The Science of Bloom: Exploring the Photoperiod Responsive Flowering Pathway: A review

ABSTRACT

Different plant species have evolved distinct needs for endogenous and external stimuli to trigger flowering. These disparate requirements were initially believed to be the result of various molecular pathways. Cultivars must precisely modify flowering times based on local photoperiod and environmental factors to guarantee that crops sustain excellent yields in a variety of latitudes. The molecular mechanisms by which phytochromes regulate photoperiodic blooming are not entirely known, despite the fact that they have been proven to be involved in photoperiodic flowering in numerous plants. Although the underlying molecular mechanisms differ between species, phytochromes play a critical role in controlling flowering in many plants. Surprisingly, a large number of photoperiod-affected processes use comparable gene networks to adapt to variations in the duration of light/dark cycles. In this review, we have concentrated on photoperiod-influenced developmental processes that have similar gene regulation networks.

Keywords: *Flowering, Phytochrome, Circadian rhythm, Regulation*

INTRODUCTION

Flowering is the transition between vegetative to reproductive stage. Phytochromes are the photo-receptors that perceives red (R) and far red (FR) light and converts inactive pr to active pfr hence, it regulates flowering. Plants adjust their flowering time to improve reproductive success by sensing seasonal changes in day length. In principle, photoperiodic flowering is regulated by photosensory receptors e.g., phytochromes and cryptochromes that sense light signals and transmit these signals to both the circadian clock to regulate protein turnover (Song *et al.,* 2015). The central regulator *CONSTANS* (*CO*) combines the output from the circadian clock and photoreceptors to induce the expression of genes that trigger flowering such *FLOWERING LOCUS T* (*FT*) (Song *et al.,* 2015). According to whether flowering occurs or is accelerated under long-day (LD) or short-day (SD) conditions, plants are classified as LD or SD plants, respectively (Garner and Allard, 1920). As red/far-red light receptors, phytochromes regulate photoperiodic flowering in both LD and SD plants. For example, in the LD plant arabidopsis (*Arabidopsis thaliana*), the red light receptor phytochrome B (phyB) has a major role in promoting the degradation of the CO protein, leading to reduced expression of *FT* and a delay in flowering. The far-red light receptor phyA, on the other hand, stabilizes *CO* and hence encourages flowering (Jung *et al.,* 2016). An important crop and a model plant for legumes, the flowering time of soybean seems to be primarily controlled by phyA rather than phyB, in contrast to the flowering of many other plants (Wang and Deng, 2003). In soybeans, *phyA2* and *phyA3* have been identified as the photoperiod receptors regulating flowering time, whereas phyBs have a minimal function. There are four phyA, two phyB and two phyE paralogs in case of soybean. The below mentioned information belongs to photoperiod responsive flowering pathway of model plants:

Arabidopsis

Forward genetics analyses in *A. thaliana* identified many flowering time mutants (Koornneef *et al.,* 1991). These mutants were classified into the maturity, photoperiod, autonomous, vernalization, light quality, and hormonal pathways that mediate endogenous and environmental cues that facilitated the floral transition (Quail, 1995 and Lazaro *et al.,* 2012). Together, these pathways control a group of genes referred to as "floral pathway integrators." These consist of *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*) and *FLOWERING LOCUS T* (*FT*). The expression of the floral meristem identity genes, including *APETALA1* (*AP1*), *AP2*, *FRUITFULL* (*FUL*), *CAULIFLOWER* (*CAL*), and *LEAFY* (*LFY*), is then regulated by these floral pathway integrators (Fig. 1). Our knowledge of the macromolecules that travel through the phloem in conjunction with sugars and hormones to control flowering and development advanced significantly with the finding that *FT* is florigen. (Kardailsky *et al.,* 1999 and Kobayashi *et al.,* 1999)

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The B-box transcription factor CONSTANS (CO), the first photoperiodic regulator, was cloned (Putterill *et al.,* 1995) and demonstrated to transfer photoperiod information to flowering time control by inducing *FT* in leaves. The circadian clock controls both transcription and posttranslational levels of *CO*. In long day (more than 12 hours of light [LD]) photoperiods, *CO* mRNA builds up during the day and peaks 16 hours after dawn. (Suarez-Lopez *et al.,* 2001). *CYCLIN DOF FACTORS* (*CDFs*) suppress *CO* mRNA levels in the morning and throughout the day, while *FLAVIN BINDING*, *KELCH REPEAT*, *F-BOX1* (*FKF1*), and *ZEITLUPE* (*ZTL*) alleviate *CDF*-mediated repression in the evening, allowing for mRNA accumulation. (Imaizumi *et al.,* 2005 and Song *et al.,* 2014). At night, *SUPPRESSOR OF PHYA-105S1* (*SPA1*) and *CONSTITUTIVE PHOTOMORPHOGENIC1* (*COP1*) target the CO protein for proteosomal breakdown (Valverde *et al.,* 2004). As a result of this intricate regulation, *CO* only builds up in LD conditions-when light and evening occur in order to attach to the FT promoter and trigger transcription. Flowering timing is also impacted by mutations in clock component genes including *GIGANTEA* (*GI*), *EARLY FLOWERING3* (*ELF3*), and pseudo response regulators (PRRs), in part because of the direct *CO* regulatory dependence on the circadian clock. (Hicks *et al.,* 1996). However, many clock components have direct transcriptional outputs affecting flowering through other pathways and themselves integrate temperature and light signals-mechanisms that are still being elucidated (Lin, 2000 and Mockler *et al.,* 2003).

A. thaliana induces the floral transition by activating *FT* expression in the companion cells (CCs) in the leaf, loading the FT protein into the sieve elements (SEs), and transporting it to the SAM, where it forms a floral activation complex with FD, FD-related proteins, and bZIP transcription factors (TFs). (Martignago *et al.,* 2023). By competing for chromatin-bound FD at common target loci, *TERMINAL FLOWER1* (*TFL1*), another FT-related phosphatidylethanolamine-binding (PEPB) protein, counteracts the action of *FT* (Goretti *et al.,* 2020). Numerous investigations have demonstrated that long-distance *FT* transfer is highly controlled and not solely the product of diffusion. *FT-INTERACTING PROTEIN 1* (*FTIP1*), *QUIRKY* (*QKY*), and *SYNTAXIN OF PLANTS121* (*SYP121*) facilitate the transfer of FT protein from CCs to SEs.

The transit of FT protein across a continuous ER network that passes through the plasmodesmata, or intercellular connections, between CCs and SEs is mediated by FTIP1, a protein found in the endoplasmic reticulum (ER) membrane (Liu *et al.,* 2012). Through the endosomal trafficking pathway, QKY and SYP121 (MCTP-SNARE Complex) work together to

enable FT export from CCs to SEs (Liu *et al.,* 2019). A protein called NaKR1 that contains a heavy metal-associated domain controls the long-distance trafficking of FT from leaves to the SAM when it enters the phloem stream (Zhu *et al.,* 2016). The process by which *FT* is emptied post-phloem and delivered to the shoot apex, however, is poorly understood (Yoo *et al.,* 2013).

Additionally, PHYTOCHROME-INTERACTING FACTOR 4 (PIF4) and its orthologs PIF5 and PIF7 enhance *FT* expression (Kumar *et al.,* 2012). Red light changes photoreceptor phyB into the active (Pfr) state at ideal ambient temperatures, which causes PIF4/5/7 and *CO* to degrade. In order to induce PIF4/CO and thereafter *FT,* the active state is quickly changed to the inactive (Pr) state at high temperatures (27 °C) (Kumar *et al.,* 2012).

By controlling *FT* transcription through *PHYTOCHROME AND FLOWERING TIME1* (*PFT1*), phyB can also control flowering time (Ishikawa *et al.,* 2005). *FT* movement from CC to SE is temperature sensitive, as is *FT* mRNA expression; low temperatures promote *FT* sequestration in the companion cell's cellular membrane because of its phospholipid-binding capabilities, which lowers soluble *FT* levels and postpones flowering (Jung *et al.,* 2020 and Liu *et al.,* 2020). Single mutations of *FTIP1*, *QKY*, and *SYP121* have varying temperature sensitivities, suggesting that ambient temperature may affect several *FT* trafficking stages. So, *FT* not only integrates environmental signals from different branches of the floral network but is itself directly regulated by these cues.

Reproductive competence in *A. thaliana* in accordance to flowering

It is necessary for the shoot apical meristem (SAM) to become competent prior to the floral transition (Nusinow *et al.,* 2011). Though not true for all species, this competency is believed to be connected to the change from juvenile to adult vegetative phase (Poethig, 2003). Certain species display different reactions in the juvenile and adult phases, while others flower without a transition from the juvenile to the adult vegetative phase (Lin *et al.,* 2021 and Andrade *et al.,* 2022). In many instances, only the adult plants can react to a variety of environmental cues, such as photoperiod or low temperature, to flower. (Hyun *et al.,* 2017).

A reduction in *microRNA156* (*miR156*) expression controls the transition from the juvenile to the adult phase by suppressing the expression of *SQUAMOSA PROMOTER BINDING-LIKE* (*SPL*) TFs (Wu and Poethig, 2006). The duration of juvenility is determined by this drop; a recent study found that the *miR156* decline rate is more closely associated with developmental age than with chronological age. The decrease in *miR156* is triggered by the start of cell division in the SAM during seed germination. The repressive histone mark trimethylation of lysine 27 of histone 3 (H3K27me3) is deposited concurrently with cell division, progressively decreasing the transcriptional activity of MIR156C (Cheng *et al.,* 2021). These findings offer a tenable explanation for the unidirectional reduction in *miR156*.

Through different methods, *miR156* controls flowering time in both leaves and SAM, according to genetic research and mis-expression tests. The *miR156/SPL* module mainly regulates blooming in leaves by targeting five AP2-like TFs with *miR172*. *SPL9* directly activates *MIR172B* (Hymowitz, 1970). By suppressing the expression of the florigen gene *FT*, which typically aids in signaling from leaves to the meristem, the five AP2-like TFs function as flowering repressors. Early flowering is caused by *miR172* overexpression, whereas late flowering, especially in non-inductive settings, is caused by the simultaneous mutation of five MIR172 genes (Cao *et al.,* 2017).

By activating a common set of targets, including as *AP1*, *FUL*, *LFY,* and *SOC1*, *miR156*-targeted *SPLs* (mostly SPL15) and *FT* directly induce blooming within the shoot apex itself. Furthermore, by activating MIR172A and MIR172D, *SPL15* relaxes the inhibition of flowering by *AP2* itself. These results indicate the interaction between meristem competence and photoperiod, as well as the highly redundant activities and feed-forward action of the *miR156/SPL* and *FT* modules in controlling blooming.

The floral repressors that lessen the meristem's receptivity to inductive cues are eliminated during vernalisation. *FRIGIDA* (*FRI*) upregulates the expression of *FLOWERING LOCUS C* (*FLC*), the primary repressor in Arabidopsis (Destro *et al.,* 2001). FLC, a MADS-box transcription factor, and *SHORT VEGETATIVE PHASE* (*SVP*), another MADS-box TF, produce a heterodimer that inhibits *FT* and *SOC1* to stop blooming. *COOLAIR* can transcribe through to the *FLC* promoter, begins just downstream of the main sense FLC poly (A) site and has many functions in *FLC* silencing (Wang *et al.,* 2021 and Du *et al.,* 2022). Every winter, *COOLAIR* homologs are induced in the semi-perennial relative *Arabis alpina*. Temperature influences the adoption of many secondary structures with distinct conformational dynamics by *COOLAIR* transcripts (Hawkes *et al.,* 2016). Cold-induced antisense transcripts are also seen in monocot *FLC* homologs (Jiao *et al.,* 2019).

In addition to raising *COOLAIR* RNA levels, cold temperatures also have an impact on how the RNA is processed, encouraging the usage of a proximal polyadenylation site and improving splicing to create the distal *COOLAIR* isoform known as Class II.ii (Zhao *et al.,* 2021 and Zhu *et al.,* 2021). NTL8, CRT/DRE-binding factors (CBFs), and the group-III WRKY transcription factor WRKY63 are among the cold-responsive TFs that aid in the cold induction of *COOLAIR* (Zhao *et al.,* 2020; Hung *et al.,* 2022 and Jeon *et al.,* 2023). These variables exhibit different cold sensitivity; for instance, NTL8 builds up during weeks of cold exposure (Zhao *et al.,* 2020), whereas CBFs are upregulated following brief cold exposure (minutes/hours) (Jeon *et al.,* 2023). Cold temperatures affect the processing of *COOLAIR* RNA, increasing its levels and promoting the use of a proximal polyadenylation site and enhancing splicing to produce the distal *COOLAIR* isoform, or Class II (Zhao *et al.,* 2021 and Zhu *et al.,* 2021). Cold-responsive transcription factors (TFs) that facilitate the cold induction of *COOLAIR* include NTL8, CRT/DRE-binding factors (CBFs), and the group-III WRKY transcription factor WRKY63 (Zhao *et al.,* 2020; Hung *et al.,* 2022; Jeon *et al.,* 2023). The cold sensitivity of these variables varies; for example, CBFs are elevated after short exposure to cold (minutes/hours) (Jeon *et al.,* 2023), whereas NTL8 accumulates during weeks of cold exposure (Zhao *et al.,* 2020).

Soybean

Lin *et al.,* 2022 used a Y2H screen to look for *phyA2/3*-interacting proteins in order to clarify the molecular mechanism by which *phyA2* and *phyA3* drive photoperiodic flowering in soybeans. They discovered that *LUX ARRHYTHMO* (*LUX*s) and *E1*s are two classes of proteins associated in photoperiodic flowering that interact with *phyA2/3*. Legumes-specific genes called *E1*s, which include *E1* and its homologs *E1la* and *E1lb*, prevent soybeans from blooming (Lin *et al.,* 2022). *LUX*s are important blooming enhancers that are essential parts of the evening complex. They may directly bind to the promoter region of *E1*s to suppress their expression (Bu *et al.,* 2021).

Lin *et al.,* used ChIP-qPCR study to better understand how *phyA2/ 3* work with *LUX*s and *E1*s to control soybean blooming time (Banerjee *et al.,* 2007). They found that phyA3 can bind with the *E1* promoter in a LUX-dependent way, which is consistent with lux1 lux2 being predominantly epistatic over *phyA2*, *phyA3* (Lin *et al.,* 2022). The authors hypothesised that the

release of *E1* suppression may be facilitated by phyA2/3-enhanced LUX breakdown (Lin *et al.,* 2022). As a result, the *phyA2 phyA3* double mutant exhibits significantly lower expression levels of *E1* and its homologs. Along with transcriptional regulation, the scientists discovered that *phyA2* and *phyA3* directly interact with and stabilise *E1*, which may contribute to the repression of the two FT homologs, *FT2a* and *FT5a*, that is dependent on *phyA2/3* and delays soybean flowering (Lin *et al.,* 2022). Overall, these findings imply that *phyA2/3* regulates *E1* in a *LUX*-dependent manner to control photoperiodic blooming in soybeans. Lin *et al.,* used a Y2H screen to look for *phyA2/3*-interacting proteins in order to clarify the molecular mechanism by which *phyA2* and *phyA3* mediate photoperiodic flowering in soybeans. They discovered that *LUX ARRHYTHMO* (*LUXs*) and *E1*s are two classes of proteins associated with photoperiodic flowering that interact with *phyA2/3* (Lin *et al.,* 2022). Legumes-specific genes called *E1*s, which include *E1* and its homologs E1la and E1lb, prevent soybeans from flowering (Lin *et al.,* 2022). *LUX*s are important blooming enhancers that are essential parts of the evening complex. They can directly bind to the promoter region of *E1*s to suppress their expression (Bu *et al.,* 2021). Lin *et al.,* used ChIP-qPCR analysis to better understand how *phyA2/3* works with *LUX*s and *E1*s to control soybean blooming time. They found that *phyA3* can connect with the *E1* promoter in a *LUX*-dependent way, which is consistent with *lux1 lux2* being predominantly epistatic over *phyA2 phyA3* (Lin *et al.,* 2022). The authors hypothesised that the release of *E1* suppression may be facilitated by *phyA2/3*-enhanced *LUX* breakdown (Lin *et al.,* 2022). As a result, the *phyA2 phyA3* double mutant exhibits significantly lower expression levels of *E1* and its homologs. Along with transcriptional regulation, the scientists discovered that *phyA2* and *phyA3* directly interact with and stabilise *E1,* which may contribute to the repression of the two *FT* homologs, *FT2a* and *FT5a*, that is dependent on *phyA2/3* and delays soybean flowering (Lin *et al.,* 2022). Overall, these findings imply that *phyA2/3* regulates *E1* in a LUX-dependent manner to control photoperiodic blooming in soybeans (Fig. 1).

Rice

FT expression stays low and the floral transition is delayed under short-day (SD) circumstances because the dark period is when circadian clock-dependent increase of *CO* expression takes place (Song *et al.,* 2015). A comparable molecular mechanism based on the *CO* and *FT* homologs *Heading date (Hd)-1* and *Hd3a* is found in the non-legume SD plant rice (Fig. 1). *Hd1* plays a dual role in rice, promoting blooming under SD conditions while inhibiting it under LD conditions, in contrast to arabidopsis, where *CO* alone increases the expression of *FT* (Song *et al.,* 2015). PhyB mediates this change in Hd1's function, turning it into a repressor in LD circumstances and preventing flowering. Furthermore, the rice-specific blooming pathway of Grain number, Plant Height, and *Heading date 7* (*Ghd7*)*–Early heading date 1* (*Ehd1*)*–Hd3a/RICE blooming LOCUS T 1* (*RFT1*) is regulated by *phyB*. *PhyB* stimulates *Ghd7* expression under LD circumstances, which suppresses *Ehd1* transcription and results in only low expression of *Hd3a* and *RFT1*, delaying flowering (Song *et al.,* 2015). Under SD circumstances, *Ehd1* repression is released by weak *Ghd7* expression, which activates *Hd3a* expression and encourages flowering. Concurrently, *Hd1* builds up at night and triggers the expression of *Hd3a* to cause the start of flowers.

Medicago

Unlike rice and arabidopsis, *phyA* is the primary regulator of photoperiodic blooming in legumes. Furthermore, *CO* homologs play a very small impact in photoperiodic flowering in legumes, but the legume-specific transcription factor *E1* and its homologs play a major role in regulating photoperiodic flowering by working downstream of *phyA*. Under LD conditions, *phyA* also stimulates the production of an *E1* homolog, *E1L*, in the LD legume plant medicago

(*Medicago truncatula*); however, unlike soybean, the *E1* homolog in medicago activates the expression of *FTa1* (Jaudal *et al.,* 2020). The beneficial effect of *phyA*s on E1s activity is diminished under SD circumstances, which encourages blooming in SD soybeans but prevents flowering in LD medicago (Fig. 1).

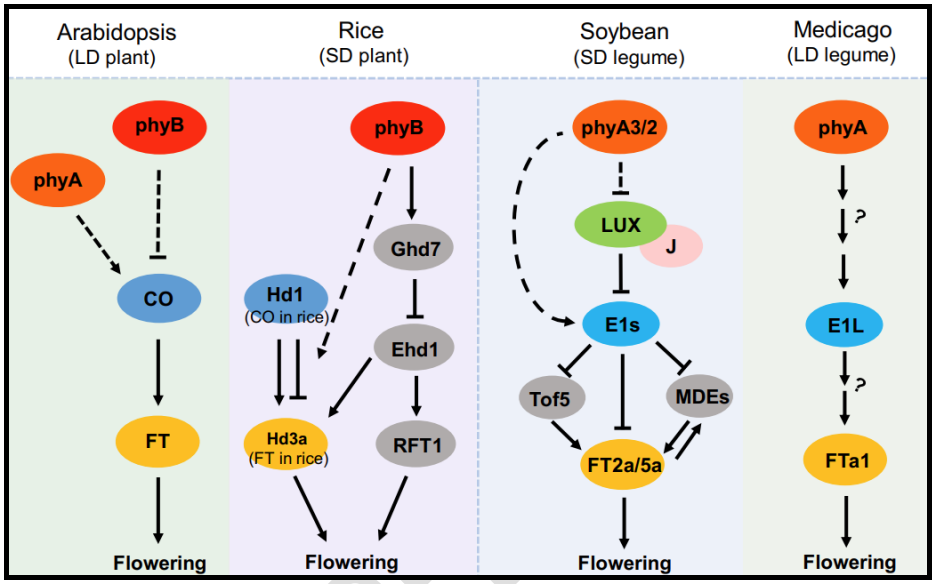

Figure 1. Dissecting the photoperiod responsive flowering pathway in model plants
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CONCLUSION

In contrast to the *phyB-CO FT* flowering route seen in many non-legume plants, including rice and arabidopsis, the recent study by Lin *et al.,* discovered a mechanism for the regulation of photoperiodic blooming in soybeans that acts through *phyA-LUXE1-FT*. Given that photoperiodic flowering in medicago (*Medicago truncatula*) and pea (*Pisum sativum*) has been shown to be primarily controlled by *phyA* (Weller and Ortega, 2015), the regulation of photoperiodic flowering by *phyA* does not appear to be soybean specific and occurs in other legumes as well.

The flowering regulator *E1*, which is present in most legumes, plays a crucial role in controlling blooming by controlling the expression of *FT*-like genes (Jaudal *et al.,* 2020). CO-like genes in medicago and pea, like those in soybean, do not seem to be involved in flowering time control. In contrast, the *CO*-like gene *COL2* may work in tandem with *E1* in common beans to suppress *FT* gene expression and postpone flowering under LD circumstances. This raises the intriguing question of why legumes have developed this particular regulatory route. First, the evolution of legumes has been significantly influenced by gene diversification and loss after two genome duplication events and major chromosomal rearrangements, which may have contributed to *phyA* acquiring new functions.

Furthermore, many legumes have the ability to host rhizobia in root nodules and form a symbiotic relationship to fix atmospheric nitrogen, which is a significant distinction from other blooming plants. The symbiotic nodulation process in soybeans has been demonstrated to be facilitated by the transmission of light signals from the shoot to the roots *via FTs* and TGACG-motif binding factors (STFs) (Ishikawa *et al.,* 2005). Because of this characteristic, legumes can need more intricate or specialised management to synchronise nodulation and flowering.

Additionally, *E1*, a flowering regulator specific to legumes, inhibits flowering in the SD plant soybean but encourages flowering in the LD plant medicago (Brambilla *et al.,* 2017). This is an intriguing parallel to *CO/Hd1* in rice and arabidopsis, which inhibit heading in the SD plant rice under LD conditions while promoting flowering in the LD plant arabidopsis. We must therefore concentrate on deepening our mechanistic understanding of flowering time gene function in natural environments and those processes that have evolved throughout adaptation in order to produce a variety of climate-proof crops on time. The quickest way to create fresh chances for crop enhancement will be this way.

REFERENCES

Andrade, L., Lu, Y., Cordeiro, A., Costa, J. M., Wigge, P. A., Saibo, N. J. and Jaeger, K. E. (2022). The evening complex integrates photoperiod signals to control flowering in rice. Proc. Natl. Acad. Sci., 119, e2122582119.

Banerjee, A. K., Chatterjee, M., Yu, Y., Suh, S.-G., Miller, W. A., and Hannapel, D. J. (2007). Dynamics of a mobile RNA of potato involved in a long-distance signaling pathway. Plant cell, 18, 3443–3457.

Brambilla, V., Gomez-Ariza, J., Cerise, M. and Fornara, F. (2017) The importance of being on time: regulatory networks controlling photoperiodic flowering in cereals. Front. Plant Sci., 8, 665.

Bu, T., Lu, S., Wang, K., Dong, L., Li, S., Xie, Q. and Kong, F. (2021). A critical role of the soybean evening complex in the control of photoperiod sensitivity and adaptation. Proc. Natl. Acad. Sci., 118, e2010241118.

Cao, D., Takeshima, R., Zhao, C., Liu, B., Jun, A. and Kong, F. (2017). Molecular mechanisms of flowering under long days and stem growth habit in soybean. J. Exp. Bot., 68, 1873–1884.

Cheng, Y. J., Shang, G. D., Xu, Z. G., Yu, S., Wu, L. Y., Zhai, D. *et al.,* (2021). Cell division in the shoot apical meristem is a trigger for *miR156* decline and vegetative phase transition in Arabidopsis. Proc. Natl. Acad. Sci., 118(46), e2115667118.

Destro, D., Carpentieri-Pípolo, V., Kiihl, R. D. S. and De Almeida, L. A. (2001). Photoperiodism and genetic control of the long juvenile period in soybean: A review. CBAB, 1, 72–92.

Du, H., Fang, C., Li, Y., Kong, F. and Liu, B. (2022) Understandings and future challenges in soybean functional genomics and molecular breeding. J. Integr. Plant Biol., 3, 202.

Garner, W. W. and Allard, H. A. (1920). Effect of the relative length of day and night and other factors of the environment on growth and reproduction in plants. J. Agric. Res., 18, 553–606

Goretti, D., Silvestre, M., Collani, S., Langenecker, T., Mendez, C. and Madueno, F. (2020). *TERMINAL FLOWER1* functions as a mobile transcriptional cofactor in the shoot apical meristem. Plant Physiol., 182(4), 2081-2095.

Hawkes, E. J., Hennelly, S. P., Novikova, I. V., Irwin, J. A., Dean, C. and Sanbonmatsu, K.Y. (2016). *COOLAIR* antisense RNAs form evolutionarily conserved elaborate secondary structures. Cell Rep., 16(12), 3087–3096.

Hicks, K. A., Millar, A. J., Carre, I. A., Somers, D. E., Straume, M., Meeks-Wagner, D. R. *et al.,* (1996). Conditional circadian dysfunction of the *Arabidopsis early- flowering 3* mutant. Science, 274(5288), 790-792.

Hung, F. Y., Shih, Y. H., Lin, P. Y., Feng, Y. R., Li, C. and Wu, K. (2022). WRKY63 transcriptional activation of *COOLAIR* and *COLDAIR* regulates vernalization- induced flowering. Plant Physiol., 190(1), 532-547.

Hymowitz, T. (1970). On the domestication of the soybean. Econ. Bot., 24, 408-421.

Hyun, Y., Richter, R. and Coupland, G. (2017). Competence to flower: age-controlled sensitivity to environmental cues. Plant Physiol., 173(1), 36-46.

Imaizumi, T., Schultz, T. F., Harmon, F. G., Ho, L. A. and Kay, S. A. (2005). FKF1 F-box protein mediates cyclic degradation of a repressor of *CONSTANS* in *Arabidopsis*. Science, 309(5732), 293–297.

Ishikawa, R., Tamaki, S., Yokoi, S., Inagaki, N., Shinomura, T., Takano, M. and Shimamoto, K. (2005). Suppression of the floral activator *Hd3a* is the principal cause of the night break effect in rice. Plant cell, 17, 3326-3336.

Jaudal, M., Wen, J., Mysore, K. S., and Putterill, J. (2020) Medicago PHYA promotes flowering, primary stem elongation and expression of flowering time genes in long days. BMC Plant Biol., 20, 329.

Jeon, M., Jeong, G., Yang, Y., Luo, X., Jeong, D. and Kyung, J. *et al.,* (2023). Vernalization-triggered expression of the antisense transcript *COOLAIR* is mediated by *CBF* genes. eLife, 12.

Jiao, F., Pahwa, K., Manning, M., Dochy, N. and Geuten, K. (2019). Cold induced antisense transcription of *FLOWERING LOCUS C* in distant grasses. Front Plant Sci., 10.

Jung, J. H., Barbosa, A. D., Hutin, S., Kumita, J. R., Gao, M., Derwort, D. and Wigge, P. A. (2020). A prion-like domain in *ELF3* functions as a thermosensor in Arabidopsis. Nature, 585, 256-260.

Jung, J. H., Domijan, M., Klose, C., Biswas, S., Ezer, D., Gao, M. and Wigge, P. A. (2016). Phytochromes function as thermosensors in Arabidopsis. Science, 354, 886-889.

Kardailsky, I., Shukla, V. K., Ahn, J. H., Dagenais, N., Christensen, S.K., Nguyen, J.T. *et al.,* (1999). Activation tagging of the floral inducer FT. Science, 286 (5446), 1962-1965.

Kobayashi, Y., Kaya, H., Goto, K., Iwabuchi, M. and Araki T (1999). A pair of related genes with antagonistic roles in mediating flowering signals. Science, 286(5446), 1960-1962.

Koornneef, M., Hanhart, C. J. and van der Veen, J. H. (1991). A genetic and physiological analysis of late flowering mutants in *Arabidopsis thaliana*. Mol. Gen. Genet., 229 (1), 57-66.

Kumar, S. V., Lucyshyn, D., Jaeger, K. E., Alos, E., Alvey, E. and Harberd, N. P. (2012). Transcription factor PIF4 controls the thermosensory activation of flowering. Nature, 484(7393), 242-245.

Lazaro, A., Valverde, F., Piñeiro, M. and Jarillo, J. A. (2012). The Arabidopsis E3 ubiquitin ligase HOS1 negatively regulates *CONSTANS* abundance in the photoperiodic control of flowering. Plant cell, 24, 982-999.

Lin, C. (2000). Photoreceptors and regulation of flowering time. Plant Physiol., 123, 39-50.

Lin, X., Dong, L., Tang, Y., Li, H., Cheng, Q., Li, H. and Kong, F. (2022) Novel and multifaceted regulations of photoperiodic flowering by phytochrome A in soybean. Proc. Natl. Acad. Sci., 119, e2208708119.

Lin, X., Fang, C., Liu, B. and Kong, F. (2021). Natural variation and artificial selection of photoperiodic flowering genes and their applications in crop adaptation. Biotech, 2, 156–169.

Liu, L., Li, C. Y., Teo, W. N., Zhang, B. and Yu, H. (2019). The *MCTP-SNARE* complex regulates florigen transport in *Arabidopsis*. Plant Cell, 31(10): 2475-2490.

Liu, L., Liu, C., Hou, X. L., Xi, W. Y., Shen, L. S. and Tao, Z. (2012). *FTIP1* is an essential regulator required for florigen transport. Plos Biol., 10(4):e1001313.

Martignago, D., Silveira Falavigna, V., Lombardi, A., Gao, H., Korwin Kurkowski, P. K., Galbiati, M., *et al.,* (2023). The bZIP transcription factor AREB3 mediates *FT* signalling and floral transition at the Arabidopsis shoot apical meristem. PLoS Genet., 19(5), e1010766.

Mockler, T., Yang, H., Yu, X., Parikh, D., Cheng, Y. C., Dolan, S. and Lin, C. (2003). Regulation of photoperiodic flowering by Arabidopsis photoreceptors. Proc. Natl. Acad. Sci., 100, 2140–2145.

Nusinow, D. A., Helfer, A., Hamilton, E. E., King, J. J., Imaizumi, T. and Kay, S. A. (2011). The ELF4-ELF3-LUX complex links the circadian clock to diurnal control of hypocotyl growth. Nature, 475, 398–402.

Poethig, R. S. (2003). Phase change and the regulation of developmental timing in plants. Science, 301(5631), 334–336.

Putterill. J., Robson, F., Lee, K., Simon, R. and Coupland, G. (1995). The CONSTANS gene of Arabidopsis promotes flowering and encodes a protein showing similarities to zinc finger transcription factors. Cell, 80(6), 847–857.

Quail, P. H. (1995). Phytochromes: Photosensory perception and signal transduction. Science, 268, 675–680.

Song, Y. H., Estrada, D. A., Johnson, R. S., Kim, S. K., Lee, S. Y., MacCoss, M. J. *et al.,* (2014). Distinct roles of FKF1, Gigantea, and Zeitlupe proteins in the regulation of *CONSTANS* stability in *Arabidopsis* photoperiodic flowering. Proc. Natl. Acad. Sci., 111(49), 17672–17677.

Song, Y. H., Shim, J. S., Kinmonth-Schultz, H. A. and Imaizumi, T. (2015). Photoperiodic flowering: time measurement mechanisms in leaves. Annu. Rev. Plant Biol., 66, 441–464.

Suarez-Lopez, P., Wheatley, K., Robson, F., Onouchi, H., Valverde, F. and Coupland G (2001). *CONSTANS* mediates between the circadian clock and the control of flowering in *Arabidopsis*. Nature, 410(6832), 1116-1120.

Valverde, F., Mouradov, A., Soppe, W., Ravenscroft, D., Samach, A. and Coupland, G. (2004). Photoreceptor regulation of *CONSTANS* protein in photoperiodic flowering. Science, 303(5660), 1003–1006.

Wang, H. and Deng, X. W. (2003). Dissecting the phytochrome A-dependent signaling network in higher plants. Trends Plant Sci., 8, 172-178.

Wang, T., Guo, J., Peng, Y., Lyu, X., Liu, B., Sun, S. and Wang, X. (2021) Light-induced mobile factors from shoots regulate rhizobium-triggered soybean root nodulation. Science, 374, 65-71.

Weller, J. L. and Ortega, R. (2015) Genetic control of flowering time in legumes. Front. Plant Sci., 6, 207.

Wu, G. and Poethig, R. S. (2006). Temporal regulation of shoot development in *Arabidopsis* thaliana by *miR156* and its target *SPL3*. Development, 133(18), 3539–3547.

Yoo, S. C., Chen, C., Rojas, M., Daimon, Y., Ham, B. K. and Araki, T. (2013). Phloem long-distance delivery of FLOWERING LOCUS T (FT) to the apex. Plant J., 75(3), 456–468.

Yuan, J., Ott, T. and Hiltbrunner, A. (2023). Phytochromes and flowering: legumes do it another way. Trends in Plant Science, 28(4), 379-381.

Zhao, Y., Antoniou-Kourounioti, R. L., Calder, G., Dean, C. and Howard, M. (2020). Temperature-dependent growth contributes to long-term cold sensing. Nature, 583(7818), 825-829.

Zhao, Y., Zhu, P., Hepworth, J., Bloomer, R., Antoniou-Kourounioti, R. L., and Doughty J, *et al.,* (2021). Natural temperature fluctuations promote *COOLAIR* regulation of *FLC*. Genes Dev., 35(11-12), 888–898.

Zhu, P., Lister, C. and Dean, C. (2021). Cold-induced Arabidopsis FRIGIDA nuclear condensates for *FLC* repression. Nature, 599(7886), 657–661.

Zhu, Y., Liu, L., Shen, L. S. and Yu, H. (2016). NaKR1 regulates long-distance movement of FLOWERING LOCUS T in *Arabidopsis*. Nat. Plants., 2(6), 16075.