintain photoperiodic sensitivity in spring barley through repression of *HvFT1* and production of active GAs, whereas *Arabidopsis* ELF3 has not been implicated in GA metabolism (Boden *et al.*, 2014). The HvPhyC photoreceptor has been found to promote floral transition by up-regulating *HvFT1* independently of the circadian clock and of HvCO1 (Nishida *et al.*, 2013). In contrast, another study showed that HvPhyC indeed affects circadian oscillations and interacts with the Ppd-H1 pathway to promote flowering (Pankin *et al.*, 2014). This is similar to wheat PhyC that activates *PPD1* and *FT1* in inductive LDs (Chen *et al.*, 2014).

In rice, the photoperiodic pathway is of major importance as rice does not require vernalization. Although flowering in SDs, rice shares many features with the photoperiodic pathway in *Arabidopsis* (Shrestha *et al.*, 2014). The orthologue of GI, *OsGI*, is rhythmically expressed and activates expression of *Heading Date 1* (*Hd1*), the *CO* orthologue, in both SDs and LDs (Hayama *et al.*, 2003). *Hd1* shows a similar rhythm to *Arabidopsis CO* but it has opposing effects on the *FT* orthologue *Heading Date 3a* (*Hd3a*), activating *Hd3a* in inductive SDs and repressing it in non-inductive LDs. Thus, Hd1 appears to be converted to a floral repressor by light exposure in LDs, and coincidence of the *Hd1* peak with darkness triggers flowering (Ishikawa *et al.*, 2011). In contrast to *Arabidopsis*, a pathway operating in parallel to the *OsGI-Hd1* module also activates *Hd3a*. *EARLY HEADING DATE 1* (*Ehd1*), a response regulator without a correlate in *Arabidopsis*, activates *RFT1*, another *FT* orthologue (Doi *et al.*, 2004). RTF1 promotes flowering in SDs but also in LDs, adjusting flowering to environments differing in photoperiod. The Ehd1–RTF1 pathway is repressed by GRAIN NUMBER, PLANT HEIGHT AND HEADING DATE7 (Ghd7), a CCT domain protein (Xue *et al.*, 2008). The rice ELF3 homologue, OsELF3, activates *Ehd1* to promote flowering in SDs and represses *Ghd7* to promote flowering in LDs (Zhao *et al.*, 2012).

The SD plant *Sorghum* uses an orthologue of Ehd1, SbEHD1, to activate flowering in SDs (Yang *et al.*, 2014). SbEHD1 itself is activated via the SbGI–SbCO module in SDs. PRR37 inhibits flowering in LDs by inhibiting *SbEHD1* and CO activity. Thus, PRR37 acts as a repressor of flowering in sorghum.

In *Beta vulgaris* that occurs in both an annual and a biennial form, BOLTING TIME CONTROL1 (BvBTC1) is a key regulator (Pin *et al.*, 2012). The biennial variety contains a recessive *Bvbtc1* allele and requires vernalization to flower in LDs. The annual variety contains a dominant BvBTC1 allele that promotes bolting independently of vernalization through inhibition of the repressor *BvFT1* and activation of *BvFT2*. While not much is known about circadian control in beet, *BvBTC1* shows homology to the clock component *PRR7*. Recently, an additional regulator of bolting time was identified that oscillates across the day and also acts upstream of *BvFT1* and *BvFT2* (Dally *et al.*, 2014). Its closest homologue in *Arabidopsis* is BBX19, with two B-box zinc fingers similar to CO but without a CCT domain. It has been proposed that BvBBX19 with its two B-box zinc fingers and BvBTC1 with a CCT domain complement each other through interaction to fulfil a function similar to *Arabidopsis* CO with both B-box protein interaction domains and a CCT DNA-binding domain combined (Dally *et al.*, 2014). As described above, protein–protein interaction between *Arabidopsi*s BBX19 and CO antagonizes CO activity (C.-Q. Wang *et al.*, 2014). A detailed comparison of the mode of action of how beet BvBBX19 and BBX19 contribute to fine-tune the expression of *FT* genes will provide valuable insights into flowering time regulation in annual and biennial varieties.

Conclusion

Photoperiodic flower induction has been extensively studied on the molecular level. Key elements are promotive and repressive factors that shape the daily expression pattern of the floral regulators CO and FT by affecting transcription, chromatin structure, protein stability, and protein interaction. Other flowering time pathway promote flowering through disabling repressors of *FT*. A common theme is transcriptional up-regulation of *FT* in inductive photoperiods. While the CO–FT module is conserved between LD and SD plants, the molecular details leading to *FT* transcription are distinct and provide the basis for differential response to SDs or LDs. New components impacting the photoperiodic response continue to be discovered in crop plants, but also in *Arabidopsis*. These will have to be characterized functionally and placed into the photoperiodic pathway.

Acknowledgements

MJ is supported by an Alexander von Humboldt fellowship. Research in our laboratory is supported by the DFG (STA653 and SPP1530).

References

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

Adrian J, Farrona S, Reimer JJ, Albani MC, Coupland G, Turck F. 2010. cis-Regulatory elements and chromatin state coordinately control

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

temporal and spatial expression of FLOWERING LOCUS T in *Arabidopsis*. *The Plant Cell* **22,** 1425–1440.

Ahn JH, Miller D, Winter VJ, Banfield MJ, Lee JH, Yoo SY, Henz SR, Brady RL, Weigel D. 2006. A divergent external loop confers antagonistic activity on floral regulators FT and TFL1. *EMBO Journal* **25,** 604–614.

Alabadi D, Oyama T, Yanovsky MJ, Harmon FG, Mas P, Kay SA. 2001. Reciprocal regulation between TOC1 and LHY/CCA1 within the *Arabidopsis* circadian clock. *Science* **293,** 880–883.

Andres F, Coupland G. 2012. The genetic basis of flowering responses to seasonal cues. *Nature Reviews Genetics* **13,** 627–639.

Boden SA, Weiss D, Ross JJ, Davies NW, Trevaskis B, Chandler PM, Swain SM. 2014. EARLY FLOWERING3 regulates flowering in spring barley by mediating gibberellin production and FLOWERING LOCUS T expression. *The Plant Cell* **26,** 1557–1569.

Brachi B, Faure N, Horton M, Flahauw E, Vazquez A, Nordborg M, Bergelson J, Cuguen J, Roux F. 2010. Linkage and association mapping of *Arabidopsis thaliana* flowering time in nature. *PLoS Genetics* **6,** e1000940.

Bünning E. 1936. Die endogene Tagesrhythmik als Grundlage der photoperiodischen Reaktion. *Berichte der Deutschen Botanischen Gessellschaft* **54,** 590–607.

Cai X, Ballif J, Endo S, Davis E, Liang M, Chen D, DeWald D, Kreps J, Zhu T, Wu Y. 2007. A putative CCAAT-binding transcription factor is a regulator of flowering timing in *Arabidopsis*. *Plant Physiology* **145,** 98–105.

Campoli C, Drosse B, Searle I, Coupland G, von Korff M. 2011. Functional characterisation of HvCO1, the barley (*Hordeum vulgare*) flowering time ortholog of CONSTANS. *The Plant Journal* **71,** 173–191.

Campoli C, Shtaya M, Davis SJ, von Korff M. 2012. Expression conservation within the circadian clock of a monocot: natural variation at barley Ppd-H1 affects circadian expression of flowering time genes, but not clock orthologs. *BMC Plant Biology* **12,** 97.

Cao S, Kumimoto RW, Gnesutta N, Calogero AM, Mantovani R, Holt BF. 2014. A distal CCAAT/NUCLEAR FACTOR Y complex promotes chromatin looping at the FLOWERING LOCUS T promoter and regulates the timing of flowering in *Arabidopsis*. *The Plant Cell* **26,** 1009–1017.

Carre I, Veflingstad S. 2013. Emerging design principles in the Arabidopsis circadian clock. *Seminars in Cell and Developmental Biology* **24,** 393–398.

Castillejo C, Pelaz S. 2008. The balance between CONSTANS and TEMPRANILLO activities determines FT expression to trigger flowering. *Current Biology* **18,** 1338–1343.

Chailakhyan MK. 1936. New facts in support of the hormonal theory of plant development. *Compte Rendu de l'Academy des Sciencesde L' URSS* **13,** 79–83.

Chen A, Li C, Hu W, Lau MY, Lin H, Rockwell NC, Martin SS, Jernstedt JA, Lagarias JC, Dubcovsky J. 2014. PHYTOCHROME C plays a major role in the acceleration of wheat flowering under long-day photoperiod. *Proceedings of the National Academy of Sciences, USA* **111,** 10037–10044.

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

Dally N, Xiao K, Holtgräwe D, Jung C. 2014. The B2 flowering time locus of beet encodes a zinc finger transcription factor. *Proceedings of the National Academy of Sciences, USA* **111**. 10365–10370.

Devlin PF. 2002. Signs of the time: environmental input to the circadian clock. *Journal of Experimental Botany* **53,** 1535–1550.

Dixon LE, Knox K, Kozma-Bognar L, Southern MM, Pokhilko A, Millar AJ. 2011. Temporal repression of core circadian genes is mediated through EARLY FLOWERING 3 in Arabidopsis. *Current Biology* **25,** 120–125.

Dodd AN, Salathia N, Hall A, Kevei E, Toth R, Nagy F, Hibberd JM, Millar AJ, Webb AA. 2005. Plant circadian clocks increase photosynthesis, growth, survival, and competitive advantage. *Science* **309,** 630–633.

Doi K, Izawa T, Fuse T, Yamanouchi U, Kubo T, Shimatani Z, Yano M, Yoshimura A. 2004. Ehd1, a B-type response regulator in rice, confers short-day promotion of flowering and controls FT-like gene expression independently of Hd1. *Genes and Development* **18,** 926–936.

Edwards KD, Anderson PE, Hall A, Salathia NS, Locke JC, Lynn JR, Straume M, Smith JQ, Millar AJ. 2006. FLOWERING LOCUS C mediates natural variation in the high-temperature response of the *Arabidopsis* circadian clock. *The Plant Cell* **18,** 639–650.

Endo M, Tanigawa Y, Murakami T, Araki T, Nagatani A. 2013. PHYTOCHROME-DEPENDENT LATE-FLOWERING accelerates flowering through physical interactions with phytochrome B and CONSTANS. *Proceedings of the National Academy of Sciences, USA* **110,** 18017–18022.

Fankhauser C, Staiger D. 2002. Photoreceptors in *Arabidopsis thaliana*: light perception, signal transduction and entrainment of the endogenous clock. *Planta* **216,** 1–16.

Farinas B, Mas P. 2011. Functional implication of the MYB transcription factor RVE8/LCL5 in the circadian control of histone acetylation. *The Plant Journal* **66,** 318–329.

Faure S, Turner AS, Gruszka D, Christodoulou V, Davis SJ, von Korff M, Laurie DA. 2012. Mutation at the circadian clock gene EARLY MATURITY 8 adapts domesticated barley (Hordeum vulgare) to short growing seasons. *Proceedings of the National Academy of Sciences, USA* **109,** 8328–8333.

Fogelmark K, Troein C. 2014. Rethinking transcriptional activation in the *Arabidopsis* circadian clock. *PLoS Computational Biology* **10,** e1003705.

Fornara F, Panigrahi KC, Gissot L, Sauerbrunn N, Ruhl M, Jarillo JA, Coupland G. 2009. Arabidopsis DOF transcription factors act redundantly to reduce CONSTANS expression and are essential for a photoperiodic flowering response. *Developmental Cell* **17,** 75–86.

Fowler S, Lee K, Onouchi H, Samach A, Richardson K, Morris B, Coupland G, Putterill J. 1999. GIGANTEA: a circadian clock-controlled gene that regulates photoperiodic flowering in *Arabidopsis* and encodes a protein with several possible membrane-spanning domains. *EMBO Journal* **18,** 4679–4688.

Garner WW, Allard HA. 1920. Effects of the relative length of day and night and other factors of the environment on growth and reproduction in plants. *Journal of Agricultural Reseach* **18,** 223–606.

Gendron JM, Pruneda-Paz JL, Doherty CJ, Gross AM, Kang SE, Kay SA. 2012. Arabidopsis circadian clock protein, TOC1, is a DNA-binding transcription factor. *Proceedings of the National Academy of Sciences, USA* **109,** 3167–3172.

Green RM, Tingay S, Wang Z-Y, Tobin EM. 2002. Circadian rhythms confer a higher level of fitness to *Arabidopsis* plants. *Plant Physiology* **129,** 576–584.

Gu X, Wang Y, He Y. 2013. Photoperiodic regulation of flowering time through periodic histone deacetylation of the florigen gene FT. *PLoS Biology* **11,** e1001649.

Hanzawa Y, Money T, Bradley D. 2005. A single amino acid converts a repressor to an activator of flowering. *Proceedings of the National Academy of Sciences, USA* **102,** 7748–7753.

Hayama R, Yokoi S, Tamaki S, Yano M, Shimamoto K. 2003. Adaptation of photoperiodic control pathways produces short-day flowering in rice. *Nature* **422,** 719–722.

Hecht V, Knowles CL, Vander Schoor JK, Liew LC, Jones SE, Lambert MJM, Weller JL. 2007. Pea LATE BLOOMER 1 is a GIGANTEA ortholog with roles in photoperiodic flowering, de-etiolation and transcriptional regulation of circadian clock gene homologs. *Plant Physiology* **144,** 648–661.

Hecht V, Laurie RE, Vander Schoor JK, Ridge S, Knowles CL, Liew LC, Sussmilch FC, Murfet IC, Macknight RC, Weller JL. 2011. The pea GIGAS gene is a FLOWERING LOCUS T homolog necessary for graft-transmissible specification of flowering but not for responsiveness to photoperiod. *The Plant Cell* **23,** 147–161.

Herrero E, Kolmos E, Bujdoso N, *et al*. 2012. EARLY FLOWERING4 recruitment of EARLY FLOWERING3 in the nucleus sustains the Arabidopsis circadian clock. *The Plant Cell* **24,** 428–443.

Hicks KA, Millar AJ, Carre IA, Somers DE, Straume M, Meeks-Wagner DR, Kay SA. 1996. Conditional circadian dysfunction of the *Arabidopsis early-flowering 3* mutant. *Science* **274,** 790–792.

Higuchi Y, Narumi T, Oda A, Nakano Y, Sumitomo K, Fukai S, Hisamatsu T. 2013. The gated induction system of a systemic floral inhibitor, antiflorigen, determines obligate short-day flowering in chrysanthemums. *Proceedings of the National Academy of Sciences, USA* **110,** 17137–17142.

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

Ho WWH, Weigel D. 2014. Structural features determining flower-promoting activity of Arabidopsis FLOWERING LOCUS T. *The Plant Cell* **26,** 552–564.

Hsu PY, Devisetty UK, Harmer SL. 2013. Accurate timekeeping is controlled by a cycling activator in *Arabidopsis*. *eLife* **2,** e00473.

Huang W, Perez-Garcia P, Pokhilko A, Millar AJ, Antoshechkin I, Riechmann JL, Mas P. 2012. Mapping the core of the *Arabidopsis* circadian clock defines the network structure of the oscillator. *Science* **336,** 75–79.

Imaizumi T, Schultz TF, Harmon FG, Ho LA, Kay SA. 2005. FKF1 F-box protein mediates cyclic degradation of a repressor of CONSTANS in *Arabidopsis*. *Science* **309,** 293–297.

Inigo S, Alvarez MJ, Strasser B, Califano A, Cerdan PD. 2011. PFT1, the MED25 subunit of the plant Mediator complex, promotes flowering through CONSTANS dependent and independent mechanisms in *Arabidopsis*. *The Plant Journal* **69,** 601–612.

Ishikawa R, Aoki M, Kurotani K-i, Yokoi S, Shinomura T, Takano M, Shimamoto K. 2011. Phytochrome B regulates Heading date 1 (Hd1)-mediated expression of rice florigen Hd3a and critical day length in rice. *Molecular Genetics and Genomics* **285,** 461–470.

Ito S, Song YH, Josephson-Day AR, Miller RJ, Breton G, Olmstead RG, Imaizumi T. 2012. FLOWERING BHLH transcriptional activators control expression of the photoperiodic flowering regulator CONSTANS in *Arabidopsis*. *Proceedings of the National Academy of Sciences, USA* **109,** 3582–3587.

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

Jang S, Marchal V, Panigrahi KC, Wenkel S, Soppe W, Deng XW, Valverde F, Coupland G. 2008. *Arabidopsis* COP1 shapes the temporal pattern of CO accumulation conferring a photoperiodic flowering response. *EMBO Journal* **27,** 1277–1288.

Johansson M, Staiger D. 2014. SRR1 is essential to repress flowering in non-inductive conditions in *Arabidopsis thaliana*. *Journal of Experimental Botany* **65,** 5811–5820.

Jung C, Müller AE. 2009. Flowering time control and applications in plant breeding. *Trends in Plant Science* **14,** 563–573.

Jung JH, Seo YH, Seo PJ, Reyes JL, Yun J, Chua NH, Park CM. 2007. The GIGANTEA-regulated MicroRNA172 mediates photoperiodic flowering independent of CONSTANS in Arabidopsis. *The Plant Cell* **19,** 2736–2748.

Kim J, Geng R, Gallenstein RA, Somers DE. 2013. The F-box protein ZEITLUPE controls stability and nucleocytoplasmic partitioning of GIGANTEA. *Development* **140,** 4060–4069.

Kim W-Y, Fujiwara S, Suh S-S, Kim J, Kim Y, Han L, David K, Putterill J, Nam HG, Somers DE. 2007. ZEITLUPE is a circadian photoreceptor stabilized by GIGANTEA in blue light. *Nature* **449,** 356–360.

Kim Y, Lim J, Yeom M, Kim H, Kim J, Wang L, Kim Woe Y, Somers David E, Nam Hong G. 2013. ELF4 regulates GIGANTEA chromatin access through subnuclear sequestration. *Cell Reports* **3,** 671–677.

Koornneef M, Hanhart CJ, van der Veen JH. 1991. A genetic and physiological analysis of late flowering mutants in *Arabidopsis thaliana*. *Molecular and General Genetics* **229,** 57–66.

Kumar SV, Lucyshyn D, Jaeger KE, Alos E, Alvey E, Harberd NP, Wigge PA. 2012. Transcription factor PIF4 controls the thermosensory activation of flowering. *Nature* **484,** 242–245.

Kumimoto RW, Adam L, Hymus GJ, Repetti PP, Reuber TL, Marion CM, Hempel FD, Ratcliffe OJ. 2008. The Nuclear Factor Y subunits NF-YB2 and NF-YB3 play additive roles in the promotion of flowering by inductive long-day photoperiods in Arabidopsis. *Planta* **228,** 709–723.

Lang A, Melchers G. 1943. Die photoperiodische Reaktion von *Hyoscyamus niger*. *Planta* **33,** 653–702.

Lau Os, Huang X, Charron J-B, Lee J-H, Li G, Deng XW. 2011. Interaction of Arabidopsis DET1 with CCA1 and LHY in mediating transcriptional repression in the plant circadian clock. *Molecular Cell* **43,** 703–712.

Laubinger S, Marchal V, Gentilhomme J, Wenkel S, Adrian J, Jang S, Kulajta C, Braun H, Coupland G, Hoecker U. 2006. Arabidopsis SPA proteins regulate photoperiodic flowering and interact with the floral inducer CONSTANS to regulate its stability. *Development* **133,** 3213–3222.

Lazaro A, Valverde F, Piñeiro M, Jarillo JA. 2012. The Arabidopsis E3 ubiquitin ligase HOS1 negatively regulates CONSTANS abundance in the photoperiodic control of flowering. *The Plant Cell* **24,** 982–999.

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

Li F, Sun J, Wang D, Bai S, Clarke AK, Holm M. 2014. The B-Box Family Gene STO (BBX24) in *Arabidopsis thaliana* regulates flowering time in different pathways. *PLoS One* **9,** e87544.

Li G, Siddiqui H, Teng Y, *et al*. 2011. Coordinated transcriptional regulation underlying the circadian clock in Arabidopsis. *Nature Cell Biology* **13,** 616–622.

Lian HL, He SB, Zhang YC, Zhu DM, Zhang JY, Jia KP, Sun SX, Li L, Yang HQ. 2011. Blue-light-dependent interaction of cryptochrome 1 with SPA1 defines a dynamic signaling mechanism. *Genes and Development* **25,** 1023–1028.

Liew LC, Hecht V, Laurie RE, Knowles CL, Vander Schoor JK, Macknight RC, Weller JL. 2009. DIE NEUTRALIS and LATE BLOOMER 1 contribute to regulation of the pea circadian clock. *The Plant Cell* **21,** 3198–3211.

Liew LC, Hecht V, Sussmilch FC, Weller JL. 2014. The pea photoperiod response gene STERILE NODES is an ortholog of LUX ARRHYTHMO. *Plant Physiology* **165,** 648–657.

Liu B, Zuo Z, Liu H, Liu X, Lin C. 2011. Arabidopsis cryptochrome 1 interacts with SPA1 to suppress COP1 activity in response to blue light. *Genes and Development* **25,** 1029–1034.

Liu H, Wang Q, Liu Y, Zhao X, Imaizumi T, Somers DE, Tobin EM, Lin C. 2013. Arabidopsis CRY2 and ZTL mediate blue-light regulation of the transcription factor CIB1 by distinct mechanisms. *Proceedings of the National Academy of Sciences, USA* **110,** 17582–17587.

Liu H, Yu X, Li K, Klejnot J, Yang H, Lisiero D, Lin C. 2008. Photoexcited CRY2 interacts with CIB1 to regulate transcription and floral initiation in *Arabidopsis*. *Science* **322,** 1535–1539.

Liu L, Liu C, Hou X, Xi W, Shen L, Tao Z, Wang Y, Yu H. 2012. FTIP1 is an essential regulator required for florigen transport. *PLoS Biology* **10,** e1001313.

Löhr B, Streitner C, Steffen A, Lange T, Staiger D. 2014. A glycine-rich RNA-binding protein affects gibberellin biosynthesis in *Arabidopsis.* *Molecular Biology Reports* **41,** 439–445.

Lu SX, Webb CJ, Knowles SM, Kim SH, Wang ZY, Tobin EM. 2012. CCA1 and ELF3 interact in the control of hypocotyl length and flowering time in *Arabidopsis*. *Plant Physiology* **158,** 1079–1088.

Mas P. 2008. Circadian clock function in Arabidopsis thaliana: time beyond transcription. *Trends in Cell Biology* **18,** 273–281.

Mas P, Kim WY, Somers DE, Kay SA. 2003. Targeted degradation of TOC1 by ZTL modulates circadian function in Arabidopsis thaliana. *Nature* **426,** 567–570.

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

Mathieu J, Yant LJ, Murdter F, Kuttner F, Schmid M. 2009. Repression of flowering by the miR172 target SMZ. *PLoS Biology* **7,** e1000148.

Matsushika A, Makino S, Kojima M, Mizuno T. 2000. Circadian waves of expression of the APRR1/TOC1 family of pseudo-response regulators in *Arabidopsis thaliana*: insight into the plant circadian clock. *Plant and Cell Physiology* **41,** 1002–1012.

McClung CR. 2013. Beyond *Arabidopsis*: the circadian clock in non-model plant species. *Seminars in Cell and Developmental Biology* **24,** 430–436.

Morris K, Thornber S, Codrai L, Richardson C, Craig A, Sadanandom A, Thomas B, Jackson S. 2010. DAY NEUTRAL FLOWERING represses CONSTANS to prevent Arabidopsis flowering early in short days. *The Plant Cell* **22,** 1118–1128.

Nakamichi N, Kiba T, Henriques R, Mizuno T, Chua NH, Sakakibara H. 2010. PSEUDO-RESPONSE REGULATORS 9, 7, and 5 are transcriptional repressors in the Arabidopsis circadian clock. *The Plant Cell* **22,** 594–605.

Nakamura Y, Andrés F, Kanehara K, Liu Y-c, Dörmann P, Coupland G. 2014. *Arabidopsis* florigen FT binds to diurnally oscillating

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

phospholipids that accelerate flowering. *Nature Commununications* **5,** 3553.

Nishida H, Ishihara D, Ishii M, *et al*. 2013. Phytochrome C is a key factor controlling long-day flowering in barley. *Plant Physiology* **163,** 804–814.

Niwa M, Daimon Y, Kurotani K-i, *et al.* 2013. BRANCHED1 interacts with FLOWERING LOCUS T to repress the floral transition of the axillary meristems in Arabidopsis. *The Plant Cell* **25,** 1228–1242.

Nusinow DA, Helfer A, Hamilton EE, King JJ, Imaizumi T, Schultz TF, Farre EM, Kay SA. 2011. The ELF4–ELF3–LUX complex links the circadian clock to diurnal control of hypocotyl growth. *Nature* **475,** 398–402.

Oda A, Narumi T, Li T, Kando T, Higuchi Y, Sumitomo K, Fukai S, Hisamatsu T. 2012. CsFTL3, a chrysanthemum FLOWERING LOCUS T-like gene, is a key regulator of photoperiodic flowering in chrysanthemums. *Journal of Experimental Botany* **63,** 1461–1477.

Osnato M, Castillejo C, Matias-Hernandez L, Pelaz S. 2012. TEMPRANILLO genes link photoperiod and gibberellin pathways to control flowering in *Arabidopsis*. *Nature Communictions* **3,** 808.

Pankin A, Campoli C, Dong X, *et al*. 2014. Mapping-by-sequencing identifies HvPHYTOCHROME C as a candidate gene for the early maturity 5 locus modulating the circadian clock and photoperiodic flowering in barley. *Genetics* **198,** 383–396.

Pin PA, Benlloch R, Bonnet D, Wremerth-Weich E, Kraft T, Gielen JJ, Nilsson O. 2010. An antagonistic pair of FT homologs mediates the control of flowering time in sugar beet. *Science* **330,** 1397–1400.

Pin PA, Zhang W, Vogt SH, *et al*. 2012. The role of a pseudo-response regulator gene in life cycle adaptation and domestication of beet. *Current Biology* **22,** 1095–1101.

Pittendrigh CS. 1972. Circadian surfaces and the diversity of possible roles of circadian organization in photoperiodic induction. *Proceedings of the National Academy of Sciences, USA* **69,** 2734–2737.

Pittendrigh CS, Minis DH. 1964. The entrainment of circadian oscillations by light and their role as photoperiodic clocks. *American Naturalist* **98,** 261–294.

Porri A, Torti S, Romera-Branchat M, Coupland G. 2012. Spatially distinct regulatory roles for gibberellins in the promotion of flowering of *Arabidopsis* under long photoperiods. *Development* **139,** 2198–2209.

Pose D, Verhage L, Ott F, Yant L, Mathieu J, Angenent GC, Immink RGH, Schmid M. 2013. Temperature-dependent regulation of flowering by antagonistic FLM variants. *Nature* **503,** 414–417.

Pruneda-Paz JL, Breton G, Nagel DH, Kang SE, Bonaldi K, Doherty CJ, Ravelo S, Galli M, Ecker JR, Kay SA. 2014. A genome-scale resource for the functional characterization of *Arabidopsis* transcription factors. *Cell Reports* **8,** 622–632.

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

Rataj K, Simpson GG. 2014. Message ends: RNA 3' processing and flowering time control. *Journal of Experimental Botany* **65,** 353–363.

Rawat R, Takahashi N, Hsu PY, Jones MA, Schwartz J, Salemi MR, Phinney BS, Harmer SL. 2011. REVEILLE8 and PSEUDO-REPONSE REGULATOR5 form a negative feedback loop within the *Arabidopsis* circadian clock. *PLoS Genet* **7,** e1001350.

Ream TS, Woods DP, Amasino RM. 2012. The molecular basis of vernalization in different plant groups. *Cold Spring Harbor Symposia on Quantitative Biology* **77,** 105–115.

Redei GP. 1962. Supervital mutants of *Arabidopsis*. *Genetics* **47,** 443–460.

Rival P, Press MO, Bale J, Grancharova T, Undurraga SF, Queitsch C. 2014. The conserved PFT1 tandem repeat is crucial for proper flowering in Arabidopsis thaliana. *Genetics* **198,** 747–754.

Roden LC, Song HR, Jackson S, Morris K, Carre IA. 2002. Floral responses to photoperiod are correlated with the timing of rhythmic expression relative to dawn and dusk in Arabidopsis. *Proceedings of the National Academy of Sciences, USA* **99,** 13313–13318.

Rosas U, Mei Y, Xie Q, *et al.* 2014. Variation in *Arabidopsis* flowering time associated with cis-regulatory variation in CONSTANS. *Nature Communications* **5,** 3651.

Rugnone ML, Faigan Soverna A, Sanchez SE, *et al*. 2013. LNK genes integrate light and clock signaling networks at the core of the Arabidopsis oscillator. *Proceedings of the National Academy of Sciences, USA* **110,** 12120–12125.

Salathia N, Davis SJ, Lynn JR, Michaels SD, Amasino RM, Millar AJ. 2006. FLOWERING LOCUS C-dependent and -independent regulation of the circadian clock by the autonomous and vernalization pathways. *BMC Plant Biology* **6,** 10.

Salazar JD, Saithong T, Brown PE, Foreman J, Locke JC, Halliday KJ, Carre IA, Rand DA, Millar AJ. 2009. Prediction of photoperiodic regulators from quantitative gene circuit models. *Cell* **139,** 1170–1179.

Samach A, Onouchi H, Gold SE, Ditta GS, Schwarz-Sommer Z, Yanofsky MF, Coupland G. 2000. Distinct roles of CONSTANS target genes in reproductive development of *Arabidopsis*. *Science* **288,** 1613–1636.

Sawa M, Kay SA. 2011. GIGANTEA directly activates flowering locus T in Arabidopsis thaliana. *Proceedings of the National Academy of Sciences, USA* **108**. 11698–11703.

Sawa M, Nusinow DA, Kay SA, Imaizumi T. 2007. FKF1 and GIGANTEA complex formation is required for day-length measurement in Arabidopsis. *Science* **318,** 261–265.

Schaffer R, Ramsay N, Samach A, Putterill J, Carre IA, Coupland G. 1998. The late elongated hypocotyl mutation of *Arabidopsis* disrupts circadian rhythms and the photoperiodic control of flowering. *Cell* **93,** 1219–1229.

Schmal C, Reimann P, Staiger D. 2013. A circadian clock-regulated toggle switch explains AtGRP7 and AtGRP8 oscillations in Arabidopsis thaliana. *PLoS Computational Biology* **9,** e1002986.

Schöning JC, Staiger D. 2005. At the pulse of time: protein interactions determine the pace of circadian clocks. *FEBS Letters* **579,** 3246–3252.

Schrader A, Uhrig J. 2013. MIDGET cooperates with COP1 and SPA1 to repress flowering in *Arabidopsis thaliana*. *Plant Signaling and Behavior* **8,** e25600.

Sgamma T, Jackson A, Muleo R, Thomas B, Massiah A. 2014. TEMPRANILLO is a regulator of juvenility in plants. *Science Reports* **4,** 3704.

Shrestha R, Gómez-Ariza J, Brambilla V, Fornara F. 2014. Molecular control of seasonal flowering in rice, arabidopsis and temperate cereals. *Annals of Botany* **114,** 1445–1458.

Song J, Irwin J, Dean C. 2013. Remembering the prolonged cold of winter. *Current Biology* **23,** R807–R811.

Song YH, Lee I, Lee SY, Imaizumi T, Hong JC. 2012a. CONSTANS and ASYMMETRIC LEAVES 1 complex is involved in the induction of FLOWERING LOCUS T in photoperiodic flowering in Arabidopsis. *The Plant Journal* **69,** 332–342.

Song YH, Smith RW, To BJ, Millar AJ, Imaizumi T. 2012b. FKF1 conveys timing information for CONSTANS stabilization in photoperiodic flowering. *Science* **336,** 1045–1049.

Srikanth A, Schmid M. 2011. Regulation of flowering time: all roads lead to Rome. *Cellular and Molecular Life Sciences* **68,** 2013–2037.

Staiger D, Allenbach L, Salathia N, Fiechter V, Davis SJ, Millar AJ, Chory J, Fankhauser C. 2003. The *Arabidopsis SRR1* gene mediates phyB signaling and is required for normal circadian clock function. *Genes and Development* **17,** 256–268.

Staiger D, Shin J, Johansson M, Davis SJ. 2013. The circadian clock goes genomic. *Genome Biology* **14,** 208.

Steinbach Y, Hennig L. 2014. Arabidopsis MSI1 functions in photoperiodic flowering time control. *Frontiers in Plant Science* **5,** 77.

Streitner C, Danisman S, Wehrle F, Schöning JC, Alfano JR, Staiger D. 2008. The small glycine-rich RNA-binding protein *At*GRP7 promotes floral transition in *Arabidopsis thaliana*. *The Plant Journal* **56,** 239–250.

Streitner C, Hennig L, Korneli C, Staiger D. 2010. Global transcript profiling of transgenic plants constitutively overexpressing the RNA-binding protein AtGRP7. *BMC Plant Biology* **10,** 221.

Suarez-Lopez P, Wheatley K, Robson F, Onouchi H, Valverde F, Coupland G. 2001. CONSTANS mediates between the circadian clock and the control of flowering in *Arabidopsis*. *Nature* **410,** 1116–1120.

Thines BC, Youn Y, Duarte MI, Harmon FG. 2014. The time of day effects of warm temperature on flowering time involve PIF4 and PIF5. *Journal of Experimental Botany* **65,** 1141–1151.

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022

Tiwari SB, Shen Y, Chang HC, *et al*. 2010. The flowering time regulator CONSTANS is recruited to the FLOWERING LOCUS T promoter via a unique cis-element. *New Phytologist* **187,** 57–66.

Turck F, Fornara F, Coupland G. 2008. Regulation and identity of florigen: flowering locus T moves center stage. *Annual Review of Plant Biology* **59,** 573–594.

Turner A, Beales J, Faure S, Dunford RP, Laurie DA. 2005. The pseudo-response regulator Ppd-H1 provides adaptation to photoperiod in barley. *Science* **310,** 1031–1034.

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

Wang C-Q, Guthrie C, Sarmast MK, Dehesh KI. 2014. BBX19 interacts with CONSTANS to repress FLOWERING LOCUS T transcription, defining a flowering time checkpoint in *Arabidopsis*. *The Plant Cell* **26,** 3589–3602.

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

Wang L, Fujiwara S, Somers DE. 2010. PRR5 regulates phosphorylation, nuclear import and subnuclear localization of TOC1 in the *Arabidopsis* circadian clock. *EMBO Journal* **29,** 1903–1915.

Wang L, Kim J, Somers DE. 2013. Transcriptional corepressor TOPLESS complexes with pseudoresponse regulator proteins and histone deacetylases to regulate circadian transcription. *Proceedings of the National Academy of Sciences, USA* **110,** 761–766.

Wang Y, Gu X, Yuan W, Schmitz Robert J, He Y. 2014. Photoperiodic control of the floral transition through a distinct polycomb repressive complex. *Developmental Cell* **28,** 727–736.

Wang Y, Wu JF, Nakamichi N, Sakakibara H, Nam HG, Wu SH. 2011. LIGHT-REGULATED WD1 and PSEUDO-RESPONSE REGULATOR9 form a positive feedback regulatory loop in the Arabidopsis circadian clock. *The Plant Cell* **23,** 486–498.

Wang ZY, Tobin EM. 1998. Constitutive expression of the CIRCADIAN CLOCK ASSOCIATED1 (CCA1) gene disrupts circadian rhythms and suppresses its own expression. *Cell* **93,** 1207–1217.

Weller JL, Liew LC, Hecht VFG, *et al*. 2012. A conserved molecular basis for photoperiod adaptation in two temperate legumes. *Proceedings of the National Academy of Sciences, USA* **109,** 21158–21163.

Weller JL, Reid JB, Taylor SA, Murfet IC. 1997. The genetic control of flowering in pea. *Trends in Plant Science* **2,** 412–417.

Wenkel S, Turck F, Singer K, Gissot L, Le Gourrierec J, Samach A, Coupland G. 2006. CONSTANS and the CCAAT box binding complex share a functionally important domain and interact to regulate flowering of *Arabidopsis*. *The Plant Cell* **18,** 2971–2984.

Xie Q, Wang P, Liu X, *et al* 2014. LNK1 and LNK2 are transcriptional coactivators in the *Arabidopsis* circadian oscillator. *The Plant Cell* **26,** 2843–2857.

Xue W, Xing Y, Weng X, *et al*. 2008. Natural variation in Ghd7 is an important regulator of heading date and yield potential in rice. *Nature Genetics* **40,** 761–767.

Yamaguchi A, Kobayashi Y, Goto K, Abe M, Araki T. 2005. TWIN SISTER OF FT (TSF) acts as a floral pathway integrator redundantly with FT. *Plant and Cell Physiology* **46,** 1175–1189.

Yang L, Xu M, Koo Y, He J, Poethig RS. 2013. Sugar promotes vegetative phase change in *Arabidopsis thaliana* by repressing the expression of MIR156A and MIR156C. *eLife* **2,** e00260.

Yang S, Weers B, Morishige D, Mullet J. 2014. CONSTANS is a photoperiod regulated activator of flowering in sorghum. *BMC Plant Biology* **14,** 148.

Young MW, Kay SA. 2001. Time zones: a comparative genetics of circadian clocks. *Nature Reviews Genetics* **2,** 702–715.

Yu S, Cao L, Zhou C-M, Zhang T-Q, Lian H, Sun Y, Wu J, Huang J, Wang G, Wang J-W. 2013. Sugar is an endogenous cue for juvenile-to-adult phase transition in plants. *eLife* **2,** e00269.

Zhao J, Huang X, Ouyang X, Chen W, Du A, Zhu L, Wang S, Deng XW, Li S. 2012. OsELF3-1, an ortholog of *Arabidopsis* EARLY FLOWERING, regulates rice circadian rhythm and photoperiodic flowering. *PLoS One* **7,** e43705.

Zuo Z, Liu H, Liu B, Liu X, Lin C. 2011. Blue light-dependent interaction of CRY2 with SPA1 regulates COP1 activity and floral initiation in Arabidopsis. *Current Biology* **21,** 841–847.

Downloaded from https://academic.oup.com/jxb/article/66/3/719/479209 by guest on 20 August 2022