How Plants Adapt to the Photoperiod

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

Review Article

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-critical 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 at 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 is 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 utilizes by plants to assess day length.

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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. In addition to these essential elements, several proteins and signalling pathways are also involved in photoperiodic responses in plants. 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 synchronize their developmental processes with seasonal changes is their capacity to react to variations in day length. Phytochromes play a key role 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.

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" [1]. "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" [2]. 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 [3,4], tuberization [5], bud set, and dormancy are some

of the most notable plant responses affected by the photoperiod [1,6]. "Season-dependent photoperiods affect the growth stoppage of perennial plants like trees and regulate senescence in annual plants" [6,7]. "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" [8]. Among a few examples of photoperiod-regulated developmental processes in plants, [9], 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" [1]. 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 thaliana* plants' flowering response is not affected by photoperiods. *Arabidopsis thaliana* becomes sensitive to photoperiods when it enters the adult phase, allowing it to respond to floral inducers [10]. 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 [11]. Also, it has been demonstrated that the photoperiod affects the plants' ability to withstand salt stress and drought stress [12,13,14]. Additionally, mounting data indicates that plant-pathogen interactions are influenced by the length of the light period [15]. 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 thaliana* 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 [16-20]. "Systemic acquired resistance (SAR) development in plants serves as a crucial line of protection against upcoming pathogen invasions" [21]. 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 [1,7]. 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. *Arabidopsis thaliana has* five different photoreceptor families that detect light at different 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 [22,23]. 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 [24]. Plants with extended days have smaller grana stacks and more chlorophyll in their chloroplasts. These qualities match the structural and photosynthetic traits typical of sun plants [25]. Plants' adaptation to light is based on redox signals generated by the chloroplasts [26]. "It is unclear which signalling pathways contribute to the photoperiodic-dependent growth of chloroplasts. The redox status of the photosynthetic electron transport chain, ROS metabolism, and chloroplast-to-nucleus retrograde communication are just a few examples of possible pathways involved in which all function independently of the photoreceptors" [24,27]. 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 [28]. A greater fraction of the fixed C is allocated to starch when there is less accessible C, such as during short photoperiods [29].

Compared to plants cultivated over 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 [30,31]. Across several photoperiods, this C-mobilization pattern is consistent [30,31]. "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" [32]. "In order to alter the internal rhythm, the clock is set daily by entrainment, particularly by light and temperature" [33]. "The circadian clock in *Arabidopsis thaliana is* made up of a number of interlocked transcription-translation feedback loops" [32]. In the morning [34,35], 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 [36]. The transcription of CCA1 and LHY is then repressed by TOC1 [37]. 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 of 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 [38]. 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 [39,40]. 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 different workers [41-44], include more details on this. Recently, a novel webtool was introduced to study the transcriptional networks influenced by light and the circadian clock [45]. 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.

GIGANTEA (GI) is one of the circadian clock-controlled genes that is essential to the photoperiod sensing system [46]. It produces a sizable chaperone-active protein that is encoded by a single big gene [47]. The interaction with GI enhances the stability of the F-box protein ZEITLUPE (ZTL) during the perception of blue light. ZTL is an evening-phased E3 ubiquitin ligase that targets the clock elements TOC1 and PRR5 for proteasomal degradation [48,49]. The evening is when GI protein quantity rises, keeping ZTL abundance high. As a result, TOC1 and PRR5 oscillations of high amplitude are maintained [50]. As a result, clock output genes like CONSTANS (CO), which encodes a key protein in photoperiod-dependent blooming, are correctly set in phase [51,52]. 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 [53]. 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 [7]. 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 [54,55]. Together with EARLY FLOWERING3 (ELF3), GI also regulates photoperiod sensing which 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 [56].
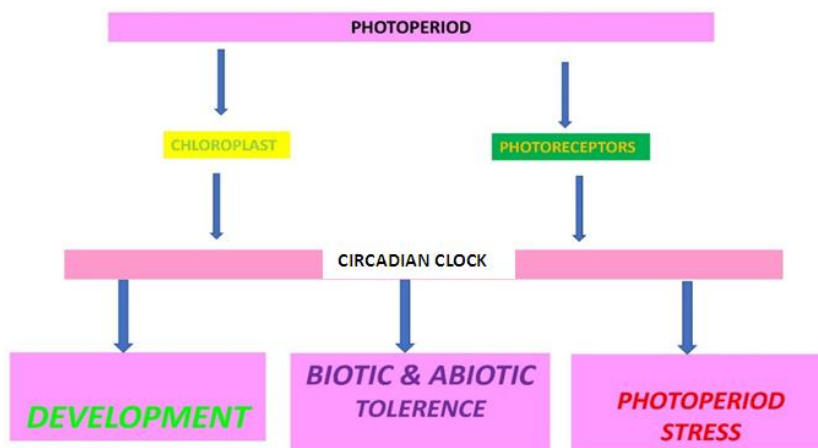

Fig. 1. Molecular mechanisms involved in light perception and the photoperiod
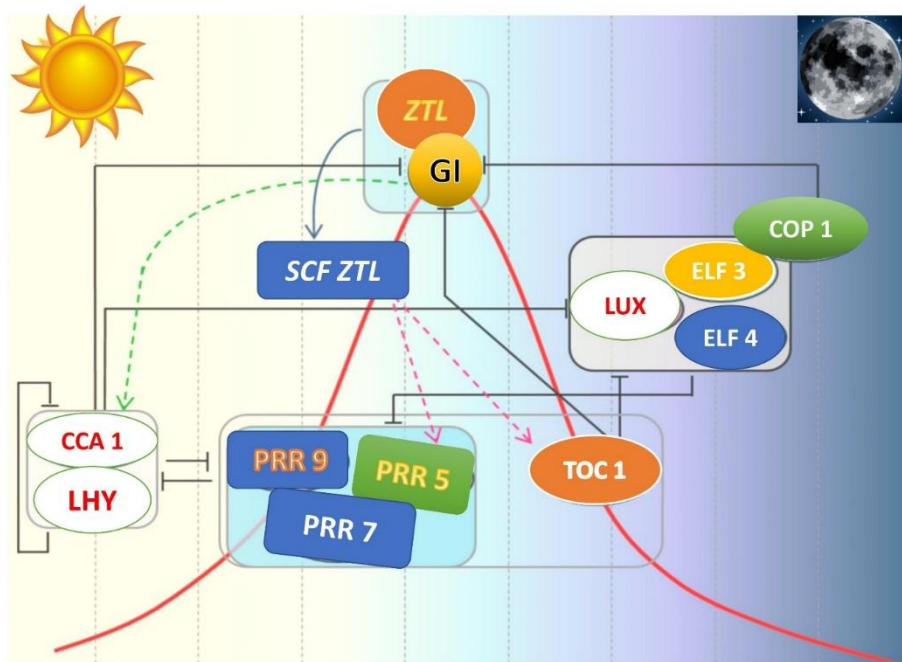
Fig. 2. Plants react to environmental stressors

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, [57,58].

2.1 Photoperiod Impacts Abiotic and Biotic Stress Reactions

2.1.1 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 [59]. 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 [60]. In *Populus tremula* and *P. tremuloides*, the major switch converting metabolism from vegetative growth to dormancy and establishing freezing tolerance is phyA-mediated apical bud development under short days [61].

Arabidopsis thaliana similarly exhibits increased cold tolerance brought on by a shortening of the photoperiod. The photoperiod conditions that different *Arabidopsis thaliana accessions* are geographically exposed to can affect their

freezing resistance [62]. 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 [63,64]. 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 [59].

The GI-CDF module controls freezing tolerance in *Arabidopsis*, one of the elements involved in photoperiodic blooming. The 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 [65,66].

As a result, in GI-100 CDF1235 mutants, this increase in COR gene expression was reduced [58]. 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 [57,66]. 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 [58]. "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" [67].

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 [68]. By facilitating the ubiquitination and proteasomal degradation of ICE1 (INDUCER OF CBF EXPRESSION1), HOS1 inhibits the body's ability to adapt to cold temperatures [69,70,59].

2.1.2 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 [71].

"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" [72]. "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" [73].

"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" [73]. "CO is necessary for the ABA-dependent activation of FT but not TSF" [74]. Increased amounts of florigen cause the floral integrator SUPPRESSOR OF OVEREXPRESSOR OF CONSTANS (SOC1) to

become active, which starts flowering. SOC1 activation aids in TSF overexpression, which further raises the levels of florigen [73]. 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 [74]. Additionally, the photoperiod-dependent flowering transcription factor family, which includes the NUCLEAR FACTOR Y (NF-Y) component C [75,76], is thought to have a role in drought resistance. The ABA-response element (ABRE)-binding factors (ABFs) engage with NF-Y subunit C-3/4/9 and cause SOC1 to stimulate flowering [77]. Along with *Avena barbata* [78], *Brassica rapus* [79], and other plants, *Arabidopsis thaliana has* a drought escape mechanism [80,81]. Other species, like rice, put their life cycle on hold during drought stress in order to continue it when the stress is gone [82]. 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. The photoperiodic regulation at least partially affects the synthesis and signalling of ABA [83].

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 [84]. A rate-limiting enzyme for the production of ABA is encoded by NCED3 [85]. 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 [84]. In GI-1 and eel mutants subjected to dehydration, NCED3 transcript abundance and ABA content reduced, which was correlated with their dehydration-sensitive phenotype [84]. These findings suggest that GI and EEL work synergistically to increase drought tolerance in plants by controlling ABA homeostasis.
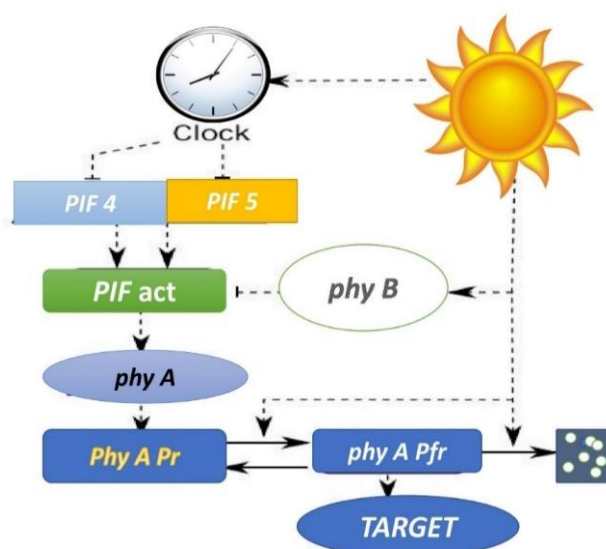
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Fig. 3. Photoperiod and Tolerance to Freezing

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 [12]. Long days and drought circumstances cause GI to encourage the production of pre-miRNA172, which suppresses WRKY44 and results in drought tolerance. In *Arabidopsis thaliana [*86,87], *Zea mays* [88,89], *Populus* [90], *Oryza sativa* [91], and *Citrus* [92], 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 [93]. ABA biosynthesis, signalling, and stress-responsive genes were expressed more often in transgenic *Arabidopsis thaliana plants* overexpressing the soybean NF-YA3 gene [87]. The overexpression of NF-YB1 in *Arabidopsis thaliana 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 [88]. In addition to their function in drought tolerance, NF-Y transcription factor overexpression enhances salt stress resistance [93] as well as freeze tolerance [94].

2.1.3 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 [17,16]. 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 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. 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 [18]. The photosystem II maximum quantum efficiency declines the following day, and PCD eventually occurs in the leaves [17,16].

Photoperiod stress was first recognised in *Arabidopsis thaliana plants* with cytokinin (CK) deficiency, which exhibit a particularly strong stress response. Among the CKs, trans-zeatin, in particular, has a protective role that is mediated via the transcriptional regulators *ARABIDOPSIS THALIANA RESPONSE* REGULATOR2 (ARR2), ARR10, and ARR12 and the *ARABIDOPSIS THALIANA HISTIDINE* KINASE3 (AHK3) receptor [19]. 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 [17,16]. Only a 4 hour increase in the light duration is needed to cause the short-day-entrained *Arabidopsis thaliana 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 [18]. 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) [95].

2.2 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 Marchantiophyta, 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 [96].
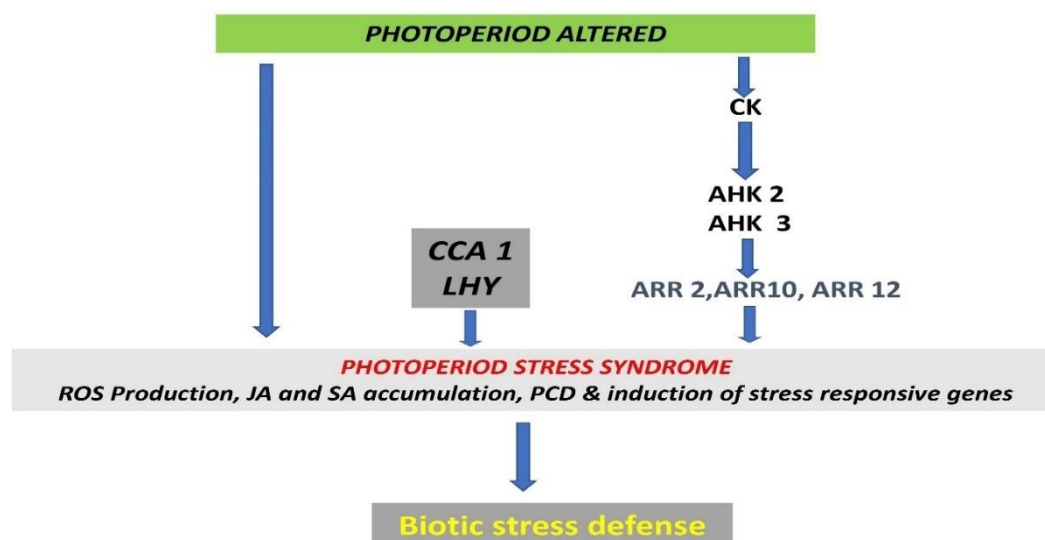
Fig. 4. Stress Caused by Photoperiod Changes

The three main types of photoperiod responses are short-day plants (SDP), long-day plants (LDP), and day-neutral plants (DNP), which do not respond to photoperiod. Short-day plants (SDP) are induced when the photoperiod is below the critical daylength (CDL), while LDP are induced when the photoperiod exceeds the CDL 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-hour 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 hours [96]. 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 [97]. 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 [96]. 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 [98]. 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.

2.3 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 [99]. 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 [99-103]. 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* [101,104,105]. 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 [106-108]. 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 [107]. 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 [109]. 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 thaliana that* have been linked to the length of the juvenile phase [110-113]. 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 [114,115], HST is involved in the synthesis or stability of some miRNAs, and SE is known to act in a miRNA gene silencing pathway [116], 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. [117,118]. 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 [112]. The hypothesis that TA-siRNAs are likely to have a more limited function in plant growth than miRNAs [119]. 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 [120].

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 [114]. 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.

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) [121]. 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 [122]. According to research [121,123], 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 [121,124].

In contrast to the pattern of expression of another miRNA, miR172, which also influences the length of the juvenile phase in maize [125], it has been demonstrated that the level of miR156 is higher in juvenile tissue than adult tissue [121]. 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.

In *Arabidopsis*, miR172 targets other AP2-like genes that are involved in suppressing the floral transition [55,126,127]. 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.
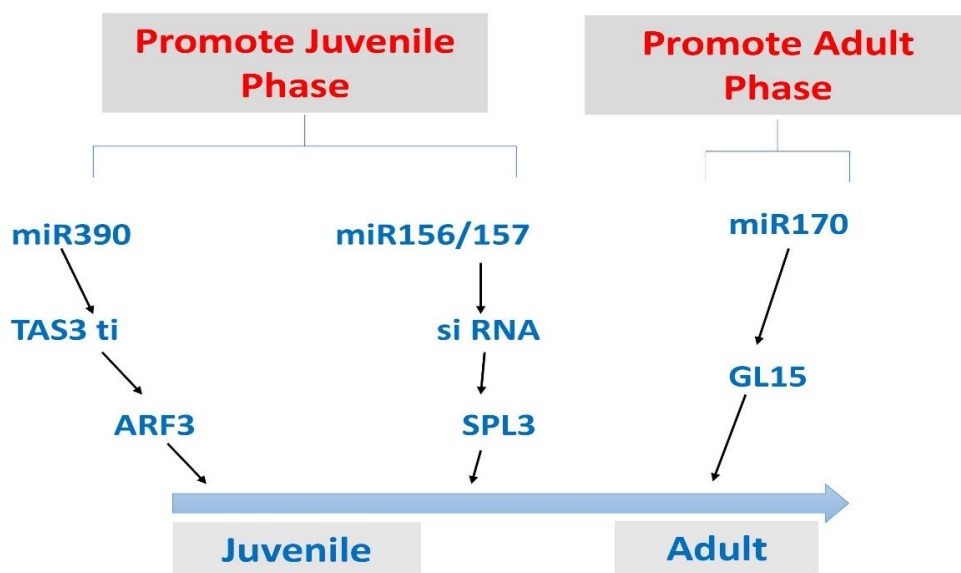

Fig. 5. Competence to Respond to Photoperiod

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 [107], 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 thaliana have* been demonstrated to be influenced by photoperiod and light quality, with levels being higher in LDs and blue light [127].

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 [128,129], 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 [130]. 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 [131]. 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 [132]. This behaviour in *Arabidopsis thaliana 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 [133]. As photoperiod was shown to modulate the effect of constitutive LFY over-expression on flowering time [134], 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 [135], and researchers are just now beginning to understand the underlying molecular mechanisms [136,137]. *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 [138-140]. 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 [141]. Recent research has demonstrated that PHY PNF double mutants block the activation of LFY by FLOWERING LOCUS T (FT) [142].

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 thaliana is* called FLOWERING LOCUS C (FLC). High FLC concentrations suppress FT expression and inhibit apical induction [143]. 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 [120,144-146].

3. 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.

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 [147-149]. 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 [150]. 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) [151]. 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 [152,153], showed that the coincidence of high levels of CO expression with light is necessary for floral induction.

3.1 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 [33,154,155]. 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 [36]. The 26S proteasome targets TOC1 for degradation, and ZTL controls the stability of the TOC1 protein in this situation [156]. The PSEUDO RESPONSE REGULATOR (PRR), GIGANTEA (GI), and LUX ARRHYTHMO (LUX) genes are involved in additional feedback loops [33,154].

3.2 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; [157]). 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 [158-160]. Light signals cause the clock's essential genes, such as LHY, CCA1, and PRR9, to express, which entrains the clock [35,161,162]. "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" [160]. 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 [53].

The CDF1 over-expressing lines and the GI and fkf1 mutants are all late flowering and have decreased amounts of CO mRNA [51,163,164]. Although FKF1, GI, and CDF1 are all regulated by the circadian rhythm, CDF1 expression peaks sooner in the morning than FKF1 and GI do [163-165]. 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 [53].

(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 [151]. 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 [166-168]. Because COP1 is excluded from the nucleus in the light [169], as well as because cryptochromes directly suppress COP1 in the light [170], 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 [51], the activity of COP1 prevents the CO protein from building up during the dark period [171]. 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 [172,173], may be involved in the degradation of CO by COP1.

3.3 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 a result of the CO protein accumulation-related genes and their somewhat different CO gene express patterns.

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 FKF 1 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 [163]. 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 [53]. Given that the GI protein is present in both LDs and SDs [174], 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 [175].

The floral integrator genes FT, TSF, and SOC1 are induced by CO to stimulate blooming [149,176]. 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 [51,153,177]. 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 thaliana 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 [178]. According to a theory put forth by Wenkel, 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 [179]. 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 [178]. In tobacco, the HAP complex has been demonstrated to bind CAAT motifs as well [180], 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" [181,182]. "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" [183]. However, unlike CO, FT can induce blooming if expressed through a meristem-specific promoter or even an epidermis-specific promoter [182]. 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 thaliana 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" [55,127].

"While CO expression is unaffected by miR172 over-expression in wild-type plants, FT expression is up-regulated" [127], 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 [127]. 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 [127]. Both of these routes are necessary for the promotion of blooming in LDs because the disruption of any one causes late blossoming in LDs.

5. 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 [96]. *Brassica napus* and *Cucurbita maxima* phloem have been found to contain the FT protein by mass spectrometry [184,185].

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 [54,186-188].

Scientist had made a strong case that the FT protein and its paralogues, such as TSF; 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 [54,187]. In order to regulate developmental processes like tuberization and leaf growth, mRNAs can also flow through the phloem throughout the plant [189-191]. Tamaki 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 [188]. 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 [192,121,123]. "However, transport of gene-silencing RNAs into the apex may be inhibited by the RNA surveillance system present at the SAM" [193].

"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)" [177,148].

"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" [177,148]. FT is also known to increase SOC1 expression in the SAM [192]. 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 [137]. 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 [98]. "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" [194,98]. "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" [195]. "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" [137,196].

6. 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 thaliana 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 (micro-RNA precursor), 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.*

COMPETING INTERESTS

Author has declared that no competing interests exist.

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