Review Article

How Plants Adapt to the Photoperiod

ABSTRACT

Plants are extremely sensitive to changes in their environment, particularly variations in photoperiod or day length. Photoperiodism refers to a plant's capacity to detect variations in day length and make use of this knowledge to control key developmental processes including flowering, growth, and dormancy. Through a process known as photoperiodism, plants can detect and react to variations in the number of daylight hours, or photoperiod. The physiological response of plants to the length of day or night is known as photoperiodism. The plant uses this physiological response to time-crucial developmental events like flowering. In this essay, I will cover the current understanding of how plants respond to photoperiod and the molecular mechanisms underpinning this response.

Three groups of plants' photoperiodic responses can be distinguished: short-day plants (SDPs), long-day plants (LDPs), and day-neutral plants (DNPs). Whereas LDPs bloom when the length of the day exceeds the crucial threshold, SDPs do so only when it is shorter than the critical threshold. Conversely, DNPs do not have a crucial day duration and can bloom in any day length. Many genes and biochemical processes control how a plant responds to the photoperiod. The creation and movement of the hormone florigen, which starts blooming in response to photoperiodic signals, is a crucial regulating mechanism. On the other hand, a class of photoreceptors known as phytochromes are involved in the biochemical mechanisms driving photoperiodic responses in plants. The perception of light's duration, quality, and amount is caused by phytochromes. The red-light-absorbing Pr form and the far-red-light-absorbing Pfr form are the two interconvertible states in which they can exist. The ratio of Pr to Pfr is altered by the duration of light exposure and is utilised by plants to assess day length.

Exposure to light in SDPs causes the expression of the CONSTANS (CO) gene, and the CO protein causes the expression of the FLOWERING LOCUS T (FT), a gene that encourages flowering. By exposing LDPs to light, a different gene called GI (GIGANTEA) is induced rather than CO, which is normally expressed. The FT gene's expression is encouraged by GI's interaction with the protein ZEITLUPE (ZTL), which also encourages flowering. Several proteins and signalling pathways are also involved in photoperiodic responses in plants in addition to these essential elements. For instance, to optimise the response to variations in day length, the photoperiodic pathway interacts with the circadian clock, which controls numerous physiological processes in plants. In some species, the hormone gibberellin (GA) also aids in the promotion of flowering.

One essential adaptation that enables plants to synchronise their developmental processes with seasonal changes is their capacity to react to variations in day length. Phytochromes play a key part in how plants perceive the day in the complex network of proteins and signalling channels that make up the molecular mechanisms behind photoperiodic responses in plants. There is still much to learn about the diversity and complexity of the photoperiodic response across several plant groupings, even if much is known about it in particular species.

Keywords: Flowering, Circadian rhythms, Cryptochromes, Dormancy, Photoperiod, Phytochrome;

1. INTRODUCTION

Many developmental reactions in animals, plants, and even fungi are regulated by photoperiod. Many life activities in eukaryotes, such as plants, are adapted to the regular rhythms of light and darkness. Due to the Earth's rotation around its axis, light and dark periods alternate in a daily cycle of around 24 hours. The photoperiod, which changes with the season and latitude, is the length of the light

period during this 24 h day-night cycle (Jackson, 2009). Daylength is a dependable predictor of the time of year, allowing developmental events to be planned to correspond with specific environmental conditions. As a result, the response to photoperiod has evolved. For optimum development and reproduction, plants coordinate their physiological decisions with the right time of the year (Casal *et. al.,* 2004). Understanding the molecular processes behind plants' response to photoperiods has come a long way. These mechanisms include detecting the light signal in the leaves, synchronising circadian rhythms, sensing and responding to the photoperiod, which are crucial plant functions for adjusting to their environment, and producing a mobile signal that is transported throughout the entire plant. Only a few of the many distinct reactions in plants that are regulated by photoperiod include flowering, tuberization, and bud formation. Comparing what is known about the molecular mechanisms regulating these responses reveals that, while there are some shared elements among them, the regulatory systems have developed significantly differently for each response. The timing of blooming (Carré, 2001; Song *et. al.,* 2015), tuberization (Sarkar, 2010), bud set, and dormancy are some of the most notable plant responses affected by the photoperiod (Jackson, 2009; Singh *et. al.,* 2017). Season-dependent photoperiods affect the growth stoppage of perennial plants like trees and regulate senescence in annual plants (Serrano-Bueno *et. al.,* 2021). (Singh *et. al.,* 2017). The photoperiod is the primary environmental factor governing the beginning and conclusion of the seasonal growing season in temperate temperature zones as well as in tropical areas (Adole *et. al.,* 2019). Among a few examples of photoperiod-regulated developmental processes in plants, Hendel-Rahmanim *et. al.,* 2007, cited the regulation of scent emission from flowers as another one. Plants can be divided into three categories short-day, long-day, and day-neutral plants based on how they respond to the photoperiod in terms of flowering. This categorization is based on the critical day length (CDL), which defines how well plants can react to changes in photoperiod. Plants grown in short days flower when the photoperiod is less than the CDL, whereas plants grown in long days only bloom when the photoperiod exceeds the CDL. Plants that are day-neutral don't react to the photoperiod (Jackson, 2009). In addition to the CDL, the stage of development of the plant impacts its capacity to recognise and then react to photoperiods. At their juvenile stage, *Arabidopsis* plants' flowering response is not affected by photoperiods. *Arabidopsis* becomes sensitive to photoperiods when it enters the adult phase, allowing it to respond to floral inducers (Matsoukas, 2014). For plants to reproduce and survive, the photoperiod sensing and intrinsic developmental programmes, or developmental phases, must be coordinated with the seasonal photoperiod.

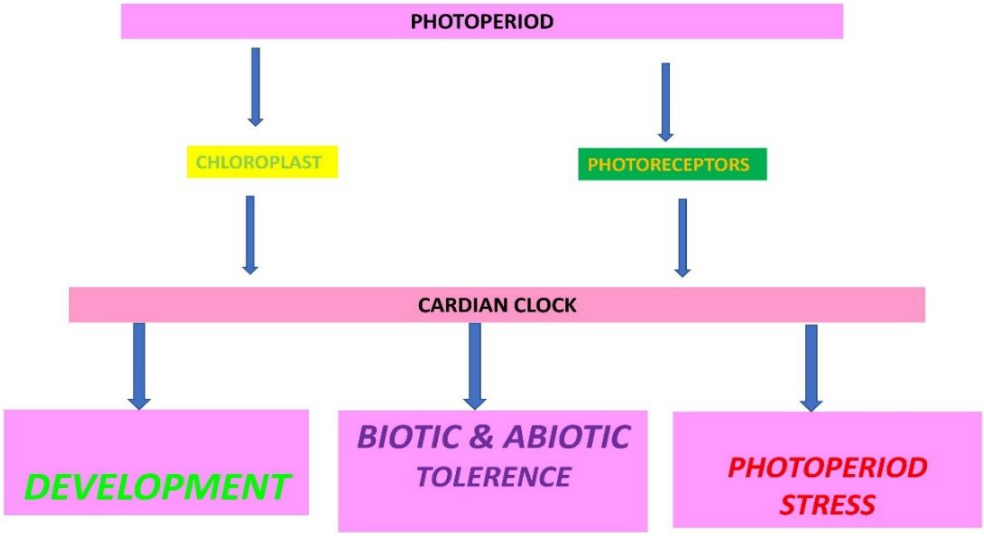
In addition to allowing plants to coordinate their growth cycles with a particular season, photoperiod sensing also lessens the effects of environmental challenges that occur at the same time every year. Interest in how the photoperiod affects how organisms react to biotic and abiotic challenges has increased recently. For instance, it has been discovered that the lengthening of days helps people acclimatise to the cold and get ready for icy winter temperatures (Ouellet and Charron, 2013). Also, it has been demonstrated that the photoperiod affects the plants' ability to withstand salt stress and drought stress (Han *et. al.,* 2013a) (Kim *et. al.,* 2013; Park *et. al.,* 2016). Additionally, mounting data indicates that plant-pathogen interactions are influenced by the length of the light period (Griebel and Zeier, 2008). Plants can thus enhance their responses to a variety of environmental challenges thanks to photoperiod sensing. Yet, abrupt modifications to the photoperiod can also cause stress. Experiments showed that changes in the photoperiod cause stress reactions in *Arabidopsis* plants that are similar to responses to pathogen attack, even though the molecular mechanisms underlying this new abiotic stress form have not yet been fully elucidated (Nitschke *et. al.,* 2016, 2017; Abuelsoud *et. al.,* 2020; Frank *et. al.,* 2020). (Cortleven *et. al.,* 2021). Systemic acquired resistance (SAR) development in plants serves as a crucial line of protection against upcoming pathogen invasions (Conrath *et. al.,* 2006). While photoperiod stress causes similar effects, this may open up new possibilities for the sustainable use of changed photoperiods to reduce pathogen infections and, as a result, reduce production losses in horticulture.

2. MOLECULAR MECHANISMS INVOLVED IN LIGHT PERCEPTION AND THE PHOTOPERIOD

Plants need a sensing mechanism that detects light (through photoreceptors or chloroplasts) and measures time (by the circadian clock) in order to perceive and respond to photoperiods (Jackson, 2009; Serrano-Bueno *et. al.,* 2021). Plants get detailed information about their surrounding light environment through their photoreceptors and chloroplasts, including the quality (spectral composition, direction), quantity, intensity, and duration of incoming irradiation. Five distinct

photoreceptor families in *Arabidopsis thaliana* sense light from various solar light spectrum wavelengths. Phytochromes (phyA to phyE) sense red and far-red light. Cryptochromes (CRY1, CRY2, CRY3), phototropins (PHOT1, PHOT2), and the F-box-containing flavin-binding proteins ZTL and FLAVIN-BINDING KELCH REPEAT F-BOX1 (FKF1)/LOV KELCH PROTEIN2 (LKP2) are all capable of detecting blue light. The UVR8 photoreceptor detects UV radiation (for a review, see Sanchez *et. al.,* 2020; Roeber *et. al.,* 2021). The light entrainment of the circadian clock involves each of the aforementioned photoreceptor groups.

In addition to photoreceptors, chloroplasts serve as a plant's light sensor and adjust their ultrastructure in response to various photoperiods (Lepisto and Rintamaki, 2012). Plants with extended days have smaller grana stacks and more chlorophyll in their chloroplasts. According to Walters and Horton (1995), these qualities match the structural and photosynthetic traits typical of sun plants. Plants' adaptation to light is based on redox signals generated by the chloroplasts (Pfannschmidt *et. al.,* 2009). It is unclear which signalling pathways contribute to the photoperiodic-dependent growth of chloroplasts. Only a few examples of potential pathways involved, all working independently of the photoreceptors, including the redox status of the photosynthetic electron transport chain, ROS metabolism, and chloroplast-to-nucleus retrograde signalling. (Lepisto and Rintamaki, 2012; Feng *et. al.,* 2016). In addition to controlling the chloroplast ultrastructure, the photoperiod also controls how much carbohydrate (C) is stored in chloroplasts and how photosynthate is partitioned to starch (Zeeman *et. al.,* 2007). A greater fraction of the fixed C is allocated to starch when there is less accessible C, such as during short photoperiods (Smith and Stitt, 2007).



.

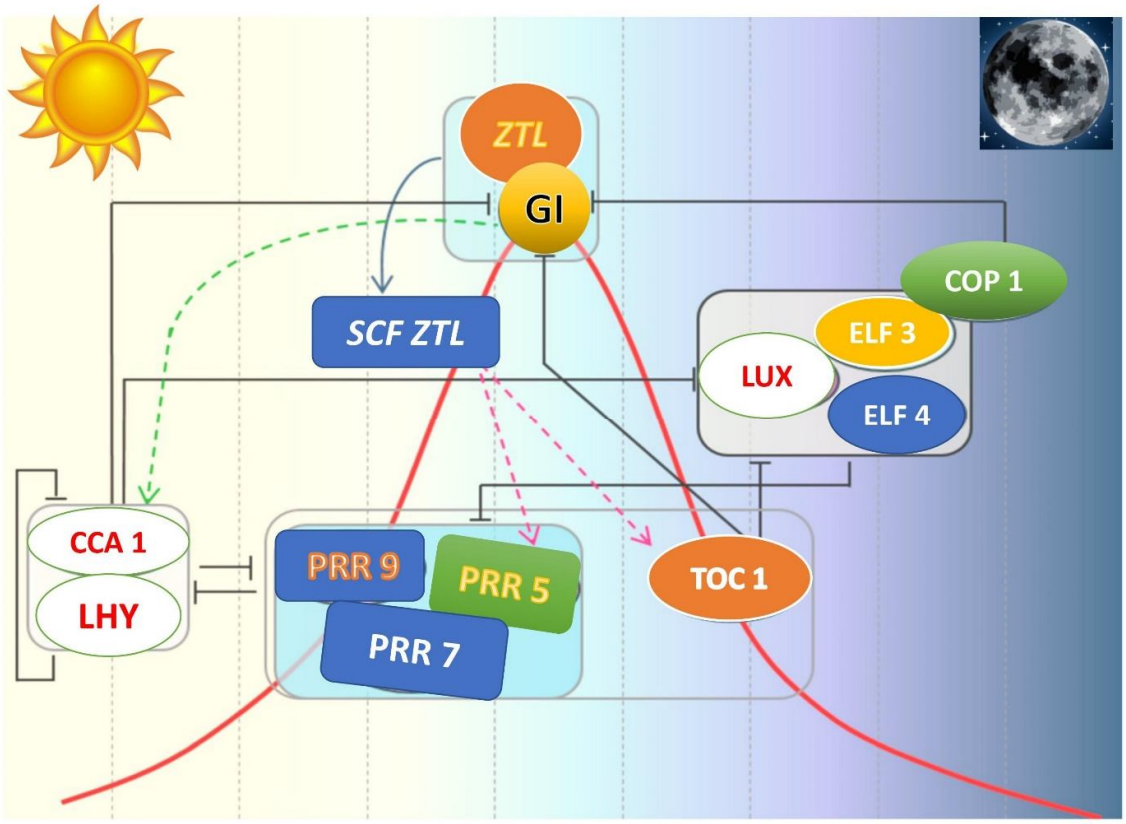
Fig .1 Molecular mechanisms involved in light perception and the photoperiod

Compared to plants cultivated in long days, the near-linear starch breakdown is slowed down during the night. By limiting C-starvation or C-excess at the end of the night period, this leads to a nearly but not entirely depleted starch content at dawn (Stitt and Zeeman, 2012; Moraes *et. al.,* 2019). Across several photoperiods, this C-mobilization pattern is consistent (Stitt and Zeeman, 2012; Moraes *et. al.,* 2019). The precise molecular mechanisms governing the synthesis of starch under different photoperiods are also unknown, but other factors such as transcriptional control of chloroplast enzymes, redox regulation, circadian regulation, and feedback inhibition from the carbohydrate metabolism may be involved. Plants may keep track of time using an endogenous clock called the circadian clock (Hsu and Harmer, 2014). In order to alter the internal rhythm, the clock is set daily entrainment, particularly by light and temperature (McClung, 2006). The circadian clock in *Arabidopsis thaliana* is made up of a number of interlocked transcription-translation feedback loops (Hsu and

Harmer, 2014). In the morning (Schaffer *et. al.,* 1998; Wang and Tobin, 1998), the MYB-domain transcription factor genes CIRCADIAN CLOCK ASSOCIATED1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY) are expressed, and these genes suppress the expression of TIMING OF CAB EXPRESSION1 (TOC1) during the day (Alabadi *et. al.,* 2001). The transcription of CCA1 and LHY is then repressed by TOC1 (Gendron *et. al.,* 2012). The Evening Complex (EC), which is made up of the proteins EARLY FLOWERING3 (ELF3), ELF4, and LUX ARRHYTHMO (LUX), suppresses TOC1 transcription late at night. The transcription of LHY and CCA1 can start up again the next morning because to this down-regulation. Throughout the day, the expression of CCA1 and LHY is suppressed by successive waves of PSEUDO-RESPONSE REGULATOR9 (PRR9), PRR7, and PRR5 expression (Nakamichi *et. al.,* 2012). The clock function is also influenced by other rhythmically expressed transcriptional activators, including REVEILLE4 (RVE4), RVE6, and RVE8, the proteins LIGHT-REGULATED WD1 (LWD1) and LWD2, and the transcription factors NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED GENE1 (LNK1) and LNK2 (Rawat *et. al.,* 2011; Rugnone *et. al.,* 2013). The circadian clock helps plants respond to different environmental challenges, but abiotic factors also have an impact on how well the clock works. Reviews by Sanchez *et. al.,* (2011), Kiebowicz-Matuk and Czarnecka (2014), Grundy *et. al.,* (2015), Seo and Mas (2015), and Sharma *et. al.,* (2021) include more details on this. Recently, a novel webtool was introduced to study the transcriptional networks influenced by light and the circadian clock (de los Reyes *et. al.,* 2020). It is possible to determine the target genes of circadian regulators using ATTRACTOR (*Arabidopsis thaliana* TRanscriptionAl Circadian neTwORk1). This could help us comprehend how the circadian clock interacts with how plants react to environmental stressors.

.

Fig.2 Plants react to environmental stressors



GIGANTEA (GI) is one of the circadian clock-controlled genes that is essential to the photoperiod sensing system (Fowler *et. al.,* 1999). It produces a sizable chaperone-active protein that is encoded by a single big gene (Cha *et. al.,* 2017). The interaction with GI enhances the stability of the F-box protein ZEITLUPE (ZTL) during the perception of blue light. According to Mas *et. al.,* 2003 and Kiba *et. al.,* 2007, ZTL is an evening-phased E3 ubiquitin ligase that targets the clock elements TOC1 and

PRR5 for proteasomal degradation. The evening is when GI protein quantity rises, keeping ZTL abundance high. As a result, TOC1 and PRR5 oscillations of high amplitude are maintained (Kim *et. al.,* 2007). As a result, clock output genes like CONSTANS (CO), which encodes a key protein in photoperiod-dependent blooming, are correctly set in phase (Suarez-Lopez *et. al.,* 2001; Shim *et. al.,* 2017). This furthers the clock's entrainment. In response to blue light perception, GI also interacts with FKF1, degrading CYCLING DOF FACTOR1 (CDF1), a transcriptional repressor of CO (Sawa *et. al.,* 2007). For photoperiodic reactions like flowering, the proper timing of protein stabilisation during long days must be coordinated with the circadian-regulated expression of FKF1, GI, and CDF1. The CO-FT-GI-CDF hub is conserved among flowering plants that are not closely related to each other, which is interesting to note (Serrano-Bueno *et. al.,* 2021). GI controls the maturation of miR172, which targets the genes APETALA2 (AP2), TARGET OF EAT1 (TOE1), TOE2, TOE3, SCHLAFMÜTZE (SMZ), and SCHNARCHZAPFEN (SNZ), as well as other AP2-like genes. Depending on the age of the plants, these floral repressors are post-transcriptionally downregulated by miR172, which controls blooming time and floral growth in the shoot apical meristem (Mathieu *et. al.,* 2009) (Aukerman and Sakai, (2003). Together with EARLY FLOWERING3 (ELF3), GI also regulates photoperiod sensing that is mediated by the circadian clock. The breakdown of the photoperiod sensing mechanism occurs when the circadian clock is unable to appropriately respond to light signals in their absence (Anwer *et. al.,* 2020).

In addition to being a key component of the photoperiod sensing mechanism, GI also plays a crucial role in modulating the effects of the photoperiod in response to a variety of stresses, such as cold stress oxidative, osmotic, and drought, (Cao *et. al.,* (2005); Fornara *et. al.,* 2015).

1. PHOTOPERIOD IMPACTS ABIOTIC AND BIOTIC STRESS REACTIONS

a. Photoperiod and Tolerance to Freezing

Freezing tolerance is one of the most well-known photoperiod-dependent stress tolerances. Plants feel the lengthening of the days in the fall, anticipating the impact of winter's colder temperatures, which increases their freezing tolerance (Lee and Thomashow, 2012). As an illustration, the red-osier dogwood (*Cornus sericea*) adapts to a shorter photoperiod by reducing the water content of the stem, which increases its freezing resistance (Karlson et al.,2003). In hybrid aspen, the major switch converting metabolism from vegetative growth to dormancy and establishing freezing tolerance is phyA-mediated apical bud development under short days (Welling *et. al.,* 2002).

Arabidopsis thaliana similarly exhibits increased cold tolerance brought on by a shortening of the photoperiod. According to Alonso-Blanco *et. al.,* 2005, the photoperiod conditions that different *Arabidopsis* accessions are geographically exposed to can affect their freezing resistance. This variation in response to day duration is mediated by the C-repeat/dehydration-responsive element-binding factor (CBF/DREB) signalling cascade, which is a key molecular mechanism. The induction of COLD-REGULATED (COR) genes by the stimulation of the CBF genes results in the development of cold tolerance (Thomashow, 2010; Pareek *et. al.,* 2017). Less freezing tolerance results from the phyB, PHYTOCHROME INTERACTING FACTOR4 (PIF4), and PIF7 repression of the CBF regulon over lengthy days. Autumnal day lengthening alleviates this suppression, increasing the expression of the CBF genes and enabling plants to adapt to impending cooler temperatures (Lee and Thomashow, 2012).

The GI-CDF module controls freezing tolerance in *Arabidopsis*, one of the elements involved in photoperiodic blooming (Fornara *et. al.,* 2015). According to Fowler and Thomashow (2002) and Cao *et. al.,* (2005), cold induces GI expression. In *Arabidopsis*, several cold-regulated genes are also co-regulated by GI and CDFs. mRNA for COR genes was found in greater amounts in gi-100 mutants than in wild type, which was consistent with increased expression of CDF1, CDF2, CDF3, and CDF5, as well as increased freezing and oxidative stress tolerance.

As a result, in gi-100 cdf1235 mutants, this increase in COR gene expression was reduced (Fornara *et. al.,* 2015). Cao *et. al.,* (2005) discovered that gi-3 mutants are hypersensitive to cold, in contrast. Given that there was no difference in the transcript levels of CBF genes under cold stress, it was

determined that GI promotes freezing tolerance without affecting CBF by changing carbohydrate metabolism. (Cao *et. al.,* 2005, 2006) The precise mechanisms are still not fully understood. These variations may result from the use of GI mutant alleles in various ecotypes and/or test setups (Fornara *et. al.,* 2015). The improved freezing tolerance of GI loss-of-function mutant *Brassica rapa* plants, however, suggests that the function of GI in resistance to freezing stress is conserved across species (Xie *et. al.,* 2015).

HOS1 (HIGH EXPRESSION OF OSMOTICALLY RESPONSIVE GENE1) is another element that has a role in controlling both photoperiod flowering and freezing tolerance. In order to ensure that the CO-dependent activation of FT only takes place when the light period reaches a specific duration, HOS1 encodes an E3 ubiquitin ligase with a RING finger that regulates the quantity of CO (Lazaro *et. al.,* 2012). By facilitating the ubiquitination and proteasomal degradation of ICE1 (INDUCER OF CBF EXPRESSION1), HOS1 inhibits the body's ability to adapt to cold temperatures (Ishitani *et. al.,* 1998; Dong *et. al.,* (2006); Lee and Thomashow, 2012).

.

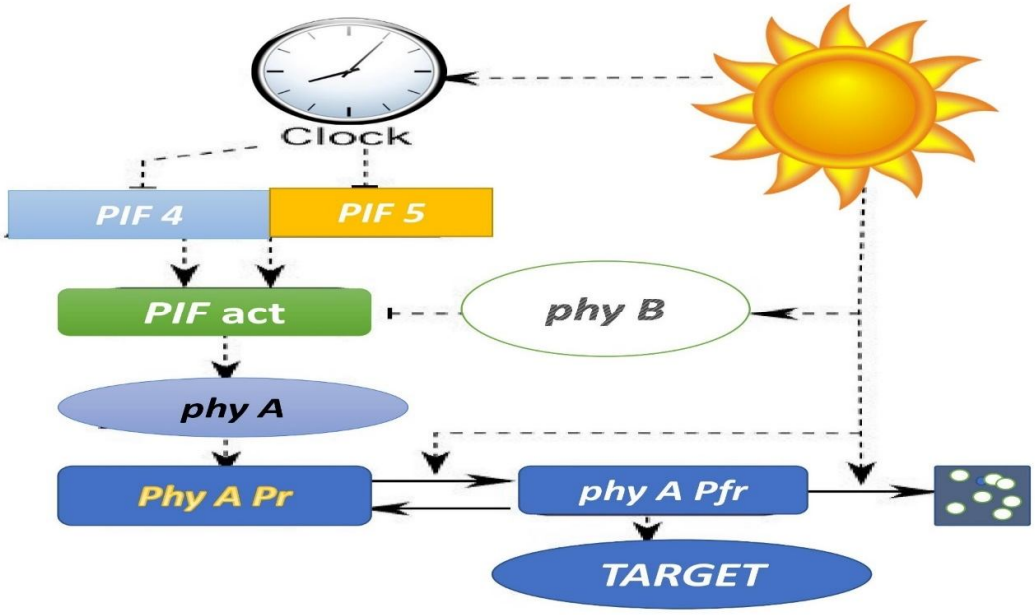


Fig.3 Photoperiod and Tolerance to Freezing

b. Drought and Tension from Photoperiod

Plants are negatively impacted by drought, which reduces their productivity. The endogenous abscisic acid (ABA) level rises in response to drought signals, causing the stomata to close to reduce water loss through transpiration (Outlaw, 2003).

Plants use the drought escape as an adaptive mechanism to hasten the reproductive development (*i.e.,* flowering) when under the stress of drought. As a result, plants can complete their life cycle without experiencing mortality from extreme stress (McKay *et. al.,* 2003). The photoperiodic response gene GI, the florigen genes FLOWERING LOCUS T (FT), and TWIN SISTER OF FT (TSF), are the sole ones necessary for drought escape under inductive long-day conditions (Riboni *et. al.,* 2013).

Drought stress promotes transcriptional activation of the florigen genes by releasing the transcriptional repression at the FT/TSF promotors in an ABA- and photoperiod (through GI)-dependent manner (Riboni *et. al.,* 2013). CO is necessary for the ABA-dependent activation of FT but not TSF (Riboni *et. al.,* 2016). Increased amounts of florigen cause the floral integrator

SUPPRESSOR OF OVEREXPRESSOR OF CONSTANS (SOC1) to become active, which starts flowering. According to Riboni *et. al.,* (2013), SOC1 activation aids in TSF overexpression, which further raises the levels of florigen. Due of the repressive effect of SHORT VEGETATIVE PHASE (SVP)/blooming LOCUS C (FLC) on SOC1, during short-day conditions, ABA delays blooming under drought stress (Riboni *et. al.,* 2016). Additionally, the photoperiod-dependent flowering transcription factor family, which includes the NUCLEAR FACTOR Y (NF-Y) component c (Kumimoto *et. al.,* 2008, 2010), is thought to have a role in drought resistance. According to Hwang *et. al.,* (2019), the ABA-response element (ABRE)-binding factors (ABFs) engage with NF-Y subunit c-3/4/9 and cause SOC1 to stimulate flowering. Along with *Avena barbata* (Sherrard and Maherali, (2006), Brassica rapus (Franks, 2011), and other plants, *Arabidopsis* has a drought escape mechanism (McMaster and Wilhelm, (2003); Gol *et. al.,* 2021). Other species, like rice, put their life cycle on hold during drought stress in order to continue it when the stress is gone (Galbiati *et. al.,* 2016). A biochemical link between stress and the photoperiodic flowering pathway is also provided here by primary integrators of day duration. When considered as a whole, drought escape is a developmental response that is photoperiod dependent since it is the immediate result of the perception of the long-day photoperiod under drought stress.

The photoperiod sensing elements GI and NF-Y are known to also affect drought tolerance without any direct connection to the perception of the photoperiod, in addition to their function during drought escape. According to Zeevaart (1971), photoperiodic regulation at least partially affects the synthesis and signalling of ABA.

The expression of NINE-CIS-EPOXYCAROTENOID DIOXYGENASE3 (NCED3) is regulated by GI in a complex with the bZIP transcription factor ENHANCED EM LEVEL (EEL), which is involved in ABA signalling responses, according to a recent study (Baek *et. al.,* 2020). A rate-limiting enzyme for the production of ABA is encoded by NCED3 (Iuchi *et. al.,* 2001). By attaching to the ABA-responsive element motif in the NCED3 gene promotor, the GI-EEL complex positively controls the diurnal ABA synthesis, increasing ABA production and improving drought tolerance (Baek *et. al.,* 2020). In gi-1 and eel mutants subjected to dehydration, NCED3 transcript abundance and ABA content reduced, which was correlated with their dehydration-sensitive phenotype (Baek *et. al.,* 2020). These findings suggest that GI and EEL work synergistically to increase drought tolerance in plants by controlling ABA homeostasis.

Han et al.'s (2013) 2013a study discussed the function of GI during the drought stress response. MiRNA172e has the strongest response to drought stress and both its level and function are elevated in response to drought stress (Han *et. al.,* 2013a). Long days and drought circumstances cause GI to encourage the production of pre-miRNA172, which suppresses WRKY44 and results in drought tolerance. Although the precise underlying process is not entirely understood, Han *et. al.,* (2013a) suggest that it may be related to sugar metabolism. In *Arabidopsis* (Li *et. al.,* 2013; Ni *et. al.,* 2013), *Zea mays* (Nelson *et. al.,* 2007; Su *et. al.,* 2018), *Populus* (Han *et. al.,* 2013b), *Oryza sativa* (Chen *et. al.,* 2015), and *Citrus* (Pereira *et. al.,* 2018), NF-Y transcription factors have been found to enhance drought tolerance. Micro-array research showed that oxidative stress-responsive genes are highly increased during drought stress in *Arabidopsis*, and overexpression of NF-YA5 improved drought tolerance (Li *et. al.,* 2008). ABA biosynthesis, signalling, and stress-responsive genes were expressed more often in transgenic *Arabidopsis* plants overexpressing the soybean NF-YA3 gene (Ni *et. al.,* 2013). The overexpression of NF-YB1 in *Arabidopsis* also improved plant drought resistance independent of ABA signalling, despite the fact that this study revealed an ABA-dependent signalling leading to greater drought resistance (Nelson *et. al.,* 2007). In addition to their function in drought tolerance, NF-Y transcription factor overexpression enhances salt stress resistance (Li *et. al.,* 2008) as well as freeze tolerance (Shi and Chan, 2014).

c. Stress Caused by Photoperiod Changes

In *Arabidopsis thaliana* plants that have acclimated to short days, abrupt changes in the photoperiod, particularly its lengthening, result in photoperiod stress (Nitschke *et. al.,* 2016, 2017). The photoperiod stress response, which was first noticed after the light period was extended by 24 h, is characterised by the following typical sequence of occurrences: The expression of stress marker genes, such as ZINC FINGER of *ARABIDOPSIS THALIANA*12 (ZAT12) and BON ASSOCIATED PROTEIN1 (BAP1), is raised during the night after an extended light period, the concentration of the stress hormones JA and SA rises, and oxidative stress takes place. The substantial decline in the ascorbic acid (ASC) redox state and the production of peroxides are associated with the nightly increase in oxidative

stress. According to Abuelsoud *et. al.,* (2020), the creation of peroxide is accompanied with an increase in the expression of the gene PEROXIDASE (PRX), as well as increased PRX and decreased catalase activity. The photosystem II maximum quantum efficiency declines the following day, and PCD eventually occurs in the leaves (Nitschke *et. al.,* 2016, 2017).

Arabidopsis plants with cytokinin (CK) deficiency, which have a very potent stress response, are where photoperiod stress was initially identified. Among the CKs, trans-zeatin, in particular, has a protective role that is mediated via the transcriptional regulators *ARABIDOPSIS* RESPONSE REGULATOR2 (ARR2), ARR10, and ARR12 and the *ARABIDOPSIS* HISTIDINE KINASE3 (AHK3) receptor (Frank *et. al.,* 2020). Some clock mutations, such as CCA1, LHY and ELF3, also exhibit a higher molecular and phenotypic response to abrupt photoperiod prolongations. CCA1 and LHY, two essential regulators of the circadian clock, have decreased expression or impaired function in both CK-deficient plants and photoperiod stress-sensitive clock mutants. This suggests that the ability to deal with photoperiod stress requires a working clock (Nitschke *et. al.,* 2016, 2017). Only a 4 h increase in the light duration is needed to cause the short-day-entrained *Arabidopsis* plants to produce ROS and express stress marker genes the next night. The power of the photoperiod stress response increases with longer light phase prolongations, suggesting that light duration affects the response's potency (Abuelsoud *et. al.,* 2020). Longer light period prolongations result in true stress (distress), whereas shorter light period prolongations, which create lower stress levels, are viewed as not harmful and may provide a positive stress (eustress) (Krasensky-Wrzaczek & Kangasjarvi, 2018).

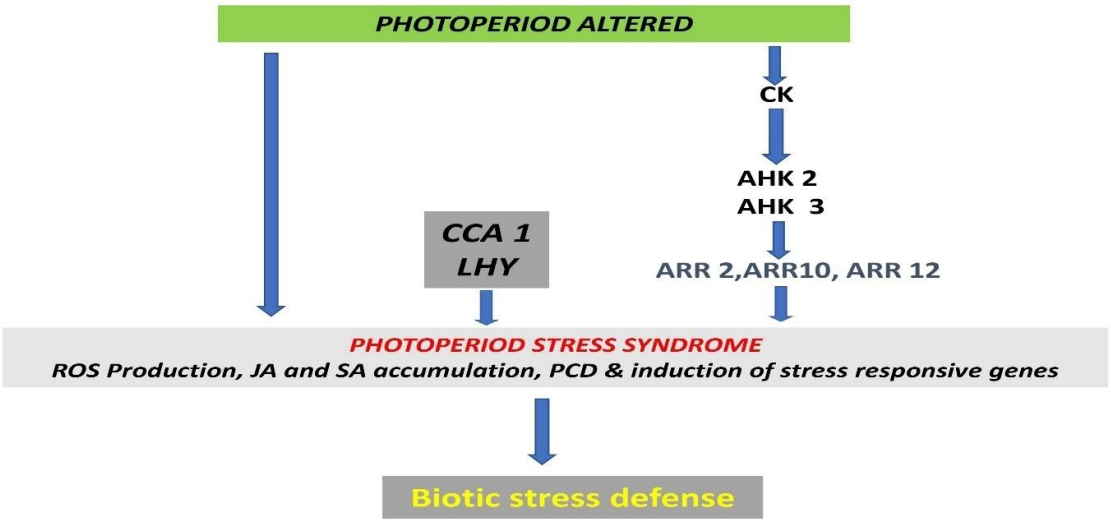


Fig.4 **Stress Caused by Photoperiod Changes**

2. IMPORTANCE OF PHOTOPERIOD

Plants depend on photoperiod as a trigger for several essential developmental processes, including flowering, seed germination, and dormancy. Photoperiodic responses assist plants adjust to environmental changes, such as the onset of new seasons, and enable them to maximise their rate of growth and procreation.

For instance, the photoperiodic response causes flowering in short-day plants during seasons with less daylight, such the fall or winter. By doing this, the plant is able to finish its reproductive cycle before the beginning of unfavourable environmental conditions like frost or drought.

In contrast, during seasons of prolonged sunshine, such the spring or summer, long-day plants rely on photoperiod to initiate blooming. This enables the plant to benefit from ideal growing circumstances and increase the likelihood of successful reproduction.

In order to control the growth and development of crops, farmers and horticulturists can also use photoperiodic reactions. Growers can encourage vegetative development, induce or suppress blooming, and regulate the timing of seed germination by adjusting the length and intensity of light exposure.

In addition, research on photoperiodic responses has aided in the discovery of numerous crucial genes and molecular mechanisms that control developmental processes in plants. The fields of agriculture, biotechnology, and conservation can all benefit from this knowledge.

There are clear benefits to being able to synchronise some developmental stages to specific seasons of the year when environmental conditions are more likely to be beneficial. For example, shifting reproduction to the spring would increase the likelihood that the young would survive by giving them as much time as possible to develop before being exposed to the harsh winter weather. Thus, plants and animals that have developed mechanisms to perceive seasonal differences through the detection and response to changes in photoperiod have a selection advantage. The photoperiod is the ratio of light to dark in a 24-hour cycle. At the equator (zero latitude), the photoperiod is always 12 hours long and 12 hours long, but as you move from the equator to either of the earth's poles, due to the tilt of the earth's axis towards the sun, the lengths of the light and dark periods change and become unequal divisions of the 24-hour cycle. The extremes of daylength and night length increase as you move closer to the poles, when photoperiods of 24 hours of light or 24 hours of darkness are occasionally encountered throughout the year. Except at the equator, the annual rotation of the earth around the sun causes the photoperiod at a given latitude to change throughout the year, with daylengths growing longer in summer and shorter in winter. The summer solstice marks the annual maximum in daylength for a given latitude, while the winter solstice marks the shortest day length.

The ability to adapt to photoperiod not only allows an organism to coordinate different responses to specific times of the year, but also allows an organism to anticipate changes in environmental conditions that are projected to occur at roughly the same time each year. For instance, many tree and perennial plant species in northern latitudes use the dwindling daylength in fall as a cue to induce cold tolerance and bud hibernation in preparation for the upcoming bitterly cold winter Additionally, the capacity to respond to photoperiod can aid an organism in occupying a niche in either space or time. For example, some species, like the liverwort, can survive in the desert by using long days as a signal to enter a dormant state during the dry summer months, while ground-level woodland plants may use short days to induce flowering in early spring, allowing them to finish seed production before the leaf canopy fully forms and reduces the amount of light available (Thomas and Vince-Prue, 1997).

Short-day plants (SDP), whose responses are induced when the photoperiod is below the critical daylength (CDL), long-day plants (LDP), whose responses are induced when the photoperiod exceeds the CDL, and day-neutral plants (DNP), which do not respond to photoperiod, are the three main types of photoperiod responses. Thus, the CDL marks the transition from a noninductive to an inductive photoperiod, and the value of the CDL varies greatly between species and between plants within a single species. Some SDPs, like *Xanthium strumarium*, have long CDLs (15.5 h), allowing them to bloom in long days (LDs) of 15 h light, whereas some LDPs, like some cultivars of *Lolium perenne* and *Lolium temulentum*, have low CDLs and are capable of blooming in short days (SDs), as low as 9 h (Thomas and Vince-Prue, 1997). The word "obligate response" refers to plants whose blooming can only take place during the inducing photoperiod, whereas the term "facultative response" refers to plants whose flowering is encouraged by LDs or SDs but which can still flower during the other photoperiod. The difference between an inducing and noninducing photoperiod in some tropical species can be as tiny as 30 min, suggesting that plants are very accurate timekeepers (Borchert *et. al.,* (2005). This is crucial because even little errors in photoperiod measurement might cause the induction of the response to occur up to a few weeks early or late. The circadian clock, an endogenous time-keeping system that is discussed in greater detail later, allows plants to measure time. In *Hyoscyamus niger*, the CDL gets shorter with lower night time temperatures, while seedlings of *Pharbitis nil* have a shorter CDL than mature plants (Thomas and Vince-Prue, 1997). The CDL is not set and is known to change with ambient circumstances and plant age.

Sometimes a particular developmental life-cycle is created by combining a response to photoperiod with other responses to other environmental cues. Although they experience inductive photoperiods, biennial plants like henbane (*Hyoscyamus niger*) need a lengthy period of cold weather during the winter to fulfil a vernalization requirement, which will then allow them to flower and set seed. As a result, the life cycle lasts for two years, with flowering occurring in the second year throughout the spring or early summer. The induction of flowering by photoperiod can also be replaced by other

environmental factors like vernalization, high temperatures, high radiation, or low nitrogen. Additionally, the response to photoperiod may also be modulated or even suppressed by other environmental factors (Bernier and Perilleux, 2005). Although the relationships between the photoperiodic system and other processes that influence blooming are still not fully understood at the molecular level, it appears that genes that work primarily in one pathway can occasionally be regulated by other pathways. Therefore, rather than thinking of the photoperiodic route in isolation, one should always see it as a part of an interconnected network of pathways that regulate flowering.

The majority of the molecular mechanisms discussed here are related to the regulation of blooming, even though flowering is just one of several reactions that plants have to photoperiod. blooming has also been the subject of the most extensive research.

3. COMPETENCE TO RESPOND TO PHOTOPERIOD

A plant's genetic make-up determines how it responds to the photoperiod, or photoperiodic competence, which can change between species and even among populations of the same species.

With the help of their red/far-red light-absorbing phytochromes and their blue light-absorbing cryptochromes, plants have photoreceptors that can recognise changes in the length of the day. These photoreceptors set off a chain of molecular and genetic occurrences that ultimately alter the physiology and behaviour of the plant.

The ability of a plant to adapt to photoperiod is dependent on the expression of particular genes that manage the synthesis and transportation of vital regulatory hormones like florigen. A number of environmental variables, including temperature, moisture content, and the availability of nutrients, can also have an impact on the timing and strength of photoperiodic responses.

In rare circumstances, selective breeding or genetic engineering can change how a plant reacts to photoperiod. For instance, scientists have created crop types with modified photoperiodic responses that enable them to flourish and bear fruit in a variety of environmental settings, including those with short growing seasons or low light levels.

During the course of plant development, the capacity to adapt to factors that promote floral growth, such as causing photoperiods, varies. The juvenile phase of most plants ensures that the plant has enough resources to support flower and subsequent fruit production by delaying floral induction until a specific developmental stage has been reached. An inductive stimulus that would be adequate to cause flowering in an adult plant does not work on juvenile plants. In herbaceous species like *Arabidopsis*, the juvenile phase can last just a few days, whereas in woody tree species, it might last for several years (Hackett, 1985). Gibberellic acid (GA), temperature, photoperiod, and light integral have all been demonstrated to influence how long the juvenile phase lasts and, consequently, the age at which the plant may respond to photoperiod (Hackett, 1985; Chien and Sussex, 1996; Telfer *et. al.,* 1997; Adams *et. al.,* 1999, 2001). In many plants, the transition from the juvenile to adult phase is associated with phenotypic changes as well as the beginning of the competence to flower. Examples include changes in leaf shape in Zea mays and ivy (Hedera helix), as well as the formation of abaxial trichomes in adult *Arabidopsis thaliana* (Poethig, 1990; Bongard-Pierce *et. al.,* 1996; Telfer *et. al.,* 1997). These phenotypical markers have been used to identify numerous mutants with altered juvenile phase lengths, such as the maize teopod (tp) and early phase change (epc) mutants, which have shortened and extended juvenile phases, respectively (Poethig, 1988; Dudley and Poethig, 1993; Vega *et. al.,* 2002). The TP1 and TP2 genes regulate juvenility non-cell-autonomously, according to research on the teopod mutants where sectors of wild-type tissue were generated in TP1 and TP2 mutants (Dudley and Poethig, 1993). The rice mori1 mutant, in contrast to most mutants, is unable to transition from the juvenile to adult phase, and as a result, will not blossom even when grown under stimulating SD photoperiods (Asai *et. al.,* 2002). Most mutants have been discovered to have altered juvenile phase lengths. HASTY (HST), ZIPPY (ZIP), SERRATE (SE), and SQUINT (SQN) are four genes studied in *Arabidopsis* that have been linked to the length of the juvenile phase (Clarke *et. al.,* 1999; Berardini *et. al.,* 2001; Bollman *et. al.,* 2003; Hunter *et. al.,* 2003). All of these genes have the ability to shorten the juvenile phase when they are mutated, suggesting that the purpose of these genes is to maintain the length of the juvenile phase. Following the discoveries that ZIP encodes an ARGONAUTE protein that is required for the production and/or stability of ta-siRNAs (Fahlgren *et. al.,* 2006; Hunter *et. al.,* 2006), HST is involved in the synthesis or stability of some

miRNAs, and SE is known to act in a miRNA gene silencing pathway (Grigg *et. al.,* (2005), it has been determined that miRNAs and trans-acting small interfere. In addition, it was shown that plants with mutations in the genes DICER-LIKE 4 (DCL4), RNA-DEPENDENT POLYMERASE 6 (RDR6), and SUPPRESSOR OF GENE SILENCING 3 (SGS3), which are known to function in gene silencing, have a shorter juvenile phase. (Peragine *et. al.,* 2004; Xie *et. al.,* 2005). These three genes, like ZIP, play a role in the biosynthesis of ta-siRNAs, and similar to ZIP, their mutant phenotypes are mostly linked to the juvenile-adult phase change rather than the extremely pleiotropic phenotypes of miRNA biosynthesis mutants like HST (Bollman *et. al.,* 2003). According to Willmann and Poethig 2005), this has led to the hypothesis that ta-siRNAs are likely to have a more limited function in plant growth than miRNAs. The target of the ta-siRNA is a gene that promotes the adult state (or represses the juvenile state), according to a model of how ta-siRNAs effect the juvenile to adult phase shift. If the manufacture of ta-siRNAs is disrupted, as it is in the mutants' zip, sgs3, rdr6, and dcl4, then the transcript of the target gene won't be destroyed, shortening the juvenile phase and hastening the transition to adulthood (Bäurle and Dean, 2006).

There is now a lot of study being done on the identities of the miRNAs responsible for producing the ta-siRNAs, as well as the identities of the ta-siRNAs and the target genes they regulate. The mRNAs of multiple AUXIN RESPONSE FACTOR (ARF) genes, including ARF3, are targeted for degradation by the TAS3 family of ta-siRNAs, which are produced by miR390 (Fahlgren *et. al.,* 2006). Juvenile phase length was demonstrated to be influenced by the regulation of ARF3 gene transcript levels by TAS3 ta-siRNAs, indicating that ARF3 is one of the target genes implicated in juvenility regulation.

The modulation of the juvenile-adult phase transition is mediated by microRNAs, trans-acting small interfering RNAs (ta-siRNAs), and the target genes of these molecules. In *Arabidopsis*, the transition to the adult phase is promoted by AUXIN RESPONSE FACTOR 3 (ARF3) and SQUAMOSA PROMOTER BINDING PROTEIN-LIKE 3 (SPL3), but GLOSSY15 (GL15) in Zea mays suppresses it.

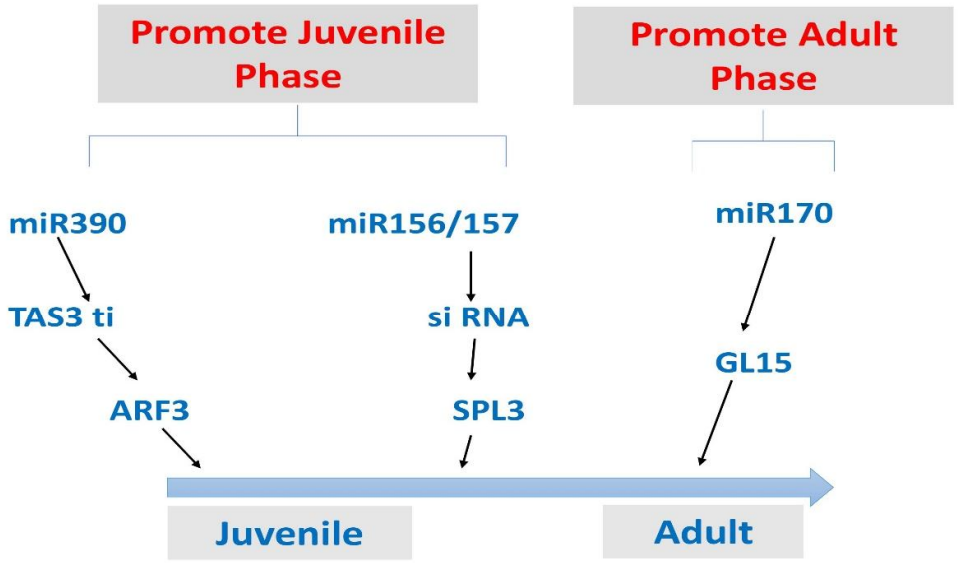


Fig.5 **Competence To Respond To Photoperiod**

It has been demonstrated that over-expression of miR156 can prolong the juvenile period, mostly through suppressing the SBP-box gene SQUAMOSA PROMOTER BINDING PROTEIN-LIKE 3 (SPL3). (Wu and Poethig, (2006). A miRNA-responsive element that is complementary to miR156 and miRNA157 is located in the 3′ untranslated region of the SPL3 mRNA and regulates SPL3 at the translational level in addition to regulating its transcript levels (Gandikota *et. al.,* 2007). According to research (Wu and Poethig, 2006); Schwarz *et. al.,* 2008), SPL3 and other miR156-regulated SBP-box

genes, SPL4, SPL5, SPL9, and SPL15, are target genes involved in promoting the adult state, the end of the juvenile phase, as well as flowering. MiR156 levels are lower and SPL3 mRNA levels are higher in the hst-6 mutant, which is compatible with the mutant's shorter juvenile phase length (Park *et. al.,* 2005); Wu and Poethig, 2006).

In contrast to the pattern of expression of another miRNA, miR172, which also influences the length of the juvenile phase in maize (Lauter *et. al.,* (2005), it has been demonstrated that the level of miR156 is higher in juvenile tissue than adult tissue (Wu and Poethig, (2006). An APETALA2 (AP2)-like gene called GLOSSY15 (GL15), which is expressed in young leaves and supports the juvenile phase, is the target of miR172 in maize.

This is covered in more detail in section III (4). In *Arabidopsis*, miR172 targets other AP2-like genes that are involved in suppressing the floral transition (Aukerman and Sakai, (2003); Schmid *et. al.,* (2003); Jung *et. al.,* 2007). It is possible that miR156 and miR172 are regulated by the same regulatory system because of the reciprocal expression patterns of their target genes SPL3 and GL15, which are known to inhibit and enhance the juvenile period, respectively.

As a result, the length of the juvenile period is regulated post-transcriptionally by both ta-siRNAs and miRNAs. It will be interesting to discover if the non-cell-autonomous behaviour of the TP1 and TP2 genes, as reported by Dudley and Poethig (1993), is explained by the intercellular migration of these short RNA molecules. It will be fascinating to find out whether the production or activity of miRNAs and ta-siRNAs is influenced by environmental factors that alter juvenile phase length. MiR172 levels in *Arabidopsis* have been demonstrated to be influenced by photoperiod and light quality, with levels being higher in LDs and blue light (Jung *et. al.,* 2007).

Both in the leaves and at the apex, there is proof that the juvenile condition occurs. Both *Bryophyllum* juvenile shoots and *Ipomaea batatas* juvenile seedlings were capable of flowering after being grafted onto florally induced mature plants (Zeevaart, 1962; Takeno, 1991), demonstrating that in these instances, the characteristics of the leaves on the stock plants were the deciding factor. On the other hand, when ripe buds of Japanese larch (Larix kaempferi) were grafted onto mature trees, flowering occurred, indicating that the condition of the apex was the determining element in this case (Robinson and Wareing, 1969). Accordingly, depending on the species, adjustments to the leaves and/or apices may play a role in the move from the juvenile to the adult, florally competent phase. Research on maize has revealed that this change takes place gradually, with leaves developing during this time displaying both juvenile and adult traits at their bases (Orkwiszewski and Poethig, 2000). These changes are a result of factors that are not related to the shoot apical meristem (SAM).

Along with the transition from the juvenile to adult phase, adult plants' ability to respond to inducing cues also evolves over time. By grafting tobacco (*Nicotiana tabacum*) apices of various ages onto stock plants, it was demonstrated that as plants age, the SAM responds to inducing signals more quickly (Singer *et. al.,* 1992). This behaviour in *Arabidopsis* may be partially explained by variations in the expression of the meristem identity gene LEAFY (LFY), which gradually increases throughout vegetative growth in noninducing conditions (Blazquez *et. al.,* 1997). As photoperiod was shown to modulate the effect of constitutive LFY over-expression on flowering time (Weigel and Nilsson, 1995), it has also been hypothesised that the apex changes in its competence to respond to LFY activity. In fact, analysis of LFY over-expression in late-flowering mutants demonstrated that some flowering time genes affected LFY transcription while others affected the response to LFY (Nilsson *et. al.,* 1998), and researchers are just now beginning to understand the underlying molecular mechanisms (Chae *et. al.,* 2008; Lee *et. al.,* 2008). *Nicotinia*, *Petunia hybrida*, and *Impatiens balsimina* are likely examples of plants whose ability to respond to LFY activity is altered, as LFY homologues have been found to be expressed in both vegetatively and florally induced apices of these plants (Kelly *et. al.,* 1995; Pouteau *et. al.,* 1997; Souer *et. al.,* 1998). The genes PENNYWISE (PNY) and POUND-FOOLISH (PNF) have also been identified as being important in regulating the apex's ability to respond to floral inducing cues. The upregulation of the floral integrator gene SUPPRESSOR OF OVEREXPRESSION OF CO 1 (SOC1) in the apex suggests that mutations in these genes hinder the vegetative to floral transition in inducing conditions despite the induced state of the plant (Smith *et. al.,* (2004). Recent research (Kanrar *et. al.,* 2008) has demonstrated that pny pnf double mutants block the activation of LFY by FLOWERING LOCUS T (FT).

Vernalization affects the ability of the apex in plants that require a vernalization response to respond to stimulating signals, such as inducing photoperiods, through the regulation of a repressor of blooming. This repressor in *Arabidopsis* is called FLOWERING LOCUS C (FLC). High FLC

concentrations suppress FT expression and inhibit apical induction (Searle *et. al.,* (2006). The FRIGIDA (FRI) gene, the autonomous and vernalization flowering pathways, and FLC itself all control FLC expression. The repression at the apex is relieved by the vernalization and autonomous pathways, which also increase the competence of FLC to be induced by other pathways, such as the photoperiodic pathway (Mouradov *et. al.,* 2002; Henderson *et. al.,* 2003); Bäurle and Dean, 2006).

1. PHOTOPERIODIC RESPONSE PATHWAY

A plant's reaction to variations in day duration is controlled by the intricate biochemical and genetic process known as the photoperiodic response pathway. The pathway comprises a number of steps that lead to the generation and movement of vital regulatory hormones, including florigen, which start flowering or other processes related to development.

The photoperiodic response pathway is described in the following way:

i. **Photoreception:** The phytochromes, which absorb red and far-red light, and the cryptochromes, which absorb blue light, are photoreceptors in plants that sense changes in the length of the day and send signals to the plant cell.
ii. **Signal transduction:** A complex web of signalling pathways involving several proteins, enzymes, and secondary messengers is used to transfer photoreceptor signals.
iii. **Gene expression:** Changes in the expression of particular genes that regulate the synthesis and movement of important regulatory hormones, such as florigen, are brought on via signal transduction pathways.
iv. **Hormone production and transport:** The regulating hormones are created in particular tissues and transferred to the apical meristem, where they start developmental processes like flowering.
v. **Response:** Initiation of various developmental stages, such as flowering, seed germination, or dormancy, is how the plant reacts to photoperiodic signals.

Temperature, moisture, and the availability of nutrients are only a few of the environmental elements that have an impact on the photoperiodic response pathway. Through selective breeding or genetic engineering, the timing and intensity of the photoperiodic response can also be changed. This has significant effects on crop output and adaptation to changing climatic conditions.

A method to measure daylength is necessary for the capacity to react to photoperiod. This mechanism has been demonstrated in the facultative LDP *Arabidopsis*, which is induced to flower earlier in LDs than in SDs. This interaction of light signals, which are detected by photoreceptors like phytochromes, cryptochromes, and the blue light receptor F-box proteins ZEITLUPE (ZTL) and FLAVIN-BINDING KELCH REPEAT F-BOX 1 (FKF1), with circadian clock components and the CONSTANS (CO) The floral integrator gene FT and the closely related TWIN SISTER OF FT (TSF) gene are directly induced by the CO protein, a key regulator of photoperiodic flowering (Samach *et. al.,* 2000; Wigge *et. al.,* 2005; Yamaguchi *et. al.,* 2005). As evidenced by the fact that delaying flowering in heterozygous plants when CO levels are cut in half, the CO protein is expressed at extremely low levels and its abundance is the limiting element in the induction of flowering by photoperiod (Robson *et. al.,* 2001). The photoperiodic system precisely controls CO protein levels throughout the day, with levels rising starting around 10 hours after dawn and peaking at least 16 hours later (*i.e.,* towards the end of an LD) (Valverde *et. al.,* 2004). Experiments showing that flowering could be induced by changing the light/dark regime or CO expression so that high levels of CO expression occurred in the light period in SDs (Roden *et. al.,* 2002; Yanovsky and Kay, 2002) showed that the coincidence of high levels of CO expression with light is necessary for floral induction.

a) Circadian rhythm

The circadian clock is a biological clock found inside all living things that enables them to anticipate and adjust to environmental changes, such as the daily cycle of light and darkness. The Latin words "circa" and "dies," which both mean "around," are the roots of the English word "circadian."

The circadian clock controls several physiological processes in plants, such as growth, development, metabolism, and reactions to biotic and abiotic stressors. The photoperiodic response pathway is timed in accordance with the circadian clock, ensuring that plants react to changes in day length appropriately.

A complex network of genes and proteins that interact with one another in a feedback loop controls the circadian clock. These genes, which encode proteins that control the expression of other clock genes, include the TIMING OF CAB EXPRESSION 1 (TOC1), CIRCADIAN CLOCK ASSOCIATED 1 (CCA1), and LATE ELONGATED HYPOCOTYL (LHY) genes in plants.

Numerous environmental signals, including light, temperature, and the availability of nutrients, have an impact on the circadian clock and can either synchronise or reset it. To synchronise the clock with the 24-hour cycle of light and darkness, for instance, exposure to light at particular times of the day might reset the clock.

The circadian clock has significant effects on plant development and growth, as well as crop productivity and environmental adaptation. An active field of study, understanding the chemical and genetic underpinnings of the circadian clock in plants has significant implications for biotechnology, agriculture, and conservation.

An endogenous clock, the circadian clock is built on a network of connected negative feedback loops. These feedback loops allow the clock to keep running under constant conditions, *i.e.,* without being entrained by zeitgeber signals like variations in light or temperature, which synchronise the circadian clock with the outside world.

The clock will not be detailed in great length here because multiple reviews on the subject have recently been published (Gardner *et. al.,* 2006; McClung, 2006; Hotta *et. al.,* 2007). The clock regulates a number of reactions that must be coordinated to specific times of the daily cycle. The TIMING OF CAB EXPRESSION 1 (TOC1) gene, whose product positively regulates two largely redundant Myb transcription factors, LATE ELONGATED HYPOCOTYL (LHY), and CIRCADIAN CLOCK ASSOCIATED 1 (CCA1), makes up the fundamental negative feedback loop. By binding to an evening element in its promoter, the LHY and CCA1 proteins subsequently feedback to negatively regulate the expression of TOC1 (Alabadi *et. al.,* 2001). The 26S proteasome targets TOC1 for degradation, and ZTL controls the stability of the TOC1 protein in this situation (Más *et. al.,* (2003). The PSEUDO RESPONSE REGULATOR (PRR), GIGANTEA (GI), and LUX ARRHYTHMO (LUX) genes are involved in additional feedback loops (Gardner *et. al.,* 2006; McClung, 2006).

b) Role of light

By controlling the timing and advancement of the flowering process, light plays a crucial role in the formation of flowers. Particularly, light plays a crucial role as an environmental cue that initiates the switch from vegetative to reproductive growth and affects a number of aspects of flower development, including the timing of flower bud initiation, the quantity and size of flowers produced, and the colour and scent of the flowers.

Plant photoreceptor proteins, such as the red/far-red-absorbing phytochromes and the blue-light-absorbing cryptochromes, pick up light signals. The complicated signalling cascade that these photoreceptors start controls the expression of numerous genes involved in flower formation.

The photoperiod, or the length and intensity of light exposure, affects when flower bud initiation occurs. While some plant species are day-neutral and can flower at any time of day, many require a certain photoperiod to begin flowering. A collection of genes called "florigen" genes, which are controlled by light and other environmental stimuli, orchestrate the photoperiodic response.

By controlling the creation of chlorophyll, which is essential for photosynthesis and the generation of energy for plant growth and development, light intensity also influences the development of flowers. In comparison to plants produced under ideal lighting circumstances, low light-grown plants may produce fewer or smaller blooms. By controlling the synthesis of pigments and secondary metabolites like anthocyanins and terpenoids, which are responsible for floral colour and aroma, the light can also affect the colour and scent of flowers.

Overall, a variety of biochemical, genetic, and physiological mechanisms are involved in the intricate and multidimensional impact that light plays in flower formation. Understanding these procedures is crucial for environmental management, conservation, and agriculture's best flower production.

The three main purposes of the light signal in the photoperiodic response mechanism are as follows.

(I)

It synchronises the clock to a 24-hour cycle since without entrainment it would quickly lose phase with the regular day/night cycle (the clock has a free-running period of between 22 and 29 hours; Michael *et. al.,* 2003). Blue light working through ZTL and the cryptochromes cry1 and cry2 as well as red light acting through the phytochromes phyA, phyB, phyD, and phyE (the involvement of phyC has not been established) are both implicated in the entrainment of the clock (Somers *et. al.,* 1998; Devlin and Kay, 2000; Kim *et. al.,* 2007b). Light signals cause the clock's essential genes, such as LHY, CCA1, and PRR9, to express, which entrains the clock (Wang and Tobin, 1998; Kim *et. al.,* 2003; Farré *et. al.,* 2005). Blue light improves ZTL's stability by encouraging its interaction with another clock component, GI. This imparts a rhythm to ZTL protein levels, which causes an amplified and sharper peak in TOC1 protein levels (Kim *et. al.,* 2007b). Light also affects clock components at the post-transcriptional level. Correct clock entrainment is crucial because it determines how the clock's outputs, such as CO, manifest themselves in relation to the daily cycle of light and dark.

(II)

It encourages the blue-light-dependent interaction between FKF1 and GI that is required for the degrading of the transcriptional repressor of CO known as CYCLING DOF FACTOR 1 (CDF1), hence promoting CO expression (Sawa *et. al.,* 2007). The CDF1 over-expressing lines and the GI and fkf1 mutants are all late flowering and have decreased amounts of CO mRNA (Suàrez-López *et. al.,* 2001; Imaizumi *et. al.,* 2003, 2005). Although FKF1, GI, and CDF1 are all regulated by the circadian rhythm, CDF1 expression peaks sooner in the morning than FKF1 and GI do (Fowler *et. al.,* 1999; Imaizumi *et. al.,* 2003, 2005). According to one theory, CDF1 binds to the CO promoter early in the day and prevents CO transcription. A blue-light-dependent complex formed by the later-produced GI and FKF1 binds to CDF1 and allows FKF1 to target CDF1 for destruction by the 26S proteasome, releasing CO from repression and permitting expression towards the end of an LD (Sawa *et. al.,* 2007).

(III)

It controls the stability of CO proteins. Far-red and blue light working through phyA and the cryptochromes, respectively, improve the stability of CO, whereas red light acting through phyB increases the breakdown of CO by the proteasome. The morning is dominated by the phyB-mediated degradation, which is then counteracted by the activity of phyA and the cryptochromes later in the day. This causes CO to stabilise towards the conclusion of an LD (Valverde *et. al.,* 2004). The CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) protein, a RING finger ubiquitin ligase that controls the stability of transcription factors involved in the plant's response to light, targets the CO protein for degradation by the proteasome in the absence of light (Osterlund *et. al.,* 2000; Holm *et. al.,* 2001; Seo *et. al.,* 2003). Because COP1 is excluded from the nucleus in the light (von Arnim and Deng, 1994), as well as because cryptochromes directly suppress COP1 in the light (Wang *et. al.,* 2001), COP1 activity rises in the dark than in the light. Despite the fact that both SDs and LDs express CO at high levels during the dark period (Suàrez-López *et. al.,* 2001), the activity of COP1 prevents the CO protein from building up during the dark period (Jang *et. al.,* 2008). Members of the SUPPRESSOR OF PHYTOCHROME A-105 (SPA) family of proteins, which have been demonstrated

to bind to both CO and COP1 and to control the ubiquitin ligase activity of COP1 (Saijo *et. al.,* 2003; Laubinger *et. al.,* 2006), may be involved in the degradation of CO by COP1.

c) Role of CO

The CO gene, also known as CONSTANS, is essential for controlling the timing of blooming in plants. The florigen genes, which are crucial for floral initiation and development, are activated by CO, a transcription factor, along with other genes involved in flower formation.

CO is produced in response to environmental cues that control the timing of blooming, such as day length. The photoperiodic pathway induces CO expression in response to the presence of light in long-day plants, which need a period of long days to trigger blooming. The photoperiodic pathway suppresses CO expression in short-day plants, which must experience a period of short days to trigger blooming.

The florigen genes are activated by CO protein, which is produced in the plant's leaves and subsequently transferred to the apical meristem. The florigen protein, which is created by the florigen genes, then causes the switch from vegetative growth to reproductive growth, resulting in the development of flowers. The protein is delivered to the shoot apex.

The genetic makeup and environmental context of the plant affect the complicated role that CO plays in flower development. The timing or length of flowering can change as a result of mutations in the CO gene or its regulatory mechanisms, which can have significant effects on crop output and response to shifting environmental conditions.

The expression and activity of CO, a key player in the control of flowering time in plants, are strictly regulated by environmental cues including day length as well as by other genes involved in flower formation.

The circadian oscillation of CO expression is modified by the light-dependent regulation of CO at both the transcriptional and post-transcriptional levels, allowing higher levels of CO expression towards the end of an LD (between 10 and 16 hours after dawn), as well as promoting the stability of the CO protein at this time of day.

This enables significant accumulation of CO protein during the light phase, which strongly induces the FT gene and ultimately leads to blooming. However, during the daytime in SDs, CO expression does not reach substantial levels. Because the circadian regularity of GI and FKF1 expression is such that the proteins are not present in sufficient numbers to overcome the CDF1-mediated suppression of CO, there is no GI/FKF1-induced daytime peak of CO expression. Both FT expression and flowering are not triggered. It must be kept in mind that a natural day length varies along a continuum between (and beyond) these values, whereas in most experimental designs an LD consists of 16 hours of light and an SD of 8 or 10 hours of light. The CDL is the moment in LDPs, like *Arabidopsis*, where the photoperiod has gotten long enough to be florally inductive, or has crossed the threshold at which CO protein levels have increased enough to induce FT and blooming. The CDL varies between and within species of plants, as was already mentioned. This must be due to somewhat varied CO gene expression patterns and CO protein accumulation-related genes.

CONSTANS (CO)-dependent photoperiodic pathway impacts of light. Not all of the clock's parts are displayed. Induction is denoted by arrows, while inhibition is denoted by bars at the ends of lines. Gene transcripts are marked with italics, whilst proteins are identified with conventional type. R stands for red, FR for far-red, and B for blue. TSF, TWIN SISTER OF FT; GI, GIGANTEA; LHY, LATE ELONGATED HYPOCOTYL; RED AND FAR RED INSENSITIVE 2, RFI2; SUPPRESSOR OF PHYTOCHROME A-105, SPA; TIMING OF CAB EXPRESSION 2, TOC2; ZEITLUPE; CCA1, ZTL, CIRCADIAN CLOCK ASSOCIATED 1.

Other variables, in addition to GI, FKF1, and CDF1, also influence CO expression. Since CO expression is not de-repressed as one might anticipate when the CDF1 repressor is expressed at lower levels in CDF1 RNAi lines, this suggests the existence of other CO transcriptional repressors. Additionally, in these CDF1 RNAi lines, the late-flowering phenotype of the fkf1 mutant cannot be fully restored by decreased levels of CDF1 expression, suggesting that FKF1 may act on these additional unknown repressors of CO in addition to CDF1 (Imaizumi *et. al.,* 2005). Similar to this, early flowering

is caused by over-expression of GI in the fkf1 mutant background, suggesting that GI impacts flowering through mechanisms other than FKF1 (Sawa *et. al.,* 2007). Given that the GI protein is present in both LDs and SDs (David *et. al.,* 2006), and that its abundance differs significantly from that of CO's expression profile, these additional elements must act to stop GI from activating CO at inconvenient times of the day. The RING finger protein RED AND FAR RED INSENSITIVE 2 (RFI2), which is known to largely suppress CO expression in LDs and is assumed to work in conjunction with GI, may be one such component (Chen and Ni, 2006).

The floral integrator genes FT, TSF, and SOC1 are induced by CO to stimulate blooming (Onouchi *et. al.,* 2000; Samach *et. al.,* 2000; Yamaguchi *et. al.,* 2005). Due to the high CO protein abundance at this time of day, FT and TSF are directly induced by CO and reach their highest levels of expression near the conclusion of an LD (Suàrez-López *et. al.,* 2001; Yanovsky and Kay, 2002; Yamaguchi *et. al.,* 2005). Because CO lacks a normal DNA-binding domain, it is likely that it must interact with additional proteins in order to bind to specific sequences in the FT promoter. The *Arabidopsis* orthologs of the mammalian HEME ACTIVATOR PROTEIN 3 (HAP3) and HAP5 as well as a tomato (*Solanum lycopersicum)*) CO homologue, tomato CO-LIKE 1 (TCOL1), have both been shown to interact with the tomato HAP5 protein in recent years (Ben-Naim *et. al.,* 2006). According to a theory put forth by Wenkel *et. al.,* 2006, CO can take the role of HAP2 in the HAP2/HAP3/HAP5 trimeric HAP complex, commonly known as nuclear factor Y (NF-Y) or the CCAAT box factor (CBF), which binds to CCAAT boxes in eukaryotic promoters. The TCOL1-HAP complex was demonstrated to bind to the CCAAT motifs of the yeast CYC1 and HEM1 promoters in tomato, proving that CO-like proteins can bind DNA via interacting with the HAP complex (Ben-Naim *et. al.,* 2006). In tobacco, the HAP complex has been demonstrated to bind CAAT motifs as well (Kusnetsov *et. al.,* 1999), and the FT promoter region contains a number of these motifs. The ability of the CO/HAP3/HAP5 complex to bind the FT promoter directly, however, has not yet been demonstrated.

The leaf experiences the duration of the day. CO is expressed in the vascular tissues of hypocotyls, cotyledons, and leaves as well as in the apex (Takada and Goto, 2003; An *et. al.,* 2004). However, while its expression from the phloem companion cell-specific sucrose transporter (SUC2) promoter was sufficient to complement the CO-2 mutation, it was not from meristem, epidermis, or root-specific promoters. These findings suggest that flowering induction by CO occurs specifically in the phloem. The expression of FT is also seen in the vascular tissue of cotyledons and in the apical region of leaves (but not in the basal regions or in the principal veins), and CO is a direct activator of FT. Flowering cannot be induced if FT expression in phloem companion cells is suppressed by synthetic miRNAs, as this expression is necessary for the stimulation of flowering (Mathieu *et. al.,* 2007). However, unlike CO, FT can induce blooming if expressed through a meristem-specific promoter or even an epidermis-specific promoter (An *et. al.,* 2004). FT gene expression was not seen in the SAM. Therefore, even though the CO protein's ability to produce FT appears to be limited to the phloem, FT can still have an impact if it exists in other plant tissues.

4. CO-INDEPENDENT PATHWAYS

There are routes that can control flower growth that are not dependent on the CO gene, even though the CO gene is a major regulator of flowering time in many plants.

The FLOWERING LOCUS T (FT) gene, which encodes a protein related to the florigen protein generated by the CO-dependent pathway, is one such pathway. The FT protein supports the change from vegetative growth to reproductive growth, which results in the creation of flowers, and is produced in leaves in response to environmental cues like photoperiod or temperature. It is subsequently transferred to the apical meristem. In many plant species, the timing of flowering is controlled by the FT pathway, which can be activated by both long-day and short-day conditions.

The SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1 (SOC1) gene, which encodes a transcription factor that promotes the expression of genes involved in flower development, is active in another CO-independent pathway. SOC1 controls the timing of flowering in *Arabidopsis* and other plant species. It is influenced by a number of environmental stimuli, such as light, temperature, and gibberellins.

CO- independent, other genes and pathways, including as the autonomous and vernalization pathways, can also control blooming time. While the vernalization process involves modulating the expression of the florigen genes to regulate flowering time, the autonomous pathway involves a group of genes that control the expression of the florigen genes.

In conclusion, whereas CO is a key regulator of flowering time in many plants, other genes and pathways can also influence flower development independently of CO, demonstrating the complexity and adaptability of the regulatory networks that govern plant growth.

Through a different mechanism without the use of CO, photoperiod is also able to control the timing of blooming. The AP2-like genes TARGET OF EAT 1 (TOE1), TOE2, TOE3, SCHLAFMUTZE (SMZ), and SCHNARCHZAPFEN (SNZ) are the target genes of miR172, which is regulated by GI and down-regulated post-transcriptionally. All of these genes, with the exception of TOE3, cause late flowering when overexpressed, proving that they are floral repressors (Aukerman and Sakai, 2003; Jung *et. al.,* 2007).

While CO expression is unaffected by miR172 over-expression in wild-type plants, FT expression is up-regulated (Jung *et. al.,* 2007), which results in unusually early flowering even in a co mutant background. Because TOE1 inhibits the expression of FT, miR172 causes flowering by reducing TOE1's repression of FT. Toe1, Toe2, SMZ, and SNZ (but not TOE3) transcript levels all decline with age in a complimentary manner, while miR172 expression increases with plant age until flowering (Jung *et. al.,* 2007). This age-related regulation may be explained by the fact that miR172 levels are partly regulated by genes connected to the autonomous pathway, including FCA, FLK, and FVE. The meristem's ability to respond to inducing signals during the juvenile phase may be inhibited by high levels of TOE1 in very young plants, but this is still unknown (as previously mentioned, miR172 is already known to regulate GL15, which affects juvenility in maize). MiR172 levels are significantly greater in plants grown in LDs than in plants cultivated in SDs, which suggests that miR172 plays a role in the promotion of flowering when LDs are induced. MiR172 levels are enhanced in blue light but lowered in red light. Since miR172 levels don't fluctuate in a rhythmic manner, GI controls miR172 abundance in a clock-independent manner. Accordingly, GI has a dual role in the photoperiodic regulation of flowering by controlling the CO- and miR172-mediated induction of FT expression (Jung *et. al.,* 2007). Both of these routes are necessary for the promotion of blooming in LDs because the disruption of any one causes late blossoming in LDs.

IV. Systemic indicators

There are systemic signals that can affect flower growth across the plant in addition to the local signals that control floral development at the location of the bloom buds.

The plant hormone auxin, which is involved in a variety of plant growth and development processes, including flower development, is one such systemic signal. Polar auxin transfer is a method by which auxin can be moved from its location of synthesis in the shoot apex to other areas of the plant. Auxin can operate as a systemic signal and control flower growth throughout the plant thanks to this transport mechanism.

The plant hormone gibberellin, which encourages the growth and elongation of stems and is also involved in the control of flowering time, is another systemic signal involved in flower formation. Gibberellins are able to be produced in the shoot apex and transmitted throughout the entire plant, where they might affect the growth of flowers in various plant regions.

Additionally, systemic stress signals like salinity, pathogen infection, or drought might have an impact on floral growth. These signals can change gene expression patterns or hormone signalling pathways, which can change how flowers develop or even prevent flowers from forming.

Systemic signals are crucial in helping the plant respond to both internal and external inputs and maximise its reproductive success in a changing environment. They also play a key role in coordinating flower development across the entire plant.

The existence of a graft-transmissible flower-inducing signal that travelled from induced leaves through the phloem to the apex was amply demonstrated by traditional grafting studies (reviewed in Thomas and Vince-Prue, 1997). *Brassica napus* and *Cucurbita maxima* phloem have been found to contain the FT protein by mass spectrometry (Giavalisco *et. al.,* 2006; Lin *et. al.,* 2007).

Given that FT is expressed in phloem companion cells and that it has been demonstrated that proteins up to 67 kDa from companion cells can be loaded non-selectively into the phloem sieve elements, there would be no restriction to the entry of the small 20-kDa FT protein into the phloem. However, numerous recent publications have demonstrated that the FT protein can move intracellularly from the end of the vasculature into the SAM in a number of species, including rice (*Oryza sativa*) and *Arabidopsis*, and even across graft unions (Corbesier *et. al.,* 2007; Jaeger and Wigge, 2007; Mathieu *et. al.,* 2007; Tamaki *et. al.,* 2007).

Jaeger and Wigge (2007) and Mathieu et al. (2007) made a strong case that the FT protein (and its paralogues, such as TSF; Mathieu et al. (2007) is a component of the mobile flower-inducing signal by using elegant experiments that dissociated the effects of the FT protein from those of FT mRNA. In order to regulate developmental processes like tuberization and leaf growth, mRNAs can also flow through the phloem throughout the plant (Kim *et. al.,* 2001; Haywood *et. al.,* (2005); Banerjee *et. al.,* (2006). Tamaki et al. (2007) did report the identification of modest quantities of mRNA of the rice FT orthologue Heading date 3a (Hd3a) in rice shoot apices despite the fact that it is not expressed there. However, transport of the FT mRNA across a graft union or into the SAM was not identified in many of the tests mentioned above. Therefore, it may still be up for contention whether or not FT mRNA moves and what it may do. Similar to this, it is unknown how tiny RNA molecules contribute to the propagation of the induced state across the plant.

MiR156 has been identified in phloem sap and has been shown to influence the floral transition by regulating SBP-box genes (Yoo *et. al.,* 2004; Wu and Poethig, 2006; Schwarz *et. al.,* 2008). However, transport of gene-silencing RNAs into the apex may be inhibited by the RNA surveillance system present at the SAM (Foster *et. al.,* 2002).

The FT protein activates the meristem identity gene AP1 when it reaches the apex by interacting with the bZIP transcription factor FLOWERING LOCUS D (FD) (Abe *et. al.,* 2005; Wigge *et. al.,* 2005).

Indicating that FT does not function through FD alone, mutations in FD do not entirely suppress the early flowering phenotype of FT over-expressing plants (Abe *et. al.,* 2005; Wigge *et. al.,* 2005). According to Yoo et al. (2005), FT is also known to increase SOC1 expression in the SAM. SOC1 joins forces with AGAMOUS-LIKE 24 (AGL24), another MADS box protein, to translocate to the nucleus where it binds to the LFY promoter to activate LFY expression (Lee *et. al.,* 2008). LFY stimulates the expression of AP1, and the other way around.

From the leaves to the apex, other substances that influence flowering are also delivered. These substances include hormones like gibberellins and cytokinins as well as metabolites like glutamine, sucrose, and nitrate, some of which have the potential to affect the pace of cell division at the SAM (Bernier and Perilleux, 2005). It is hypothesised that photoperiodic induction in Sinapis and Xanthium causes an increase in sucrose and cytokinin export from the leaf, which causes an increase in hexoses at the SAM and causes the observed increase in cell division at the SAM in Sinapis (Gonthier *et. al.,* 1987; Bernier and Perilleux, 2005). It has been challenging to determine whether these compounds are a component of the inducing signal per se or whether their transport to the apex is an early event following induction. Increasing cell division in the SAM has been shown to cause early flowering in tobacco (Cockcroft *et. al.,* 2000). The second possibility is supported by the finding that tobacco callus obtained from induced plants could form flowers when grown on media enriched with glucose, but callus derived from noninduced plants could not (Chailkhyan *et. al.,* 1975).

5. CONCLUSION AND FUTURE RESEARCH PERSPECTIVES

Plants can synchronise their developmental schedule with the prevailing season by using the photoperiod information. It is used to match the ideal circumstances for the birth of offspring and to lessen the dangers of seasonal pressures that occur at the same time each year. We have outlined the intricate photoperiod sensing systems in this review, with a particular emphasis on the function of the photoperiod in plant responses to cold, drought, osmotic, and biotic stressors. While the molecular mechanisms governing how cold, drought, and osmotic stress are regulated by photoperiod are at least somewhat understood, the effect of photoperiod on biotic stress responses is still only descriptive. Photoperiod stress signals might be useful for adaptation, for instance by serving as a priming agent that makes plants more resilient to subsequent stresses. It is necessary to reveal the

ecological significance of photoperiod stress. The output of the photoperiod sensing system has been hypothesised to be modulated by variations in intensity and ratios of wavelengths at dawn and dusk that rely on weather conditions, however there is no experimental evidence to support this claim. The basic processes for light detection, timekeeping, and integrating these exogenous and endogenous signals are necessary for plants to respond to photoperiod. From a growing number of plant species, genes that are similar to many of the *Arabidopsis* genes that are known to control flowering through photoperiodic regulation have been identified. Many of these genes have been proved to be real orthologues because they perform the same function. However, there is evidence that RNA can also operate as a signal, as is the case with potato tuberization, therefore this may not be true for all photoperiodic responses in all species. It has also been demonstrated that miRNAs have a role in the regulation of blooming. One such miRNA, miR156, has been found to exist in the phloem and is probably mobile there. Since different species have obviously developed distinct mechanisms to respond to photoperiod, there is still considerable research to be done to understand the photoperiodic regulatory mechanisms in species other than *Arabidopsis*.

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.

Abuelsoud, W., Cortleven, A., and Schmülling, T. (2020). Photoperiod stress induces an oxidative burst-like response and is associated with increased apoplastic peroxidase and decreased catalase activities. J. Plant Physiol. 253:153252. doi: 10.1016/j.jplph.2020.153252

Achard P, Herr A, Baulcombe DC, Harberd NP. (2004). Modulation of floral development by a gibberellin-regulated microRNA. ***Development*** **131**: 3357– 3365.

Adams SR, Pearson S, Hadley P, Patefield WM. 1999). The effects of temperature and light integral on the phases of photoperiod sensitivity in *Petunia×hybrida*. ***Annals of Botany*** **83**: 263– 269.

Adams SR, Pearson S, Hadley P. (2001). Improving quantitative flowering models through a better understanding of the phases of photoperiod sensitivity. ***Journal of Experimental Botany*** **52**: 655– 662.

Adole, T., Dash, J., Rodriguez-Galiano, V., and Atkinson, P. M. (2019). Photoperiod controls vegetation phenology across Africa. ***Commun. Biol.*** 2:391. doi: 10.1038/s4(2003)-019-0636-7

Alabadi, D., Oyama, T., Yanovsky, M. J., Harmon, F. G., Mas, P., and Kay, S. A. (2001). Reciprocal regulation between TOC1 and LHY/CCA1 within the *Arabidopsis* circadian clock. ***Science*** 293, 880–883. doi: 10.1126/science.1061320

Alonso-Blanco, C., Gomez-Mena, C., Llorente, F., Koornneef, M., Salinas, J., and Martinez-Zapater, J. M. (2005). Genetic and molecular analyses of natural variation indicate CBF2 as a candidate gene for underlying a freezing tolerance quantitative trait locus in *Arabidopsis*. ***Plant Physiol.*** 139, 1304–1312. doi: 10.1104/pp.105.068510

An H, Roussot C, Suárez-López P, Corbesier L, Vincent C, Piñeiro M, Hepworth S, Mouradov A, Justin S, Turnbull C *et al* . (2004). CONSTANS acts in the phloem to regulate a systemic signal that induces photoperiodic flowering of *Arabidopsis*. ***Development*** **131**: 3615– 3626.

Anwer, M. U., Davis, A., Davis, S. J., and Quint, M. (2020). Photoperiod sensing of the circadian clock is controlled by EARLY FLOWERING 3 and GIGANTEA. ***Plant J.*** 101, 1397–1410. doi: 10.1111/tpj.14604

Asai K, Satoh N, Sasaki H, Satoh H, Nagato Y. (2002). A rice heterochronic mutant, mori1, is defective in the juvenile-adult phase change. *Development* **129**: 265– 273.

Aukerman, M. J., and Sakai, H. (2003). Regulation of flowering time and floral organ identity by a microRNA and its APETALA2-like target genes. ***Plant Cell*** 15, 2730–2741. doi: 10.1105/tpc.016238

Baek, D., Kim, W. Y., Cha, J. Y., Park, H. J., Shin, G., Park, J., *et. al.,* (2020). The GIGANTEA-ENHANCED EM LEVEL complex enhances drought tolerance via regulation of abscisic acid synthesis. ***Plant Physiol***. 184, 443–458. doi: 10.1104/pp.20.00779

Baerenfaller, K., Massonnet, C., Hennig, L., Russenberger, D., Sulpice, R., Walsh, S., *et. al.,* (2015). A long photoperiod relaxes energy management in *Arabidopsis* leaf six. **Curr. Plant Biol.** 2, 34–45. doi: 10.1016/j.cpb.2015.07.001

Balasubramanian S, Sureshkumar S, Lempe J, Weigel D. (2006). Potent induction of *Arabidopsis thaliana* flowering by elevated growth temperature. ***PLoS Genetics* 2**: 0980– 0989.

Banerjee AK, Chatterjee M, Yu Y, Suh SG, Miller WA, Hannapel DJ. (2006). Dynamics of a mobile RNA of potato involved in a long-distance signaling pathway. ***Plant Cell* 18**: 3443– 3457.

Batutis EJ, Ewing EE. (1982). Far-red reversal of red-light effect during long-night induction of potato (*Solanum tuberosum* L.) tuberization. ***Plant Physiology* 69**: 672– 674.

Bäurle I, Dean C. (2006). The timing of developmental transitions in plants. ***Cell* 125**: 655– 664.

Beales J, Turner A, Griffiths S, Snape JW, Laurie DA. (2007). A pseudo-response regulator is misexpressed in the photoperiod insensitive Ppd-D1a mutant of wheat (*Triticum aestivum* L.). ***Theoretical and Applied Genetics* 115**: 721– 733.

Becker, B., Holtgrefe, S., Jung, S., Wunrau, C., Kandlbinder, A., Baier, M., *et. al.,* (2006). Influence of the photoperiod on redox regulation and stress responses in *Arabidopsis thaliana* L. (Heynh.) plants under long- and short-day conditions. ***Planta*** 224, 380–393. doi: 10.1007/s00425-006-0222-3

Ben-Naim O, Eshed R, Parnis A, Teper-Bamnolker P, Shalit A, Coupland G, Samach A, Lifschitz E. (2006). The CCAAT binding factor can mediate interactions between CONSTANS-like proteins and DNA. ***Plant Journal* 46**: 462– 476.

Berardini TZ, Bollman K, Sun H, Poethig RS. (2001). Regulation of vegetative phase change in *Arabidopsis thaliana* by cyclophilin 40. ***Science* 291**: 2405– 2407.

Bernier G, Perilleux C. (2005). A physiological overview of the genetics of flowering time control. ***Plant Biotechnology Journal* 3**: 3– 16.

Blázquez MA, Ahn JH, Weigel D. (2003). A thermosensory pathway controlling flowering time in *Arabidopsis thaliana*. ***Nature Genetics* 33**: 168– 171.

Blazquez MA, Soowal LN, Lee I, Weigel D. (1997). LEAFY expression and flower initiation in *Arabidopsis*. ***Development* 124**: 3835– 3844.

Blázquez MA, Weigel D. (20000. Integration of floral inductive signals in *Arabidopsis*. ***Nature* 404**: 889– 892.

Böhlenius H, Huang T, Charbonnel-Campaa L, Brunner AM, Jansson S, Strauss SH, Nilsson O. (2006). CO/FT regulatory module controls timing of flowering and seasonal growth cessation in trees. ***Science* 312**: 1040– 1043.

Bollman KM, Aukerman MJ, Park MY, Hunter C, Berardini TZ, Poethig RS. (2003). HASTY, the *Arabidopsis* ortholog of exportin 5/MSN5, regulates phase change and morphogenesis. ***Development* 130**: 1493– 1504.

Bongard-Pierce DK, Evans MMS, Poethig RS. (19960. Heteroblastic features of leaf anatomy in maize and their genetic regulation. ***International Journal of Plant Sciences* 157**: 331– 340.

Borchert R, Renner SS, Calle Z, Navarrete D, Tye A, Gautier L, Spichiger R, Von Hildebrand P. (2005). Photoperiodic induction of synchronous flowering near the Equator. ***Nature* 433**: 627– 629.

Bouveret R, Schönrock N, Gruissem W, Hennig L. (2006). Regulation of flowering time by *Arabidopsis* MSI1. ***Development* 133**: 1693– 1702.

Cagnola, J. I., Cerdan, P. D., Pacin, M., Andrade, A., Rodriguez, V., Zurbriggen, M. D., *et. al.,* (2018). Long-day photoperiod enhances jasmonic acid-related plant defense. ***Plant Physiol.*** 178, 163–173. doi: 10.1104/pp.18.00443

Cao, S., Jiang, S., and Zhang, R. (2006). The role of GIGANTEA gene in mediating the oxidative stress response in *Arabidopsis*. ***Plant Growth Regul.*** 48, 261–270. doi: 10.1007/s10725-006-0012-8

Cao, S., Ye, M., and Jiang, S. (2005). Involvement of GIGANTEA gene in the regulation of the cold stress response in *Arabidopsis*. ***Plant Cell Rep***. 24, 683–690. doi: 10.1007/s00299-005-0061-x

Carré, I. A. (2001). Day-length perception and the photoperiodic regulation of flowering in *Arabidopsis*. ***J. Biol. Rhythms*** 16, 415–423. doi: 10.1177/07487300112900(2006)

Casal, J. J., Fankhauser, C., Coupland, G., and Blazquez, M. A. (2004). Signalling for developmental plasticity. ***Trends Plant Sci.*** 9, 309–314. doi: 10.1016/j.tplants.(2004).04.007

Cecchini, E., Geri, C., Love, A. J., Coupland, G., Covey, S. N., and Milner, J. J. (2002). Mutations that delay flowering in *Arabidopsis* de-couple symptom response from cauliflower mosaic virus accumulation during infection. ***Mol. Plant Pathol.*** 3, 81–90. doi: 10.1046/j.1464-6722.2001.00097.x

Cejudo, F. J., Sandalio, L. M., and Van Breusegem, F. (2021). Understanding plant responses to stress conditions: redox-based strategies. ***J. Exp. Bot.*** 72, 5785–5788. doi: 10.1093/jxb/erab324

Cerdán PD, Chory J. (2003). Regulation of flowering time by light quality. ***Nature* 423**: 881– 885.

Cha, J. Y., Kim, J., Kim, T. S., Zeng, Q., Wang, L., Lee, S. Y., *et. al.,* (2017). GIGANTEA is a co-chaperone which facilitates maturation of ZEITLUPE in the *Arabidopsis* circadian clock. ***Nat. Commun.*** 8:3. doi: 10.1038/s41467-016-0014-9

Chae E, Tan QK, Hill TA, Irish VF. (2008). An *Arabidopsis* F-box protein acts as a transcriptional co-factor to regulate floral development. ***Development* 135**: 1235– 1245.

Chailkhyan MK, Aksdenova NP, Konstantinova TN, Bavrina TL. (19750. The callus model of plant flowering. ***Proceedings of the Royal Society of London,*** *Series B* **190**: 333– 340.

Chaouch, S., Queval, G., Vanderauwera, S., Mhamdi, A., Vandorpe, M., Langlois-Meurinne, M., *et. al.,* (2010). Peroxisomal hydrogen peroxide is coupled to biotic defense responses by ISOCHORISMATE SYNTHASE1 in a daylength-related manner. ***Plant Physiol.*** 153, 1692–1705. doi: 10.1104/pp.110.153957

Chen H, Banerjee AK, Hannapel DJ. (2004). The tandem complex of BEL and KNOX partners is required for transcriptional repression of ga20ox1. ***Plant Journal* 38**: 276– 284.

Chen M, Ni M. (2006). RFI2, a RING-domain zinc finger protein, negatively regulates CONSTANS expression and photoperiodic flowering. ***Plant Journal* 46**: 823– 833.

Chen, M., Zhao, Y., Zhuo, C., Lu, S., and Guo, Z. (2015). Overexpression of a NF-YC transcription factor from bermudagrass confers tolerance to drought and salinity in transgenic rice. ***Plant Biotechnol. J***. 13, 482–491. doi: 10.1111/pbi.12270

Chien JC, Sussex IM. (19960. Differential regulation of trichome formation on the adaxial and abaxial leaf surfaces by gibberellins and photoperiod in *Arabidopsis thaliana* (L.) Heynh***. Plant Physiology* 111**: 1321– 1328.

Chincinska IA, Liesche J, Krugel U, Michalska J, Geigenberger P, Grimm B, Kuhn C. (2008). Sucrose transporter StSUT4 from potato affects flowering, tuberization, and shade avoidance response. ***Plant Physiology* 146**: 515– 528.

Clarke JH, Tack D, Findlay K, Van Montagu M, Van Lijsebettens M. (1999). The SERRATE locus controls the formation of the early juvenile leaves and phase length in *Arabidopsis*. ***Plant Journal* 20**: 493– 501.

Cockcroft CE, Den Boer BG, Healy JM, Murray JA. (2000). Cyclin D control of growth rate in plants*. **Nature* **405**: 575– 579.

Conrath, U., Beckers, G. J., Flors, V., Garcia-Agustin, P., Jakab, G., Mauch, F., *et. al.,* (2006). Priming: getting ready for battle. ***Mol. Plant. Microbe Interact.*** 19, 1062–1071. doi: 10.1094/MPMI-19-1062

Corbesier L, Vincent C, Jang S, Fornara F, Fan Q, Searle I, Giakountis A, Farrona S, Gissot L, Turnbull C. *et al* . (2007). FT protein movement contributes to long-distance signaling in floral induction of *Arabidopsis*. ***Science* 316**: 1030– 1033.

Cortleven, A., Roeber, V. M., Frank, M., Bertels, J., Lortzing, V., Beemster, G., *et. al.,* (2021). The transcriptomic landscape of the photoperiodic stress response in *Arabidopsis thaliana* resembles the response to pathogen infection. bioRxiv [Preprint]. doi: 10.1101/2021.04.13.439491

David KM, Armbruster U, Tama N, Putterill J. (2006). *Arabidopsis* GIGANTEA protein is post-transcriptionally regulated by light and dark. ***FEBS Letters* 580**: 1193– 1197.

de los Reyes, P., Romero-Losada, A. B., and Romero-Campero, F. J. (2020). ATTRACTOR, *Arabidopsis thaliana* Transcriptional Circadian Network v1.0. Zenodo. doi: 10.5381/zenodo.3780022

Devlin PF, Kay SA. (2000). Cryptochromes are required for phytochrome signaling to the circadian clock but not for rhythmicity. ***Plant Cell* 12**: 2499– 2509.

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.

Dong, C. H., Agarwal, M., Zhang, Y., Xie, Q., and Zhu, J. K. (2006). The negative regulator of plant cold responses, HOS1, is a RING E3 ligase that mediates the ubiquitination and degradation of ICE1. ***Proc. Natl. Acad. Sci.*** U.S.A. 103, 8281–8286. doi: 10.1073/pnas.0602874103

Dudley M, Poethig RS. (1993). The heterochronic teopod1 and teopod2 mutations of maize are expressed non-cell-autonomously. ***Genetics* 133**: 389– 399.

Duncan MJ. (2007). Circannual prolactin rhythms: calendar-like timer revealed in the pituitary gland. ***Trends in Endocrinology and Metabolism* 18**: 259– 260.

Evrard, A., Ndatimana, T., and Eulgem, T. (2009). FORCA, a promoter element that responds to crosstalk between defense and light signaling. ***BMC Plant Biol.*** 9:2. doi: 10.1186/1471-2229-9-2

Fahlgren N, Montgomery TA, Howell MD, Allen E, Dvorak SK, Alexander AL, Carrington JC. (2006). Regulation of AUXIN RESPONSE FACTOR3 by TAS3 ta-siRNA affects developmental timing and patterning in *Arabidopsis*. ***Current Biology* 16**: 939– 944.

Farré EM, Harmer SL, Harmon FG, Yanovsky MJ, Kay SA. (2005). Overlapping and distinct roles of PRR7 and PRR9 in the *Arabidopsis* circadian clock. ***Current Biology* 15**: 47– 54.

Feng, P., Guo, H., Chi, W., Chai, X., Sun, X., Xu, X., *et. al.,* (2016). Chloroplast retrograde signal regulates flowering. ***Proc. Natl. Acad. Sci.*** U.S.A. 113, 10708–10713. doi: 10.1073/pnas.1521599113

Fornara, F., de Montaigu, A., Sanchez-Villarreal, A., Takahashi, Y., Ver Loren van Themaat, E., Huettel, B., *et. al.,* (2015). The GI-CDF module of *Arabidopsis* affects freezing tolerance and growth as well as flowering. ***Plant J.*** 81, 695–706. doi: 10.1111/tpj.12759

Foster TM, Lough TJ, Emerson SJ, Lee RH, Bowman JL, Forster RL, Lucas WJ. (2002). A surveillance system regulates selective entry of RNA into the shoot apex. ***Plant Cell* 14**: 1497– 1508.

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.

Fowler, S., and Thomashow, M. F. (2002). *Arabidopsis* transcriptome profiling indicates that multiple regulatory pathways are activated during cold acclimation in addition to the CBF cold response pathway. ***Plant Cell*** 14, 1675–1690. doi: 10.1105/tpc.003483

Fowler, S., Lee, K., Onouchi, H., Samach, A., Richardson, K., Morris, B., *et. al.,* (1999). GIGANTEA: a circadian clock-controlled gene that regulates photoperiodic flowering in *Arabidopsis* and encodes a protein with several possible membrane-spanning domains. ***EMBO J.*** 18, 4679–4688. doi: 10.1093/emboj/18.17.4679

Frank, M., Cortleven, A., Novak, O., and Schmülling, T. (2020). Root-derived trans-zeatin cytokinin protects *Arabidopsis* plants against photoperiod stress. ***Plant Cell Environ.*** 43, 2637–2649. doi: 10.1111/pce.13860

Franks, S. J. (2011). Plasticity and evolution in drought avoidance and escape in the annual plant *Brassica rapa*. ***New Phytol.*** 190, 249–257. doi: 10.1111/j.1469-8137.2010.03603.x

Galbiati, F., Chiozzotto, R., Locatelli, F., Spada, A., Genga, A., and Fornara, F. (2016). Hd3a, RFT1 and Ehd1 integrate photoperiodic and drought stress signals to delay the floral transition in rice. ***Plant Cell Environ.*** 39, 1982–1993. doi: 10.1111/pce.12760

Gallé, A., Czekus, Z., Toth, L., Galgoczy, L., and Poor, P. (2021). Pest and disease management by red light. ***Plant Cell Environ.*** 44, 3197–3210. doi: 10.1111/pce.14142

Gandikota M, Birkenbihl RP, Hohmann S, Cardon GH, Saedler H, Huijser P.(2007). The miRNA156/157 recognition element in the 3′ UTR of the *Arabidopsis* SBP box gene SPL3 prevents early flowering by translational inhibition in seedlings. ***Plant Journal* 49**: 683– 693.

Gao, X., Liu, H., Zhao, H., Sun, Y., Li, X., & Li, C. (2021). Advances in molecular mechanisms of photoperiodic flowering in rice. ***Frontiers in Plant Science,*** 12, 653994. https://doi.org/10.3389/fpls.2021.653994

Gardner MJ, Hubbard KE, Hotta CT, Dodd AN, Webb AAR. (2006). How plants tell the time. ***Biochemical Journal* 397**: 15– 24.

Gendron, J. M., Pruneda-Paz, J. L., Doherty, C. J., Gross, A. M., Kang, S. E., and Kay, S. A. (2012). *Arabidopsis* circadian clock protein, TOC1, is a DNA-binding transcription factor. *Proc. Natl. Acad. Sci.* U.S.A. 109, 3167–3172. doi: 10.1073/pnas.1(2003)55109

Giavalisco P, Kapitza K, Kolasa A, Buhtz A, Kehr J. (2006). Towards the proteome of *Brassica napus* phloem sap. ***Proteomics* 6**: 896– 909.

Gol, L., Haraldsson, E. B., and von Korff, M. (2021). Ppd-H1 integrates drought stress signals to control spike development and flowering time in barley. ***J. Exp. Bot.*** 72, 122–136. doi: 10.1093/jxb/eraa261

Goldman BD. (2001). Mammalian photoperiodic system: formal properties and neuroendocrine mechanisms of photoperiodic time measurement. ***Journal of Biological Rhythms* 16**: 283–301.

Gonthier R, Jacqmard A, Bernier G. (1987). Changes in cell-cycle duration and growth fraction in the shoot meristem of *Sinapis* during floral transition. ***Planta* 170**: 55– 59.

Griebel, T., and Zeier, J. (2008). Light regulation and daytime dependency of inducible plant defenses in *Arabidopsis*: phytochrome signaling controls systemic acquired resistance rather than local defense. ***Plant Physiol.*** 147, 790–801. doi: 10.1104/pp.108.119503

Grigg SP, Canales C, Hay A, Tsiantis M. (2005). SERRATE coordinates shoot meristem function and leaf axial patterning in *Arabidopsis*. *Nature* **437**: 1022– 1026.

Grundy, J., Stoker, C., and Carré, I. A. (2015). Circadian regulation of abiotic stress tolerance in plants. ***Front. Plant Sci.*** 6:648. doi: 10.3389/fpls.2015.00648

Hackett WP. (1985). Juvenility, maturation and rejuvenation in woody plants. ***Horticultural Reviews* 7**: 109– 155.

Halliday KJ, Salter MG, Thingnaes E, Whitelam GC. (2003). Phytochrome control of flowering is temperature sensitive and correlates with expression of the floral integrator *FT*. ***Plant Journal* 33**: 875– 885.

Han, X., Tang, S., An, Y., Zheng, D. C., Xia, X. L., and Yin, W. L. (2013b). Overexpression of the poplar NF-YB7 transcription factor confers drought tolerance and improves water-use efficiency in *Arabidopsis*. ***J. Exp. Bot.*** 64, 4589–4601. doi: 10.1093/jxb/ert262

Han, Y., Zhang, X., Wang, W., Wang, Y., and Ming, F. (2013a). The suppression of WRKY44 by GIGANTEA-miR172 pathway is involved in drought response of *Arabidopsis thaliana*. ***PLoS One 8***:e73541. doi: 10.1371/journal.pone.0073541

Hayama R, Agashe B, Luley E, King R, Coupland G. (2007). A circadian rhythm set by dusk determines the expression of FT homologs and the short-day photoperiodic flowering response in *Pharbitis*. ***Plant Cell* 19**: 2988– 3000.

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.

Haywood V, Yu TS, Huang NC, Lucas WJ. (2005). Phloem long-distance trafficking of GIBBERELLIC ACID-INSENSITIVE RNA regulates leaf development. ***Plant Jounal* 42**: 49– 68.

He Y, Tang RH, Hao Y, Stevens RD, Cook CW, Ahn SM, Jing L, Yang Z, Chen L, Guo F, *et. al.,* (2004). Nitric oxide represses the *Arabidopsis* floral transition. ***Science* 305**: 1968– 1971.

He YH, Amasino RM. (2005). Role of chromatin modification in flowering-time control. ***Trends in Plant Science* 10**: 30– 35.

Hendel-Rahmanim, K., Masci, T., Vainstein, A., and Weiss, D. (2007). Diurnal regulation of scent emission in rose flowers. ***Planta*** 226, 1491–1499. doi: 10.1007/s00425-007-0582-3

Henderson IR, Shindo C, Dean C. (2003). The need for winter in the switch to flowering. ***Annual Review of Genetics* 37**: 371– 392.

Hertwig, B., Streb, P., and Feierabend, J. (1992). Light dependence of catalase synthesis and degradation in leaves and the influence of interfering stress conditions. ***Plant Physiol.*** 100, 1547–1553. doi: 10.1104/pp.100.3.1547

Heyer AG, Mozley D, Landschutze V, Thomas B, Gatz C. (1995). Function of phytochrome A in potato plants as revealed through the study of transgenic plants. ***Plant Physiology* 109**: 53– 61.

Holm M, Hardtke CS, Gaudet R, Deng XW. (2001). Identification of a structural motif that confers specific interaction with the WD40 repeat domain of *Arabidopsis* COP1. ***EMBO Journal* 20**: 118– 127.

Hotta CT, Gardner MJ, Hubbard KE, Baek SJ, Dalchau N, Suhita D, Dodd AN, Webb AAR. (2007). Modulation of environmental responses of plants by circadian clocks. ***Plant, Cell and Environment* 30**: 333– 349.

Hsu CY, Liu Y, Luthe DS, Yuceer C. (2006). Poplar FT2 shortens the juvenile phase and promotes seasonal flowering. ***Plant Cell* 18**: 1846– 1861.

Hsu, P. Y., and Harmer, S. L. (2014). Global profiling of the circadian transcriptome using microarrays. ***Methods Mol. Biol.*** 1158, 45–56. doi: 10.1007/978-1-4939-0700-7_3

Hunter C, Sun H, Poethig RS. (2003). The *Arabidopsis* heterochronic gene ZIPPY is an ARGONAUTE family member. ***Current Biology* 13**: 1734– 1739.

Hunter C, Willmann MR, Wu G, Yoshikawa M, De La Luz Gutierrez-Nava M, Poethig SR. (2006). Trans-acting siRNA-mediated repression of ETTIN and ARF4 regulates heteroblasty in *Arabidopsis*. ***Development* 133**: 2973– 2981.

Hwang, K., Susila, H., Nasim, Z., Jung, J. Y., and Ahn, J. H. (2019). *Arabidopsis* ABF3 and ABF4 transcription factors act with the NF-YC complex to regulate SOC1 expression and mediate drought-accelerated flowering. ***Mol. Plant*** 2, 489–505. doi: 10.1016/j.molp.2019.01.002

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.

Imaizumi T, Tran HG, Swartz TE, Briggs WR, Kay SA. (2003). FKF1 is essential for photoperiodic-specific light signalling in *Arabidopsis*. ***Nature*** **426**: 302– 306.

Ishitani, M., Xiong, L., Lee, H., Stevenson, B., and Zhu, J. K. (1998). HOS1, a genetic locus involved in cold-responsive gene expression in *Arabidopsis*. ***Plant Cell*** 10, 1151–1161. doi: 10.1105/tpc.10.7.1151

Iuchi, S., Kobayashi, M., Taji, T., Naramoto, M., Seki, M., Kato, T., *et. al.,* (2001). Regulation of drought tolerance by gene manipulation of 9-cis-epoxycarotenoid dioxygenase, a key enzyme in abscisic acid biosynthesis in *Arabidopsis*. ***Plant J.*** 27, 325–333. doi: 10.1046/j.1365-313x.2001.01096.x

Izawa T, Oikawa T, Sugiyama N, Tanisaka T, Yano M, Shimamoto K. (2002). Phytochrome mediates the external light signal to repress FT orthologs in photoperiodic flowering of rice. ***Genes and Development*** **16**: 2006– 2020.

Jackson SD, Heyer A, Dietze J, Prat S. (1996). Phytochrome B mediates the photoperiodic control of tuber formation in potato. ***Plant Journal*** **9**: 159– 166.

Jackson SD, James PE, Carrera E, Prat S, Thomas B. (2000). Regulation of transcript levels of a potato gibberellin 20-oxidase gene by light and phytochrome B. **Plant Physiology 124**: 423–430.

Jackson, S. D. (2009). Plant responses to photoperiod. ***New Phytol.*** 181, 517–531. doi: 10.1111/j.1469-8137.2008.02681.x

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.

Jung, J. H., Seo, Y. H., Seo, P. J., Reyes, J. L., Yun, J., Chua, N. H., *et. al.,* (2007). The GIGANTEA-regulated microRNA172 mediates photoperiodic flowering independent of CONSTANS in *Arabidopsis*. ***Plant Cell*** 19, 2736–2748. doi: 10.1105/tpc.107.054528

Kanrar S, Bhattacharya M, Arthur B, Courtier J, Smith HM. (2008). Regulatory networks that function to specify flower meristems require the function of homeobox genes PENNYWISE and POUND-FOOLISH in *Arabidopsis*. ***Plant Journal*** **54**: 924– 937.

Karlson, D. T., Fujino, T., Kimura, S., Baba, K., Itoh, T., and Ashworth, E. N. (2003). Novel plasmodesmata association of dehydrin-like proteins in cold- acclimated Red-osier dogwood (*Cornus sericea*). ***Tree Physiol.*** 23, 759–767. doi: 10.1093/treephys/23.11.759

Kelly AJ, Bonnlander MB, Meeks-Wagner DR. (1995). NFL, the tobacco homolog of FLORICAULA and LEAFY, is transcriptionally expressed in both vegetative and floral meristems. ***Plant Cell*** **7**: 225– 234.

Kenyon, D. M., Dixon, G. R., and Helfer, S. (2002). Effects of relative humidity, light intensity and photoperiod on the colony development of Erysiphe sp. on Rhododendron. ***Plant Pathol***. 51, 103–108. doi: 10.1046/j.0032-0862.2001.x

Kiba, T., Henriques, R., Sakakibara, H., and Chua, N. H. (2007). Targeted degradation of PSEUDO-RESPONSE REGULATOR5 by an SCFZTL complex regulates clock function and photomorphogenesis in *Arabidopsis thaliana*. ***Plant Cell*** 19, 2516–2530. doi: 10.1105/tpc.107.053033

Kim JY, Song HR, Taylor BL, Carre IA. (2003). Light-regulated translation mediates gated induction of the *Arabidopsis* clock protein LHY. ***EMBO Journal* 22**: 935– 944.

Kim M, Canio W, Kessler S, Sinha N. (20010. Developmental changes due to long-distance movement of a homeobox fusion transcript in tomato. ***Science* 293**: 287– 289.

Kim SL, Lee S, Kim HJ, Nam HG, An G. (2007a). OsMADS51 is a short-day flowering promoter that functions upstream of Ehd1, OsMADS14, and Hd3a. ***Plant Physiology* 145**: 1484– 1494.

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

Kim, W. Y., Ali, Z., Park, H. J., Park, S. J., Cha, J. Y., Perez-Hormaeche, J., *et. al.,* (2013). Release of SOS2 kinase from sequestration with GIGANTEA determines salt tolerance in *Arabidopsis*. ***Nat. Commun.*** 4:1352. doi: 10.1038/ncomms2357

Kim, W. Y., Fujiwara, S., Suh, S. S., Kim, J., Kim, Y., Han, L., *et. al.,* (2007). ZEITLUPE is a circadian photoreceptor stabilized by GIGANTEA in blue light. ***Nature*** 449, 356–360. doi: 10.1038/nature06132

Kojima S, Takahashi Y, Kobayashi Y, Monna L, Sasaki T, Araki T, Yano M. (2002). Hd3a, a rice ortholog of the *Arabidopsis* FT gene, promotes transition to flowering downstream of Hd1 under short-day conditions. ***Plant and Cell Physiology* 43**: 1096– 1105.

Komiya R, Ikegami A, Tamaki S, Yokoi S, Shimamoto K. (2008). Hd3a and RFT1 are essential for flowering in rice. ***Development* 135**: 767– 774.

Kotake T, Takada S, Nakahigashi K, Ohto M, Goto K. (2003). *Arabidopsis* TERMINAL FLOWER 2 gene encodes a heterochromatin protein 1 homolog and represses both FLOWERING LOCUS T to regulate flowering time and several floral homeotic genes. **Plant and Cell Physiology 44**: 555– 564.

Krasensky-Wrzaczek, J., and Kangasjarvi, J. (2018). The role of reactive oxygen species in the integration of temperature and light signals. ***J. Exp. Bot.*** 69, 3347–3358. doi: 10.1093/jxb/ery074

Kumimoto, R. W., Adam, L., Hymus, G. J., Repetti, P. P., Reuber, T. L., Marion, C. M., *et. al.,* (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. doi: 10.1007/s00425-008-0773-6

Kumimoto, R. W., Zhang, Y., Siefers, N., and Holt, B. F. III (2010). NF-YC3, NF-YC4 and NF-YC9 are required for CONSTANS-mediated, photoperiod-dependent flowering in *Arabidopsis thaliana*. ***Plant J.*** 63, 379–391. doi: 10.1111/j.1365-313X.2010.04247.x

Kusnetsov V, Landsberger M, Meurer J, Oelmuller R. (1999). The assembly of the CAAT-box binding complex at a photosynthesis gene promoter is regulated by light, cytokinin, and the stage of the plastids. ***The Journal of Biological Chemistry* 274**: 36009– 36014.

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.

Lauter N, Kampani A, Carlson S, Goebel M, Moose SP. (2005). microRNA172 down-regulates glossy15 to promote vegetative phase change in maize. ***Proceedings of the National Academy of Sciences****, USA* **102**: 9412– 9417.

Lazaro, A., Valverde, F., Pineiro, 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. doi: 10.1105/tpc.110.081885

Lazzarin, M., Meisenburg, M., Meijer, D., van Ieperen, W., Marcelis, L. F. M., Kappers, I. F., *et. al.,* (2021). LEDs make it resilient: effects on plant growth and defense. ***Trends Plant Sci.*** 26, 496–508. doi: 10.1016/j.tplants.2020.11.013

Lee J, Oh M, Park H, Lee I. (2008). SOC1 translocated to the nucleus by interaction with AGL24 directly regulates leafy. ***Plant Journal* 55**: 832– 843.

Lee JH, Yoo SJ, Park SH, Hwang I, Lee JS, Ahn JH. (2007). Role of SVP in the control of flowering time by ambient temperature in *Arabidopsis*. **Genes and Development 21**: 397– 402.

Lee, C. M., and Thomashow, M. F. (2012). Photoperiodic regulation of the C-repeat binding factor (CBF) cold acclimation pathway and freezing tolerance in *Arabidopsis thaliana*. ***Proc. Natl. Acad. Sci.*** U.S.A. 109, 15054–15059. doi: 10.1073/pnas.1211295109

Legris, M., Klose, C., Burgie, E. S., Rojas, C. C. M., Neme, M., Hiltbrunner, A., Wigge, P. A., & Schafer, E. (2021). Phytochrome B integrates light and temperature signals in *Arabidopsis*. ***Science Advances,*** 7(6), eabd7692. https://doi.org/10.1126/sciadv.abd7692

Lepisto, A., and Rintamaki, E. (2012). Coordination of plastid and light signaling pathways upon development of *Arabidopsis* leaves under various photoperiods. ***Mol. Plant*** 5, 799–816. doi: 10.1093/mp/ssr106

Li, W. X., Oono, Y., Zhu, J., He, X. J., Wu, J. M., Iida, K., *et. al.,* (2008). The *Arabidopsis* NF-YA5 transcription factor is regulated transcriptionally and post-transcriptionally to promote drought resistance. ***Plant Cell*** 20, 2238–2251. doi: 10.1105/tpc.108.059444

Li, Y., Chen, L., Mu, J., and Zuo, J. (2013). LESION SIMULATING DISEASE1 interacts with catalases to regulate hypersensitive cell death in *Arabidopsis*. ***Plant Physiol.*** 163, 1059–1070. doi: 10.1104/pp.113.225805

Lin M, Belanger H, Lee Y, VarkonyiGasic E, Taoka K-C, Miura E, XoconostleCazares B, Gendler K, Jorgensen R, Phinney B *et a.,* (2007). FLOWERING LOCUS T protein may act as the long-distance florigenic signal in the Cucurbits. ***Plant Cell* 19**: 1488– 1506.

Macioszek, V. K., Sobczak, M., Skoczowski, A., Oliwa, J., Michlewska, S., Gapińska, M., *et. al.,* (2021). The effect of photoperiod on necrosis development, photosynthetic efficiency and 'Green Islands' formation in *Brassica juncea* infected with *Alternaria brassicicola*. ***Int. J. Mol. Sci.*** 22:8435. doi: 10.3390/ijms22168435

Mahajan, S., and Tuteja, N. (2005). Cold, salinity and drought stresses: an overview. ***Arch. Biochem. Biophys.*** 444, 139–158. doi: 10.1016/j.abb.(2005).10.018

Marla, S. R., Chu, K., Chintamanani, S., Multani, D. S., Klempien, A., DeLeon, A., *et. al.,* (2018). Adult plant resistance in maize to northern leaf spot is a feature of partial loss-of-function alleles of Hm1. ***PLoS Pathog.*** 14:e1007356. doi: 10.1371/journal.ppat.1007356

Martínez C, Pons E, Prats G, León J. (2004). Salicylic acid regulates flowering time and links defence responses and reproductive development. ***Plant Journal* 37**: 209– 217.

Martinez-Garcia JF, Virgos-Soler A, Prat S. (2002). Control of photoperiod-regulated tuberization in potato by the *Arabidopsis* flowering-time gene CONSTANS. ***Proceedings of the National Academy of Sciences,*** *USA* **99**: 15211– 15216.

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

Mas, P., Kim, W. Y., Somers, D. E., and Kay, S. A. (2003). Targeted degradation of TOC1 by ZTL modulates circadian function in *Arabidopsis thaliana*. ***Nature*** 426, 567–570. doi: 10.1038/nature02163

Mateo, A., Muhlenbock, P., Rusterucci, C., Chang, C. C., Miszalski, Z., Karpinska, B., *et. al.,* (2004). LESION SIMULATING DISEASE 1 is required for acclimation to conditions that promote excess excitation energy. ***Plant Physiol.*** 136, 2818–2830. doi: 10.1104/pp.104.043646

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, L. J., Murdter, F., Kuttner, F., and Schmid, M. (2009). Repression of flowering by the miR172 target SMZ. ***PLoS Biol***. 7:e1000148. doi: 10.1371/journal.pbio.1000148

Matsoukas, I. G. (2014). Attainment of reproductive competence, phase transition, and quantification of juvenility in mutant genetic screens. ***Front. Plant Sci.*** 5:32. doi: 10.3389/fpls.2014.00032

McClung, C. R. (2006). Plant circadian rhythms. ***Plant Cell*** 18, 792–803. doi: 10.1105/tpc.106.040980

McKay, J. K., Richards, J. H., and Mitchell-Olds, T. (2003). Genetics of drought adaptation in *Arabidopsis thaliana*: I. Pleiotropy contributes to genetic correlations among ecological traits. ***Mol. Ecol.*** 12, 1137–1151. doi: 10.1046/j.1365-294x.(2003).01833.x

McMaster, G. S., and Wilhelm, W. W. (2003). Phenological responses of wheat and barley to water and temperature: improving simulation models. ***J. Agric. Sci.*** 141, 129–147. doi: 10.1017/S0021859603003460

Meng, X., Chen, L., Zhang, Y., & Gao, J. (2022). Molecular regulation of soybean flowering by photoperiod. ***Plant Cell Reports,*** 41(1), 1-14. https://doi.org/10.1007/s00299-021-02795-5

Michael TP, Salome PA, Yu HJ, Spencer TR, Sharp EL, McPeek MA, Alonso JM, Ecker JR, McClung CR. (2003). Enhanced fitness conferred by naturally occurring variation in the circadian clock. ***Science*** **302**: 1049– 1053.

Mittler, R. (2017). ROS are good. ***Trends Plant Sci.*** 22, 11–19. doi: 10.1016/j.tplants.2016.08.002

Moon J, Suh SS, Lee H, Choi KR, Hong CB, Paek NC, Kim SG, Lee I. (2003). The SOC1 MADS-box gene integrates vernalization and gibberellin signals for flowering in *Arabidopsis*. ***Plant Journal*** **35**: 613– 623.

Moraes, T. A., Mengin, V., Annunziata, M. G., Encke, B., Krohn, N., Hohne, M., *et. al.,* (2019). Response of the circadian clock and diel starch turnover to one day of low light or low CO2. ***Plant Physiol.*** 179, 1457–1478. doi: 10.1104/pp.18.01418

Mouradov A, Cremer F, Coupland G. (2002). Control of flowering time: interacting pathways as a basis for diversity. ***Plant Cell*** **14** (Suppl): S111– 130.

Munns, R. (2002). Comparative physiology of salt and water stress. ***Plant Cell Environ.*** 25, 239–250. doi: 10.1046/j.0016-8025.2001.00808.x

Munns, R., and Tester, M. (2008). Mechanisms of salinity tolerance. ***Annu. Rev. Plant Biol.*** 59, 651–681. doi: 10.1146/annurev.arplant.59.032607.092911

Nakamichi, N., Kiba, T., Kamioka, M., Suzuki, T., Yamashino, T., Higashiyama, T., *et. al.,* (2012). Transcriptional repressor PRR5 directly regulates clock-output pathways. ***Proc. Natl. Acad. Sci.*** U.S.A. 109, 17123–17128. doi: 10.1073/pnas.1205156109

Nelson, D. E., Repetti, P. P., Adams, T. R., Creelman, R. A., Wu, J., Warner, D. C., *et. al.,* (2007). Plant nuclear factor Y (NF-Y) B subunits confer drought tolerance and lead to improved corn yields on water-limited acres. ***Proc. Natl. Acad. Sci.*** U.S.A. 104, 16450–16455. doi: 10.1073/pnas.0707193104

Ni, Z., Hu, Z., Jiang, Q., and Zhang, H. (2013). GmNFYA3, a target gene of miR169, is a positive regulator of plant tolerance to drought stress. ***Plant Mol. Biol.*** 82, 113–129. doi: 10.1007/s11103-013-0040-5

Nilsson O, Lee I, Blazquez MA, Weigel D. (19980. Flowering-time genes modulate the response to LEAFY activity. ***Genetics*** **150**: 403– 410.

Nitschke, S., Cortleven, A., and Schmülling, T. (2017). Novel stress in plants by altering the photoperiod. ***Trends Plant Sci.*** 22, 913–916. doi: 10.1016/j.tplants.2017.09.005

Nitschke, S., Cortleven, A., Iven, T., Feussner, I., Havaux, M., Riefler, M., *et. al.,* (2016). Circadian stress regimes affect the circadian clock and cause jasmonic acid-dependent cell death in cytokinin-deficient *Arabidopsis* plants. ***Plant Cell*** 28, 1616–1639. doi: 10.1105/tpc.16.00016

Onouchi H, Igeño MI, Périlleux C, Graves K, Coupland G. (2000). Mutagenesis of plants overexpressing CONSTANS demonstrates novel interactions among *Arabidopsis* flowering-time genes. ***Plant Cell*** **12**: 885– 900.

Orkwiszewski JA, Poethig RS. (2000). Phase identity of the maize leaf is determined after leaf initiation. ***Proceedings of the National Academy of Sciences,*** *USA* **97**: 10631– 10636.

Osterlund MT, Hardtke CS, Wei N, Deng XW. 2000. Targeted destabilization of HY5 during light-regulated development of *Arabidopsis*. ***Nature*** **405**: 462– 466.

Outlaw, J. W. H. (2003). Integration of cellular and physiological functions of guard cells. ***Crit. Rev. Plant Sci.*** 22, 503–529. doi: 10.1080/713608316

Pareek, A., Khurana, A., Sharma, A. K., and Kumar, R. (2017). An overview of signaling regulons during cold stress tolerance in plants. ***Curr. Genomics*** 18, 498–511. doi: 10.2174/1389202918666170228141345

Park MY, Wu G, Gonzalez-Sulser A, Vaucheret H, Poethig RS. (2005). Nuclear processing and export of microRNAs in *Arabidopsis*. ***Proceedings of the National Academy of Sciences,*** *USA* **102**: 3691– 3696.

Park, H. J., Kim, W. Y., and Yun, D. J. (2016). A new insight of salt stress signaling in plant. ***Mol. Cells*** 39, 447–459. doi: 10.14348/molcells.2016.0083

Peragine A, Yoshikawa M, Wu G, Albrecht HL, Poethig RS. (2004). SGS3 and SGS2/SDE1/RDR6 are required for juvenile development and the production of trans-acting siRNAs in *Arabidopsis*. ***Genes and Development*** **18**: 2368– 2379.

Pereira, S. L. S., Martins, C. P. S., Sousa, A. O., Camillo, L. R., Araujo, C. P., Alcantara, G. M., *et. al.,* (2018). Genome-wide characterization and expression analysis of citrus NUCLEAR FACTOR-Y (NF-Y) transcription factors identified a novel NF-YA gene involved in drought-stress response and tolerance. ***PLoS One*** 13:e0199187. doi: 10.1371/journal.pone.0199187

Pfannschmidt, T., Brautigam, K., Wagner, R., Dietzel, L., Schroter, Y., Steiner, S., *et. al.,* (2009). Potential regulation of gene expression in photosynthetic cells by redox and energy state: approaches towards better understanding. ***Ann. Bot.*** 103, 599–607. doi: 10.1093/aob/mcn081

Pineiro M, Gomez-Mena C, Schaffer R, Martinez-Zapater JM, Coupland G. (2003). EARLY BOLTING IN SHORT DAYS is related to chromatin remodeling factors and regulates flowering in *Arabidopsis* by repressing FT. ***Plant Cell*** **15**: 1552– 1562.

Poethig RS. (1988). Heterochronic mutations affecting shoot development in maize. ***Genetics*** **119**: 959– 973.

Poethig RS. (1990). Phase change and the regulation of shoot morphogenesis in plants. ***Science*** **250**: 923– 930.

Pouteau S, Nicholls D, Tooke F, Coen E, Battey N. (1997). The induction and maintenance of flowering in *Impatiens*. ***Development*** **124**: 3343– 3351.

Qiu, Q. S., Guo, Y., Dietrich, M. A., Schumaker, K. S., and Zhu, J. K. (2002). Regulation of SOS1, a plasma membrane Na+/H+ exchanger in *Arabidopsis thaliana*, by SOS2 and SOS3. ***Proc. Natl. Acad. Sci.*** U.S.A. 99, 8436–8441. doi: 10.1073/pnas.122224699

Queval, G., Issakidis-Bourguet, E., Hoeberichts, F. A., Vandorpe, M., Gakiere, B., Vanacker, H., *et. al.,* (2007). Conditional oxidative stress responses in the *Arabidopsis* photorespiratory mutant cat2 demonstrate that redox state is a key modulator of daylength-dependent gene expression, and define photoperiod as a crucial factor in the regulation of H2O2-induced cell death. ***Plant J.*** 52, 640–657. doi: 10.1111/j.1365-313X.2007.03263.x

Rawat, R., Takahashi, N., Hsu, P. Y., Jones, M. A., Schwartz, J., Salemi, M. R., *et. al.,* (2011). REVEILLE8 and PSEUDO-REPONSE REGULATOR5 form a negative feedback loop within the *Arabidopsis* circadian clock. ***PLoS Genet.*** 7:e1001350. doi: 10.1371/journal.pgen.1001350

Razem FA, El-Kereamy A, Abrams SR, Hill RD. (2006). The RNA-binding protein FCA is an abscisic acid receptor. ***Nature*** **439**: 290– 294.

Reeves PA, He Y, Schmitz RJ, Amasino RM, Panella LW, Richards CM. (2007). Evolutionary conservation of the FLOWERING LOCUS C-mediated vernalization response: evidence from the sugar beet (*Beta vulgaris*). ***Genetics*** **176**: 295– 307.

Riboni, M., Galbiati, M., Tonelli, C., and Conti, L. (2013). GIGANTEA enables drought escape response via abscisic acid-dependent activation of the florigens and SUPPRESSOR OF OVEREXPRESSION OF CONSTANS. ***Plant Physiol.*** 162, 1706–1719. doi: 10.1104/pp.113.217729

Riboni, M., Robustelli, T. A., Galbiati, M., Tonelli, C., and Conti, L. (2016). ABA-dependent control of GIGANTEA signalling enables drought escape via up-regulation of FLOWERING LOCUS T in *Arabidopsis thaliana*. ***J. Exp. Bot.*** 67, 6309–6322. doi: 10.1093/jxb/erw384

Roberts A, Blake P, Lewis R, Taylor J, Dunstan D. (1999). The effect of gibberellins on flowering in roses. ***Journal of Plant Growth Regulation*** **18**: 113– 119.

Robinson LW, Wareing PF. (1969). Experiments on the juvenile-adult phase change in some woody species. ***New Phytologist*** **68**: 67– 78.

Robson F, Costa MMR, Hepworth SR, Vizir I, Piñeiro M, Reeves PH, Putterill J, Coupland G. (2001). Functional importance of conserved domains in the flowering-time gene CONSTANS demonstrated by analysis of mutant alleles and transgenic plants. ***Plant Journal*** **28**: 619– 631.

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.

Rodriguez-Falcon M, Bou J, Prat S. (2006). Seasonal control of tuberization in potato: conserved elements with the flowering response. ***Annual Review of Plant Biology*** **57**: 151– 180.

Roeber, V. M., Bajaj, I., Rohde, M., Schmulling, T., and Cortleven, A. (2021). Light acts as a stressor and influences abiotic and biotic stress responses in plants. ***Plant Cell Environ.*** 44, 645–664. doi: 10.1111/pce.13948

Rugnone, M. L., Faigon Soverna, A., Sanchez, S. E., Schlaen, R. G., Hernando, C. E., Seymour, D. K., *et. al.,* (2013). LNK genes integrate light and clock signaling networks at the core of the *Arabidopsis* oscillator. ***Proc. Natl. Acad. Sci. U.S.A.*** 110, 12120–12125. doi: 10.1073/pnas.1302170110

Saijo Y, Sullivan JA, Wang H, Yang J, Shen Y, Rubio V, Ma L, Hoecker U, Deng XW. (2003). The COP1-SPA1 interaction defines a critical step in phytochrome A-mediated regulation of HY5 activity. ***Genes and Development*** **17**: 2642– 2647.

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– 1616.

Sanchez, A., Shin, J., and Davis, S. J. (2011). Abiotic stress and the plant circadian clock. ***Plant Signal. Behav***. 6, 223–231. doi: 10.4161/psb.6.2.14893

Sanchez, S. E., Rugnone, M. L., and Kay, S. A. (2020). Light perception: a matter of time. **Mol. Plant** 13, 363–385. doi: 10.1016/j.molp.2020.02.006

Sarkar, D. (2010). Photoperiodic inhibition of potato tuberization: an update. ***Plant Growth Regul.*** 62, 117–125. doi: 10.1007/s10725-010-9502-9

Sawa, M., Nusinow, D. A., Kay, S. A., and Imaizumi, T. (2007). FKF1 and GIGANTEA complex formation is required for day-length measurement in *Arabidopsis*. ***Science*** 318, 261–265. doi: 10.1126/science.1146994

Schaffer, R., Ramsay, N., Samach, A., Corden, S., Putterill, J., Carré, I. A., *et. al.,* (1998). The late elongated hypocotyl mutation of *Arabidopsis* disrupts circadian rhythms and the photoperiodic control of flowering. ***Cell*** 93, 1219–1229. doi: 10.1016/s0092-8674(00)81465-8

Schmid M, Uhlenhaut NH, Godard F, Demar M, Bressan R, Weigel D, Lohmann JU. (2003). Dissection of floral induction pathways using global expression analysis. ***Development*** **130**: 6001– 6012.

Schwarz S, Grande AV, Bujdoso N, Saedler H, Huijser P. (2008). The microRNA regulated SBP-box genes SPL9 and SPL15 control shoot maturation in *Arabidopsis*. ***Plant Molecular Biology*** **67**: 183– 195.

Searle I, He YH, Turck F, Vincent C, Fornara F, Kröber S, Amasino RA, Coupland G. (2006). The transcription factor FLC confers a flowering response to vernalization by repressing meristem competence and systemic signaling in *Arabidopsis*. ***Genes and Development*** **20**: 898– 912.

Seo HS, Yang JY, Ishikawa M, Bolle C, Ballesteros ML, Chua NH. (2003). LAF1 ubiquitination by COP1 controls photomorphogenesis and is stimulated by SPA1. ***Nature*** **423**: 995– 999.

Seo, P. J., and Mas, P. (2015). STRESSing the role of the plant circadian clock. ***Trends Plant Sci.*** 20, 230–237. doi: 10.1016/j.tplants.2015.01.001

Serrano-Bueno, G., Sanchez de Medina Hernandez, V., and Valverde, F. (2021). Photoperiodic signaling and senescence, an ancient solution to a modern problem? ***Front. Plant Sci.*** 12:634393. doi: 10.3389/fpls.2021.634393

Sharma, M., Irfan, M., Kumar, A., Kumar, P., and Datta, A. (2021). Recent insights into plant circadian clock response against abiotic stress. ***J. Plant Growth Regul.*** 221:112403. doi: 10.1007/s00344-021-10531-y

Sherrard, M. E., and Maherali, H. (2006). The adaptive significance of drought escape in *Avena barbata*, an annual grass. ***Evolution*** 60, 2478–2489. doi: 10.1554/06-150.1

Shi, H., and Chan, Z.-L. (2014). AtHAP5A modulates freezing stress resistance in *Arabidopsis* independent of the CBF pathway. ***Plant Signal. Behav***. 9:e29109. doi: 10.4161/psb.29109

Shi, H., Ishitani, M., Kim, C., and Zhu, J. K. (2000). The *Arabidopsis thaliana* salt tolerance gene SOS1 encodes a putative Na+/H+ antiporter. ***Proc. Natl. Acad. Sci. U.S.A.*** 97, 6896–6901. doi: 10.1073/pnas.120170197

Shim, J. S., and Imaizumi, T. (2015). Circadian clock and photoperiodic response in *Arabidopsis*: from seasonal flowering to redox homeostasis. ***Biochemistry*** 54, 157–170. doi: 10.1021/bi500922q

Shim, J. S., Kubota, A., and Imaizumi, T. (2017). Circadian clock and photoperiodic flowering in *Arabidopsis*: CONSTANS is a hub for signal integration. ***Plant Physiol.*** 173, 5–15. doi: 10.1104/pp.16.01327

Shimizu, S., Yamauchi, Y., and Ishikawa, A. (2021). Photoperiod following inoculation of *Arabidopsis* with *Pyricularia oryzae* (syn. *Magnaporthe oryzae*) influences on the plant–pathogen interaction. ***Int. J. Mol. Sci.*** 22:5004. doi: 10.3390/ijms22095004

Singer SR, Hannon CH, Huber SC. (1992). Aquisition of competence for floral development in *Nicotiana* buds. ***Planta*** **188**: 546– 550.

Singh, R. K., Svystun, T., Al Dahmash, B., Jonsson, A. M., and Bhalerao, R. P. (2017). Photoperiod- and temperature-mediated control of phenology in trees - a molecular perspective. ***New Phytol.*** 213, 511–524. doi: 10.1111/nph.14346

Sivitz AB, Reinders A, Johnson ME, Krentz AD, Grof CP, Perroux JM, Ward JM. (2007). *Arabidopsis* sucrose transporter AtSUC9. High-affinity transport activity, intragenic control of expression, and early flowering mutant phenotype. ***Plant Physiology*** **143**: 188– 198.

Smith HM, Campbell BC, Hake S. (2004). Competence to respond to floral inductive signals requires the homeobox genes PENNYWISE and POUND-FOOLISH. ***Current Biology*** **14**: 812– 817.

Smith, A. M., and Stitt, M. (2007). Coordination of carbon supply and plant growth. ***Plant Cell Environ.*** 30, 1126–1249. doi: 10.1111/j.1365-3040.2007.01708.x

Somers DE, Devlin PF, Kay SA. (19980. Phytochromes and cryptochromes in the entrainment of the *Arabidopsis* circadian clock. ***Science* 282**: 1488– 1490.

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. doi: 10.1146/annurev-arplant-043014-115555

Souer E, Van Der Krol A, Kloos D, Spelt C, Bliek M, Mol J, Koes R. (1998). Genetic control of branching pattern and floral identity during *Petunia* inflorescence development. ***Development* 125**: 733– 742.

Stadler R, Wright KM, Lauterbach C, Amon G, Gahrtz M, Feuerstein A, Oparka KJ, Sauer N. (2005). Expression of GFP-fusions in *Arabidopsis* companion cells reveals nonspecific protein trafficking into sieve elements and identifies a novel post-phloem domain in roots. ***Plant Journal* 41**: 319– 331.

Steward F, Moreno V, Roca W. (1981). Growth form and composition of potato plants as affected by environment. ***Annals of Botany* 48** (Suppl. 2): 45.

Stitt, M., and Zeeman, S. C. (2012). Starch turnover: pathways, regulation and role in growth. ***Curr. Opin. Plant Biol.*** 15, 282–292. doi: 10.1016/j.pbi.2012.03.016

Su, H., Cao, Y., Ku, L., Yao, W., Cao, Y., Ren, Z., *et. al.,* (2018). Dual functions of ZmNF-YA3 in photoperiod-dependent flowering and abiotic stress responses in maize. ***J. Exp. Bot.*** 69, 5177–5189. doi: 10.1093/jxb/ery299

Suàrez-López 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.

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, 1116–1120. doi: 10.1038/35074138

Swiezewski S, Crevillen P, Liu F, Ecker JR, Jerzmanowski A, Dean C. (2007). Small RNA-mediated chromatin silencing directed to the 3′ region of the *Arabidopsis* gene encoding the developmental regulator, ***FLC. Proceedings of the National Academy of Sciences,*** *USA* **104**: 3633– 3638.

Takada S, Goto K. (2003). TERMINAL FLOWER 2, an *Arabidopsis* homolog of HETEROCHROMATIN PROTEIN 1, counteracts the activation of FLOWERING LOCUS T by CONSTANS in the vascular tissues of leaves to regulate flowering time. ***Plant Cell* 15**: 2856– 2865.

Takeno K. (1991). Flowering response of *Ipomaea batatas* scions grafted onto *Pharbitis nil* stocks. ***Physiologia Plantarum* 63**: 682– 686.

Tamaki S, Matsuo S, Wong HL, Yokoi S, Shimamoto K. (2007). Hd3a protein is a mobile flowering signal in rice. ***Science* 316**: 1033– 1036.

Tavares, S., Moreira, S., Figueiredo, D., & Santos, M. A. (2021). Adaptation of *Arabidopsis thaliana* to high light intensity conditions through changes in gene expression and metabolism. ***Plant Science***, 311, 111025. https://doi.org/10.1016/j.plantsci.2021.111025

Telfer A, Bollman KM, Poethig RS. (19970. Phase change and the regulation of trichome distribution in *Arabidopsis thaliana*. ***Development* 124**: 645– 654.

Tessadori F, Schulkes RK, Van Driel R, Fransz P. (2007). Light-regulated large-scale reorganization of chromatin during the floral transition in *Arabidopsis*. ***Plant Journal* 50**: 848– 857.

Thomas B, Vince-Prue D. (1997). *Photoperiodism in plants*. London, ***UK: Academic Press.***

Thomashow, M. F. (2010). Molecular basis of plant cold acclimation: insights gained from studying the CBF cold response pathway. ***Plant Physiol.*** 154, 571–577. doi: 10.1104/pp.110.161794

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.

Vega SH, Sauer M, Orkwiszewski JAJ, Poethig RS. (2002). The early phase change gene in maize. ***Plant Cell* 14**: 133– 147.

Von Arnim AG, Deng XW. (1994). Light inactivation of *Arabidopsis* photomorphogenic repressor COP1 involves a cell-specific regulation of its nucleocytoplasmic partitioning. *Cell* **79**: 1035– 1045.

Walters, R. G., and Horton, P. (1995). Acclimation of *Arabidopsis thaliana* to the light environment: regulation of chloroplast composition***. Planta*** 197, 475–481. doi: 10.1007/BF00196669

Wang H, Ma LG, Li JM, Zhao HY, Deng XW. (2001). Direct interaction of *Arabidopsis* cryptochromes with COP1 in light control development. ***Science* 294**: 154– 158.

Wang, Z. Y., and Tobin, E. M. (1998). Constitutive expression of the CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) gene disrupts circadian rhythms and suppresses its own expression. ***Cell*** 93, 1207–1217. doi: 10.1016/s0092-8674(00)81464-6

Weigel D, Nilsson O. (19950. A developmental switch sufficient for flower initiation in diverse plants. ***Nature* 377**: 495– 500.

Welling, A., Moritz, T., Palva, E. T., and Junttila, O. (2002). Independent activation of cold acclimation by low temperature and short photoperiod in hybrid aspen. ***Plant Physiol.*** 129, 1633–1641. doi: 10.1104/pp.003814

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*. ***Plant Cell* 18**: 2971– 2984.

Wigge PA, Kim MC, Jaeger KE, Busch W, Schmid M, Lohmann JU, Weigel D. (2005). Integration of spatial and temporal information during floral induction in *Arabidopsis*. ***Science* 309**: 1056– 1059.

Willmann MR, Poethig RS. (2005). Time to grow up: the temporal role of small RNAs in plants. ***Current Opinion in Plant Biology* 8**: 548– 552.

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

Wu, Y., Wang, Q., Zhou, X., & Zhang, T. (2022). The photoperiodic pathway in the regulation of plant growth and development. ***Journal of Integrative Plant Biology,*** 64(1), 57-70. https://doi.org/10.1111/jipb.13253

Xie ZX, Allen E, Wilken A, Carrington JC. (2005). DICER-LIKE 4 functions in trans-acting small interfering RNA biogenesis and vegetative phase change in *Arabidopsis thaliana*. ***Proceedings of the National Academy of Sciences,*** *USA* **102**: 12984– 12989.

Xie, Q., Lou, P., Hermand, V., Aman, R., Park, H. J., Yun, D. J., *et. al.,* (2015). Allelic polymorphism of GIGANTEA is responsible for naturally occurring variation in circadian period in *Brassica rapa*. ***Proc. Natl. Acad. Sci.*** U.S.A. 112, 3829–3834. doi: 10.1073/pnas.1421803112

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.

Yan L, Loukoianov A, Blechl A, Tranquilli G, Ramakrishna W, SanMiguel P, Bennetzen JL, Echenique V, Dubcovsky J. (2004). The wheat VRN2 gene is a flowering repressor down-regulated by vernalization. ***Science* 303**: 1640– 1644.

Yang, Y. X., Wang, M. M., Yin, Y. L., Onac, E., Zhou, G. F., Peng, S., *et. al.,* (2015). RNA-seq analysis reveals the role of red light in resistance against *Pseudomonas syringae* pv. tomato DC3000 in tomato plants. ***BMC Genomics*** 16:120. doi: 10.1186/s12864-015-1228-7

Yanovsky MJ, Kay SA. (2002). Molecular basis of seasonal time measurement in *Arabidopsis*. ***Nature*** **419**: 308– 312.

Yoo BC, Kragler F, Varkonyi-Gasic E, Haywood V, Archer-Evans S, Lee YM, Lough TJ, Lucas WJ. (2004). A systemic small RNA signaling system in plants. ***Plant Cell*** **16**: 1979– 2000.

Yoo SK, Chung KS, Kim JK, Lee JH, Hong SM, Yoo SJ, Yoo SY, Lee JS, Ahn JH. (2005). CONSTANS activates SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1 through FLOWERING LOCUS T to promote flowering in *Arabidopsis*. ***Plant Physiology*** **139**: 770– 778.

Yu H, Ito T, Zhao Y, Peng J, Kumar P, Meyerowitz EM. (2004). Floral homeotic genes are targets of gibberellin signaling in flower development. ***Proceedings of the National Academy of Sciences,*** *USA* **101**: 7827– 7832.

Zeeman, S. C., Smith, S. M., and Smith, A. M. (2007). The diurnal metabolism of leaf starch. ***Biochem. J.*** 401, 13–28. doi: 10.1042/BJ(2006)1393

Zeevaart JAD. (19620. The juvenile phase in *Bryophyllum daigremontianum*. ***Planta*** **58**: 531– 542.

Zeevaart, J. A. D. (1971). Abscisic acid content of spinach in relation to photoperiod and water stress. ***Plant Physiol***. 48, 86–90. doi: 10.1104/pp.48.1.86