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Review

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Choosing which models best explain photoperiodic time measurement mechanisms in plants

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Seasonal responses can be triggered by photoperiod changes. To explain photoperiodic time measurement, three main models (hourglass, external coincidence, and internal coincidence) have been proposed based on physiological observations in plants and animals. It has been discussed which model fits best to explain each response. Studies in model plants like *Arabidopsis* and rice suggest their photoperiodic mechanisms incorporate features that fit more than one of these models.

"Photoperiodism," meaning the response to relative photoperiods or length of day and night, was coined more than 100 years ago to originally describe the day-length dependent flowering responses first characterized in soybeans, tobacco, and several other agronomic plants[1]. After the findings in plants in the early 20th century, animal physiologists discerned that many seasonal animal behaviors and development were also regulated by photoperiods[2,3]. Thus, photoperiodism has been recognized as a universal phenomenon in multicellular eukaryotes adapted to yearly changing environments. A recent finding has further expanded the extensive list of species demonstrating photoperiodism to the unicellular photosynthetic cyanobacteria, *Synechococcus elongatus*, as short-day conditions enhance resistance to cold temperature by increasing the membrane-lipid desaturation rates in preparation for winter[4].

In plants, in addition to flowering regulation, various responses, such as stem elongation, onset of bud dormancy and bud break, formation of storage organs (tubers and bulbs) and root nodules, leaf growth, etc., are subject to photoperiodism in a large variety of species[3,5,6]. Because of the physiological implications of the mechanistic links between circadian rhythms and photoperiodic responses, the photoperiodic time measurement mechanism has been one of the fundamental questions in chronology for decades[6].

Unlike other seasonal environmental factors, such as temperature and precipitation, photoperiod changes occur in a predictable manner from year to year, and the rates of change in photoperiods are highest around equinoxes and the lowest during summer and winter[7]. It is estimated that organisms in temperate regions should be able to discriminate changes in photoperiod as short as 30 minutes around the equinoxes[7]. Indeed, short-day plants, such as cocklebur (*Xanthium strumarium/Xanthium saccharatum/Xanthium orientale*, related *Xanthium*), morning glory (*Pharbitis nil*), soybean (*Glycine max*), etc., can often distinctly differentiate a less than 30 min difference in photoperiod around its critical day or night length to initiate flowering[3]. How do organisms precisely sense the difference in photoperiods? Classical physiological studies have long sought to explain the underlying mechanisms of photoperiodism. To explain the time sensing mechanisms, broadly speaking, three principal models have been proposed: the hourglass model, which posits a unidirectional timer initiated by dawn or dusk; Bünning's hypothesis and its extended model, the external coincidence model, where a light signal coincides with a circadian-regulated phase to trigger a response; and the internal coincidence model, involving the phase relationship between two internal oscillators entrained independently at dawn and dusk. These models have provided critical conceptual frameworks that guided decades of experimental research and shaped our understanding of how organisms control photoperiodism. Our way of discussing the underlying mechanism has often been which model best explains specific photoperiodic responses in each species. In the molecular genetic era, the results from both plant and animal-model organisms for photoperiodism have confirmed the significance of circadian clock-associated timing mechanisms to measure photoperiodic time differences[6]. Currently, the external coincidence model is being discussed as the model that best fits the molecular mechanisms of photoperiodism in these model organisms[8–10]. But these detailed molecular genetic results also indicate that some aspects are a better fit with other models. Therefore, the three theoretical models might not be mutually exclusive. This review mainly focuses on the discussion of photoperiod time measurement mechanisms in plants. Detailed updates on the molecular interactions of photoperiodism mechanisms in plants can be found in several recent reviews[10–18].

Classical models of photoperiodic time measurement

Classical models of photoperiodic time measurement provided conceptual foundations for decades of experimental research. All the models discussed here can explain the qualitative (whether response occurs or not) and quantitative (different degrees of responses) nature of photoperiodic responses. Here, I first revisit the three conceptual photoperiodic time measurement mechanisms [the hourglass, Bünning's hypothesis, and the external coincidence, and internal coincidence (two-oscillator) models], including brief historical aspects and some limitations.

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The hourglass model

The hourglass theory posits a simple mechanism: the transition from dark to light or light to dark initiates a chemical reaction, and once it reaches a certain threshold, the photoperiodic reaction will be induced. For plant physiological research, this model has been particularly supported by photobiologists who work on light responses for plant flowering. In 1960, Richard Hendrick proposed that the hourglass-type time measurement mechanism could be based on the general photochemical characteristic of a red/far-red light phototeceptor, phytochrome[19]. The onset of darkness initiates a reversion of the physiologically active phytochrome Pfr form to the inactive Pr form, called thermal reversion (Fig. 1a). (This used to be called dark reversion[20].) It was hypothesized that the amount of Pfr decreasing during nighttime constitutes an hourglass timer[3,19]. Soon after this, the theory was supported by the photoperiodic responses of seasonal morphotype conversion of insects, such as aphids (*Megoura viciae*)[21,22]. Thus, it became a more generalized model in which the onset of light or dark periods initiated count-up or count-down timer mechanisms. The key ideas of this model are that the timing of light on/off initiates the processes and that the photoperiodic reactions are not influenced by circadian-time modulated biochemical mechanisms.

In plants, the original mechanistic explanation for this model was mainly based on photoperiodism studies using short-day plants, such as *Xanthium* and soybean. The results of studying the action spectra of photoperiodic flowering—red light (660 nm) inhibits flowering in short-day plants while far-red light (730 nm) cancels the effect—together with photochemical characterization of phytochrome that controls other physiological responses (germination, anthocyanin accumulation, etc.), contributed to the formation of the hypothesis. Soon after the finding of photoperiodism, the presence of a threshold day length, referred to as the "critical day length", which triggers photoperiodic flowering, was recognized in many plant species. Short-day plants often displayed abrupt changes in photoperiodic responses when the photoperiods became shorter than their critical day length. Further studies with altering the length of either light (day) or dark (night) duration revealed that the length of night is more critical than the length of day; hence, the "critical night length," rather than day length, determines photoperiodic responses in these plants. In addition, a short exposure (often only a few minutes) of light interruption in the middle of the night, technically known as "night break", inhibits flowering in many short-day plants[23]. Moreover, the action spectra of dark-period-interrupting light to control *Xanthium saccharatum* flowering demonstrated red light inhibits flowering while far-red light promotes it[24], which was later known as phytochrome photoreversibility.

If the hourglass timer is used to measure time, the photoperiodic responses (for plants, mainly photoperiodic flowering) should be triggered after a certain length of day or night (Fig. 1a and b). By the combination of night-break experiments and extending night length (for several days), it has been shown that sensitivity to the light that causes flowering inhibition oscillates in a circadian fashion, rather than reaching a plateau after a certain amount of dark hours[3,23,25,26], indicating the photoperiodic timing mechanism is not regulated by the hourglass timer. The experimental procedure with various night lengths to study the contribution of circadian oscillators on photoperiodic responses has been commonly utilized for various species in the chronobiology field[26,27]. This procedure, later established as the "Nanda-Hamner protocols", has been used to investigate the presence of circadian resonance on the response (Fig. 1c and d)[26–28]. Unlike in animal chronobiology experiments, skeleton photoperiod experiments (applying two light pulses a day that define dawn and dusk at various timing combinations) have not been widely utilized in plant photoperiodism research, possibly because of their light-dependent autotroph nature.

In addition to a significant amount of evidence of the contribution of circadian mechanisms to photoperiodic time measurement, the thermal reversion rates of phytochrome in vitro (using extracts derived from etiolated tissues) and in vivo did not always fit the time scale to differentiate long and short nights[3]. Because of these observations, support for the hourglass model as a photoperiodic time mechanism in plants faded away during the 1960s-70s.

Bünning's hypothesis and the external coincidence model

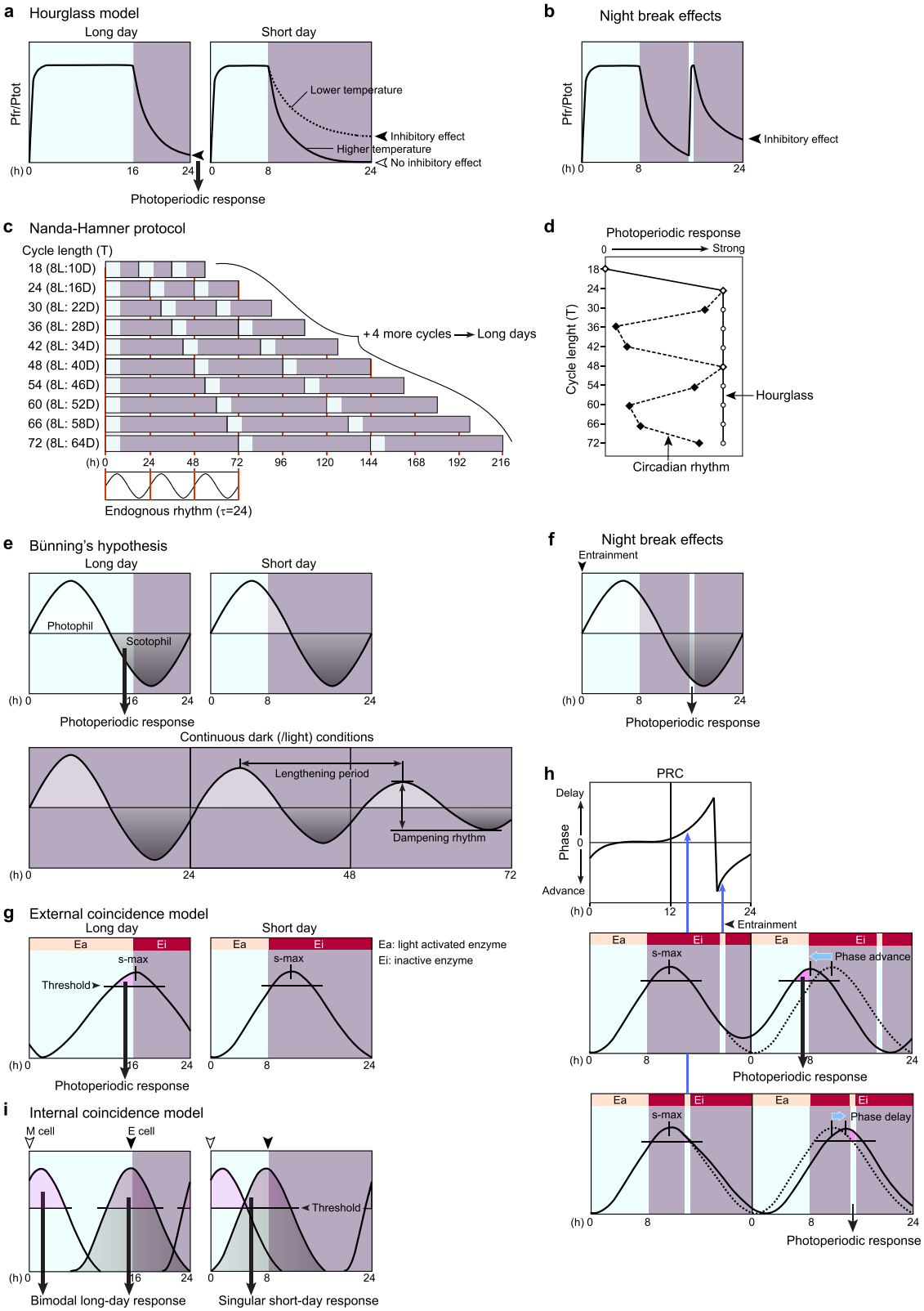
In 1936, based on the observation of circadian leaf movement of *Phaseolus* plants, Erwin Bünning was the first to propose that the endogenous daily rhythm (circadian oscillator) was the foundation for controlling photoperiodic responses, although his model was not widely known to animal chronobiologists until the Cold Spring Harbor Symposium on Biological Clocks in 1959[2,29,30]. In this model, often referred to as "Bünning's hypothesis," the photoperiodic time measurement mechanism consists of two twelve-hour-long half-cycles, upon each of which the circadian clock confers different light sensitivities (Fig. 1e)[29]. The morning half-cycle is named the photophil (light-loving) phase, while the evening one is named the scotophil (dark-loving) phase. Light in the scotophil phase induces photoperiodic responses (Fig. 1e and f). Bünning also recognized the role of light in entrainment of the circadian oscillator. The entrainment was thought to occur at dawn to accurately set the 24-hour rhythm. Based on the observation of light sensitivity changes in night break experiments in Namda-Hamner cycles, he proposed that the oscillations of these two phases become dampened, and the period length of the circadian rhythm becomes less accurate under continuous dark (or light) conditions (Fig. 1e)[29].

Based on Bünning's hypothesis and also mainly circadian phase-adjustment responses of *Drosophila pseudoobscura* eclosion rhythms induced by light pulses (in skeleton photoperiods), Colin Pittendrigh and Dorothea Minis proposed the external coincidence model in 1964[31]. In this model, photoperiodic responses are triggered only if light exposure occurs when a certain level of the circadian-regulated substrates exists (Fig. 1g). There are two differences between Bünning's hypothesis and the external coincidence model. The first one is the way of entraining the rhythms important for photoperiodic time measurement. Entrainment was thought to happen at dawn in Bünning's hypothesis (Fig. 1f). In contrast, in the external coincidence model, entrainment (both phase advance and phase delay of the rhythms) occurs at both dawn and dusk in response to internal circadian time (Fig. 1h). The external coincidence model incorporates the phases of circadian rhythms into the explanation of photoperiodic responses. The second difference is the length of the light-sensitive phase. Bünning's hypothesis postulated a 12-hour-long scotophil to induce photoperiodic responses, while the timing of the light-sensitive period (hypothesized to be defined by a threshold of diel oscillation of a substrate coinciding with a light-activated enzyme) is limited to a shorter window in the external coincidence model (Fig. 1g).

These two models also exhibit contrast in the impact of night break treatments. As the onset of light (dawn) initiates the rhythm in Bünning's hypothesis (Fig. 1f), whether the light pulse in the dark works or not depends on whether it is given during the scotophil. On the contrary, in the external coincidence model, the night break treatment would activate the light coincidence mechanism if it is given around the substrate max (s-max), but it would also induce phase change in the rhythms depending on the night-break timing applied (Fig. 1h). In actual observations, plant sensitivity to the night-break treatment varies. Short-day plants often respond to shorter night-break treatments (only a few minutes of light is enough to delay flowering, and 30 min is usually sufficient to prevent flowering in *Perilla*, *Kalanchoe*, soybean, and *Xanthium*), but many long-day plants require more prolonged exposure (hours). However, some long-day plants like barley and *Hyoscyamus niger* also responded to short (30 min or less) exposure. Others like carnation and *Brassica campestris* require hours, and the night-break effects did not saturate the flowering induction rates[3].

The internal coincidence model (two-oscillator model)

The internal coincidence model proposes that photoperiodic responses are regulated by the phase relationship between two (or more) internal circadian oscillators entrained on either dawn or dusk (Fig. 1i)[32]. A response is induced when these internal rhythms achieve a specific phase alignment under certain photoperiods. This model is more widely supported by



physiological and molecular biological observations in animals, especially insects. In this model, changes in light or dark conditions only function as Zeitgebers (cues to entrain at least two circadian oscillators), each entrained or responded to at dawn or dusk.

This model originated based on one of the theoretical interpretations of light and temperature effects on *Drosophila pseudoobscura* eclosion rhythms—two distinct oscillators exist, and one is sensitive to light while the other is sensitive to temperature changes, but both are coupled to entrain the phases of the eclosion rhythms[33]. This two-oscillator internal coincidence model was further discussed and generalized with several observations in birds, rodents, and fish[34]. In addition, a difference only in thermoperiod (durations of 23 and 13° C periods with 24-hour cycles in complete

Fig. 1 | The classical theoretical time measurement models for photoperiodism. **a** How the hourglass model works in plants. Phytochrome is a photoreceptor that controls photoperiodic flowering. During the light period, a majority of phytochrome becomes the active FR-absorbing form (Pfr). Thus, the ratio of Pfr over the total amount of phytochrome (Ptot) becomes higher. When the light is turned off, Pfr becomes the inactive R-absorbing form (Pr) through thermal reversion. This process is the night-length measurement mechanism. In this model, if Pfr exists at the end of the night, it inhibits flowering (the threshold for Pfr-dependent repression is very low). In short days, the night is long enough to revert all Pfr to Pr, releasing the repression of flowering; therefore, short-day plants can flower. The thermal reversion rate slows down under colder temperatures. In all figures throughout this review, light periods are indicated by shades of pale blue, while shades of purple indicate dark (night) periods. **b** How the night break works in the hourglass model. Night break treatment converts Pr to Pfr, so the thermal reversion process is reset by the light pulse. **c** An experimental procedure example for the Nanda-Hamner protocol. The Nanda-Hamner protocol has been utilized to investigate the potential contribution of the circadian oscillator to photoperiodic responses. This protocol is usually used to study short-day plants and animals. In this protocol, the length of the day is fixed, and only the night length was varied, typically ranging from 16 hours with increments of 4-6 hours. The example drawing shows 10 different cycle lengths (donated T), conditions of which contain 8 hours of light (L) followed by 10 hours to 64 hours of dark (D). The different cycle lengths are repeated a certain number of times (e.g., 7 times, as originally chosen by Nanda and Hamner[26]) and then returned to non-inductive long-day photoperiods. The effects of each T on photoperiodic response are assessed. The theoretical endogenous circadian rhythm, which exhibits a 24-hour rhythm (denoted τ: tau) over three days, is illustrated below. **d** Possible interpretations of the Nanda-Hamner protocol results. The results of the Nanda-Hamner protocol could indicate whether the subject's photoperiodic response is controlled by the mechanism explained by the hourglass model or regulated by the circadian oscillator. If the hourglass-type mechanism regulates the photoperiodic response, short-day organisms will exhibit photoperiodic responses when the night is equal to or longer than 16 hours in duration. Therefore, the response should be similar to the one shown by the open circles. If the response is regulated by the circadian clock, when the cycle length (T) is close to (or a multiple of) endogenous rhythms (τ, 2τ, 3τ, etc.), stronger responses can be induced (as shown by solid diamonds). This coincidence of external cycles and internal rhythms is often described as circadian resonance. **e** How Bünning's hypothesis works. When light exists in the scotophil in long-day conditions, a photoperiodic response (i.e., an inhibition of flowering in short-day plants) is induced. The endogenous oscillator (circadian oscillator) that controls these phases becomes dampened under continuous dark/light conditions, and the period length of the oscillation lengthens. Due to these changes in circadian rhythm, the circadian impact of night break treatment often became lengthened and weakened when plants were kept for a long time in continuous dark conditions. **f** How the night break works in Bünning's hypothesis. When the night break is applied during the scotophil phase, the photoperiodic response is induced. The strength of the induction is thought to often depend on the light intensity and duration. **g** How the external coincidence model works. In this model, the timing and the maxima of the substrate (s-max) are controlled by the circadian oscillator. Light controls the activity of the enzyme (Ea or Ei) that catalyzes the substrate. The photoperiodic reaction is only induced when Ea coincides above the threshold of the substrate (depicted by the pink area). **h** How the night break works in the external coincidence model. The night break treatment has two roles. One is resetting the clock depending on the timing of the light applied. The phase response curve (PRC) depicts the impact of the light break given at different times. Generally speaking, a light pulse induces a phase delay during the early night and a phase advance at the end of the night. External coincidence could occur between the presence of night break light and the adjusted substrate rhythm. Depending on the timing of the night break treatment on the first day, the phase of the theoretical substrate's rhythm is reset from its original phase (shown by dotted lines) to a new phase (solid lines) on the second day. The blue arrows from the night break timing to the PRC indicate the effects of night break treatments on the circadian entrainment. The pale blue arrows in the diagrams show the direction of changes in phases (advance or delay) of the substrate rhythms. **i** How the internal coincidence model (two-oscillator model) works. Two circadian oscillators control photoperiodic responses (i.e., a long-day specific bimodal activity). One controls activities at dawn (M cells) and the other controls activities at dusk (E cells). M and E cells are entrained only at dawn or dusk. The photoperiodic response can be induced when the amplitudes of theoretical inducers reach certain thresholds (depicted by the pick areas).

darkness) was sufficient to induce larval diapause under the short-day version of thermoperiod in the parasitic wasp *Nasonia vitripennis*. This indicates that the external coincidence mechanism with light is not essential for this photoperiodic response, and possibly supporting the presence of an internal coincidence mechanism[35].

At the molecular level in *Drosophila melanogaster*, we now know that circadian morning and evening oscillators that reside in neuropeptide Pigment Dispersing Factor (PDF)-expressing small ventral lateral neurons (s-LNv) and dorsal lateral neurons (LNd), respectively, (often called M and E cells/neurons), control morning and evening locomotor activities independently[36,37]. Both M and E cells are functionally coupled and share the same downstream neuronal targets[36–38], indicating internal coincidence could happen with M and E cells, where dawn and dusk signals are separately integrated to regulate a specific photoperiodic target. This model can explain the photoperiodic difference in crepuscular bimodal activities of many animals. Recently, the two-oscillator model has been further developed into the four-oscillator model (with two activity oscillators and two sleep oscillators) to more precisely explain *Drosophila*'s acute bimodal locomotor activities[39].

Although clear evidence of the presence of morning and evening oscillators was shown in insects and some other animals (which do not require light for survival), some plants may have a similar regulatory mechanism. Classical evidence suggests that photoperiodic flowering responses in morning glory could be regulated by time measurement mechanisms reset by two theoretical circadian oscillators, each entrained by either dawn or dusk[40]. Based on a circadian gene reporter assay and the fine-scale transcriptional analysis of various clock genes under several photoperiods in *Arabidopsis*, it is known that the plant circadian oscillator is dominantly reset at dawn (light onset), although the timing of dusk also influences phases of some clock and clock-controlled genes[41,42]. In morning glory, it has been shown that the phases of the expression peaks of photoperiodic flowering florigen genes [*Pharbitis nil FLOWERING LOCUS T 1* (Pn *FT1*) and Pn *FT2*] strictly follow the timing of dark onset[43], suggesting the presence of an oscillatory mechanism solely entrained by dusk. The phases of other circadian clock output genes [*Pharbitis nil CONSTANS* (Pn*CO*) and Pn*CAB2*] in the same species are controlled by light and dark transitions[43]. Interestingly, the phases of *Pharbitis nil* homologs of morning and evening core clock genes (*PnLHY* and *PnTOC1*) and *Arabidopsis* photoperiodic flowering genes (*PnFKF1* and *PnCDF2*) are all entrained by the dawn, dark-to-light transition[44]. Does this support the possibility of two circadian oscillators resetting at dawn or dusk? Of course, we cannot rule out the possibility (as Hayama et al. also discussed[44]), that the *early flowering 3* (*elf3*) mutant, which shows continuous light-specific arrhythmicity, can follow the timing of dark onset to control circadian phases[41,45]. Similar to animals, plants have tissue-specific variations of circadian oscillators[46–48]. Therefore, this phenomenon could be explained by variation in the circadian light input pathway (i.e., lack of *ELF3* expression) in the cells where *Pharbitis nil* florigen genes are induced. With some variations, the single oscillators could explain photoperiodic phenomena that fit internal coincidence. Although not as extensively explored, the internal coincidence model provides a plausible explanation in cases where external light cues seem less directly involved in triggering the response.

Molecular coincidence timers in photoperiodic flowering

In contrast to the large numbers of physiological analyses performed in various plant species[3,5], molecular insights into photoperiodic time measurement have been limited to only a few species. We have learned the most about time measurement mechanisms from the photoperiodic flowering response of a facultative long-day plant, *Arabidopsis thaliana*. The final output of the photoperiodic time measurement mechanism for flowering is long-day specific expression of the florigen *FT* gene[49,50]. There are at least

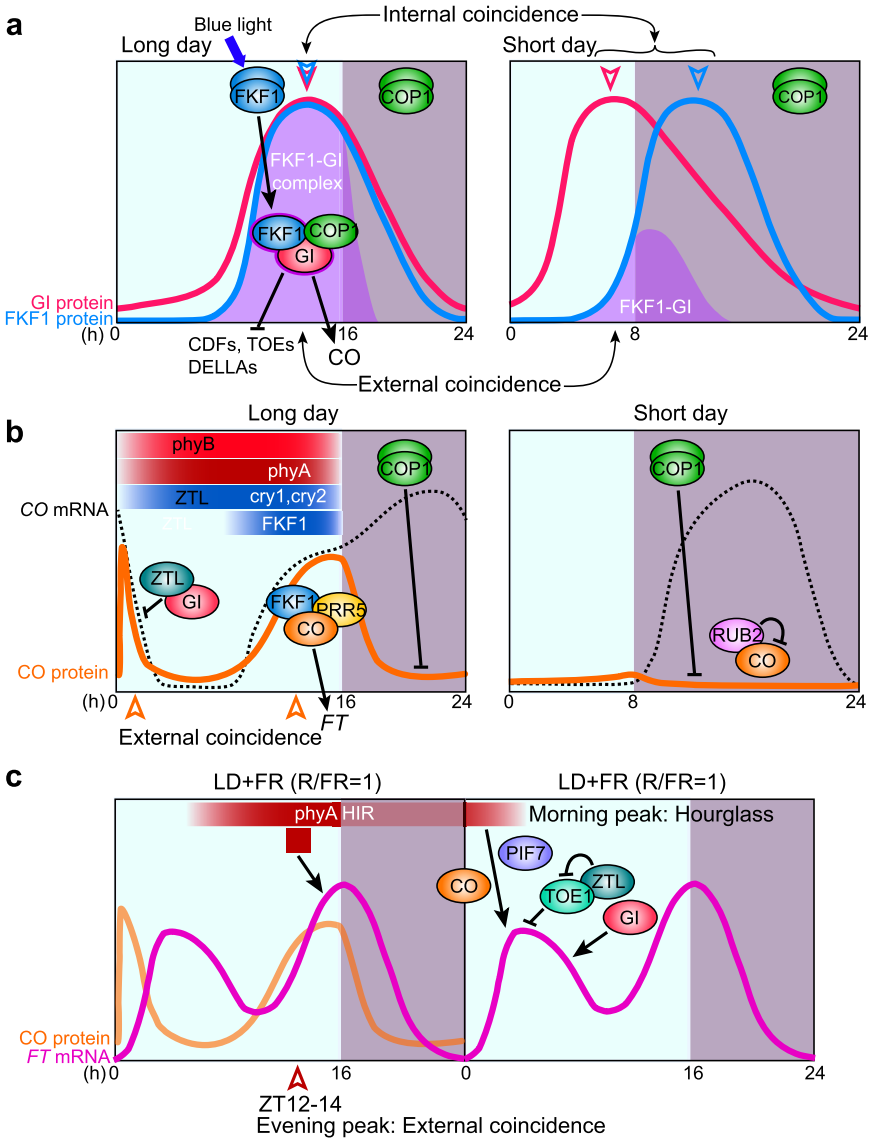


Fig. 2 | Time measurement mechanisms in the photoperiodic flowering pathway in *Arabidopsis*. **a** The coincidence mechanisms control the formation of the FKF1-GI complex. The timing of FKF1 and GI protein expression coincides in long days but not in short days. With blue-light dependent FKF1 binding to GI, the FKF1-GI complex formation (shown in purple) is maximized in long-day afternoon. Thus, this regulation contains aspects that fit the internal and external coincidence models. In the long-day afternoon, the FKF1-GI complex degrades CDF and DELLA proteins and represses TOE1 function and simultaneously stabilizes CO protein. FKF1 can form a homodimer, but the one in the FKF1-GI complex is a monomer. FKF1 monomer also binds to COP1 monomer to prevent COP1 homodimerization, which requires its E3 ubiquitin ligase activity. COP1 degrades many transcription factors, including CO during the nighttime. **b** The external coincidence mechanism controls the stabilization of CO protein in long days. The diurnal oscillation patterns of *CO* are regulated by the circadian clock. *CO* expression occurs from the afternoon to evening in long days and at night in short days. Once CO protein is synthesized under light in long-day afternoons, CO protein stability is regulated by red, blue, and far-red light perceived by phyA, phyB, cry1, cry2, ZTL, and FKF1 proteins. The presence of each photoreceptor in long days is shown by different color bars corresponding to each absorbance. phyB and ZTL are negative regulators of CO, while the rest are positive regulators. FKF1 and PRR5 bind to CO in the long-day afternoon to stabilize CO, which in turn induces the expression of *FT*, leading to floral induction. The external coincidence between light signaling and CO protein only occurs in long days. Note that there is a short window at the beginning of the day in which CO is also stabilized. **c** The mechanism for generating bimodal expression of *FT*. In long days in which the red/far-red light ratio (R/FR) is adjusted to approximately 1 to mimic sunlight (denoted LD + FR), *FT* expression shows a bimodal pattern with morning and evening peaks. The evening peak is regulated by the external coincidence mechanism between CO and light signaling discussed in Fig.2b, and the morning peak is controlled by the phyA-mediated HIR (High Irradiance Response). In addition to CO, which is required for the morning *FT* induction, other factors (PIF7, ZTL, TOE1, and GI) are also involved in this regulation. CO protein is more stabilized in the morning in LD + FR than in LD.

two layers of light coincidence mechanisms in the photoperiodic flowering pathway. One regulates FKF1-GI complex formation, and the other CO protein stability (Fig. 2a, b)[8]. The former influences the latter, so they are connected, but light information is integrated mainly through the different photoreceptors. In addition, recent work has suggested that the hourglass-type mechanism also exists to control photoperiodic flowering in *Arabidopsis* (Fig. 2c).

External and internal coincidence mechanisms regulate FKF1-GI complex in long-day plants

The timing of flowering is a critical determinant of vegetative growth and reproductive success (including fruit and seed yields), particularly in annual plants, which include most crop species. Plants are adapted to specific locations where certain day length changes are anticipated during the growth seasons. This intrinsic ability to precisely measure photoperiod

changes has been problematic when we grow plants at different locations with different latitudes. Domestication is closely linked to photoperiodism as one of the important parts of domestication is suppressing photoperiodic flowering[51,52]

In *Arabidopsis*, the components of photoperiodic time measurement found originally were the causal genes of flowering mutants. The *gigantea* (*gi*) mutant was one of the earliest identified mutants before the molecular genetic era[53]. It was recognized for its vigorous vegetative growth (a "supervital" mutant), and it turned out that its growth phenotype was caused by late flowering[54,55]. The *flavin-binding, kelch repeat, f-box 1* (*fkf1*) mutant also displays similar late flowering, especially under long-day conditions[56], underscoring the importance of these genes in photoperiodic flowering regulation. FKF1 belongs to a small gene family and comprises three distinct functional domains: N-terminal LOV (Light, Oxygen or Voltage), F-box, and C-terminal Kelch repeat. The LOV domain is known as a blue light-sensing module, also found in the blue-light photoreceptors phototropin and aureochrome, and plays essential roles in various light-mediated processes, such as phototropism, stomatal opening, chloroplast movements for phototropins, and photomorphogenesis of algae for aureochromes[57,58]. In chronobiology, it has been well known that the LOV domain-containing proteins WHITE COLLAR-1 (WC-1) and VIVID (VVD) play important roles in the entrainment of the fungus (*Neurospora crassa*) circadian clock[59,60], indicating the commonality of usage of blue light to sense surrounding diel environmental light conditions. Similar to the other LOV domains, the FKF1 LOV domain binds to flavin mononucleotide (FMN) to absorb blue light and exhibits an unusually long photocycle, maintaining a light-activated state for days in vitro[61]. This blue-light perception is crucial for FKF1's function as an E3 ubiquitin ligase that targets CYCLING DOF FACTORs (CDFs) and DELLA proteins for degradation, both of which are negative regulators of flowering[62–64].

Importantly, FKF1 interacts with GI through its LOV domain in a blue light-dependent manner, and this interaction is essential for FKF1 function[65]. Because the circadian expression of both *FKF1* and *GI* peaks at the end of the day under long-day conditions, their light-induced interaction mainly happens in the long-day afternoon, and this mechanism fits the external coincidence model well (Fig. 2a)[8,66]. Interestingly, under short-day conditions, the peak expressions of FKF1 and GI proteins are desynchronized, indicating the presence of an additional layer of regulation through internal coincidence mechanisms that modulate their expression phases independently depending on photoperiods[66]. This misalignment of peak timing reduces the chance of light-induced FKF1-GI complex formation. Thus, the photoperiodic FKF1-GI formation mechanism operates with regulations that fit both the external and internal coincidence models (Fig. 2a).

In the afternoon of long days, FKF1 also stabilizes CO protein, a key activator of *FT*, by directly binding through the LOV domain in response to blue light[67], and additionally interacts with PSEUDO RESPONSE REGULATOR 5 (PRR5)[68], which also physically contributes to CO stabilization[69]. One of the mechanisms by which FKF1 stabilizes CO is directly inhibiting CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) function, an E3 ligase responsible for CO degradation, by capturing a monomer of COP1[70]. This is a common molecular strategy for many photoreceptors to control photomorphogenesis. Once phyA, phyB, cryptochrome 1 (cry1), cry2, and UV RESISTANCE LOCUS 8 (UVR8) are activated by absorbing different wavelengths of light, they all physically suppress the COP1 function to prevent either COP1 dimerization or complex formation with SUPPRESSOR OF PHYA-105 1 (SPA1)[71–75]. Although FKF1 can form homo- and heterodimers within the ZTL/FKF1/LKP2 protein family, its interaction with GI is specifically as a monomer[76–78]. It is unclear whether light regulates FKF1 monomerization or modulates the interaction between its LOV and Kelch domains, although similar LOV-Kelch interactions are observed in ZEITLUPE (ZTL)[77]. Light-induced monomer/dimer exchange and interdomain interactions (i.e., LOV vs Kelch repeats) are also likely common structure changes observed in the LOV domains of LOV domain-containing photoreceptor proteins[79–81].

This FKF1-GI coincidence mechanism for photoperiodic time measurement seems evolutionarily conserved in land plants. In *Marchantia polymorpha*, a basal land plant, far-red-light-enriched long-day conditions induce the formation of gametangiophores (reproductive organs containing either male or female gametophytes). Transcripts of *MpFKF* and *MpGI* show diel oscillation with peaks coinciding at the end of long days, and MpFKF and MpGI proteins form a complex like their *Arabidopsis* counterparts. In addition, they are essential for gametangiophore formation under long-day conditions[82], although how they control gametangiophore formation is currently unknown. Unlike *Arabidopsis*, *Marchantia* displays weaker diel transcriptional oscillations under continuous light conditions, and *MpGI* lacks rhythmic expression in such conditions[82,83], reflecting a simpler regulatory architecture with fewer redundant components in the circadian oscillator. Notably, the *Marchantia* genome lacks a homolog of the morning clock genes but retains homologs of other core clock components such as *REVEILLE* (*RVE*), *TIMING OF CAB2 EXPRESSION 1* (*TOC1*), *PRR*s, *GI*, and the evening complex (although all of them are single copies)[83]. Despite its simplified signal transduction system, *Marchantia* contains two *DOF* genes, including a homolog of *CDF*[84], possibly suggesting an ancestral role of the FKF1-GI-CDF module in photoperiodic time measurement in bryophytes. Thus, MpGI and MpFKF (the circadian clock protein and the 1st order clock-regulated photoreceptor) are also required for gametangiophore formation in *Marchantia*, suggesting the requirement of both light and the circadian clock for photoperiod measurement. However, the recent study of the circadian clock gene mutants as well as classical Nanda-Hamner protocol experiments indicated that the duration of light periods rather than circadian timing is critical for photoperiod measurement in *Marchantia*[85], indicating the hourglass-type measurement mechanism might be sufficient for time measurement in *Marchantia*.

The FKF1-GI complex has been repurposed during land plant evolution to regulate diverse photoperiodic responses. In short-day plants like soybean, the soybean GmFKF1 and GmGI (also known as E2) form a complex and interact with J (a homolog of ELF3) protein to promote its degradation under long-day conditions. Since J represses *E1* (a repressor of *GmFT2* and *GmFT5*), this degradation pathway activates flowering-related genes[86]. In contrast, in wild potato, which tuberizes under short days, domestication selected for a mutation in *StCDF1* that renders it constitutively stable, decoupling tuberization from day length[87]. StFKF1 and StGI promote tuberization by targeting StCDF1 for degradation[87]. In addition, a recent work demonstrated that leaf senescence (or maturity), which tightly links the onset of tuberization and affects tuber yields and quality, is regulated by StCDF1[88], revealing their conserved yet flexible roles in diverse seasonal developmental processes. Together, these findings highlight the FKF1-GI complex as one of the core photoperiodic time measurement modules, shaped often by both external and internal coincidence mechanisms, and co-opted for various adaptive functions in seasonal responses across land plant evolution.

External coincidence mechanism to stabilize the CO protein

The targets of the FKF1-GI complex-mediated photoperiodic time measurement mechanism are the *CO* transcript and protein expression patterns. Various reports describe the transcriptional and posttranslational regulation of CO. Here, I will focus on the mechanisms related to seasonal time measurement. CO interacts with many proteins, including several transcription factors, which co-activate or repress *FT* transcription (see more comprehensive details of CO-interacting proteins in recent reviews[8,16]).

Both classes of blue-light photoreceptors (crys and the ZTL/FKF1/LKP2 family) and red/far-red light photoreceptors (phyA and phyB) are involved in the regulation of CO protein stabilization happening at the end of long days (around ZT12-ZT16). They are not involved in short days[89], which comprise another layer of the external coincidence mechanisms (Fig. 2b). phyB and ZTL destabilize CO from morning to early afternoon, while the rest of the photoreceptors (crys, FKF1, and phyA) stabilize CO in the afternoon[89,90]. As all photoreceptors except for FKF1 are expressed throughout the day, it has been proposed that the FKF1-GI complex

discussed above, together with PRR5, conveys the daily time information in this regulation. A homolog of FKF1, ZTL, also plays an antagonistic role with FKF1. CO is degraded with the ZTL-GI complex in the morning[90]. GI, which has molecular chaperone activity, can stabilize both FKF1 and ZTL[91].

Even though CO protein levels are kept very low during the night in short days by being actively degraded by the COP1/SPA1 complex[89,92,93], there might be an additional mechanism to ensure that the activity of CO is repressed during nighttime. That would ensure that *FT* won't be induced during the night in short days under more natural light conditions, including UV-B spectrum[94].

A recent report showed that CO also provides feedback to adjust circadian clock gene expression[95]. CO associates with the promoter regions (mainly where G-box motifs exist) of many circadian clock genes and affects the gene expression profiles of some. CO likely works as a protein complex with PRR5 and ELONGATED HYPOCOTYL 5 (HY5). *CO* overexpression caused short circadian rhythms, indicating that *CO* could contribute to phase advance under a light-dark cycle. *CO* is mainly expressed in vascular tissues and shoot apical meristems (although CO expressed only in phloem is functionally relevant for flowering regulation)[96,97], and stabilized in early morning and late afternoon, particularly in long days[89]. Therefore, CO is not likely to be a general regulator for the circadian clock. Rather, CO's function may specifically affect photoperiodic time measurement. CO may provide positive feedback for photoperiodic flowering by slightly advancing the phases of the circadian oscillator or particular genes, such as *FKF1*, *GI* and *PRR5* (as de Los Reyes et al. also discussed[95]), particularly in CO-expressing phloem companion cells to expand the time windows of external coincidence happening.

Hourglass mechanisms to induce morning *FT*

In addition to the photoperiodic time measurement mechanisms discussed above, a recent finding indicates the presence of a mechanism that fits more with the idea of the hourglass model within the photoperiodic time measurement mechanisms in *Arabidopsis*. The expression pattern of *FT* in the plants grown under natural long days shows a bimodal pattern with peaks in the morning (ZT4) and evening (ZT16), which is different from that grown in the lab[98]. The difference in *FT* profiles was caused by a difference in the red/far-red light (R/FR) ratio—the R/FR ratio equals approximately 1 in nature, while the ratio was more than 2 in the lab— and diurnal temperature changes. Adjusting the R/FR ratio to the same as the natural conditions was crucial to induce *FT* in the morning (Fig. 2c). Importantly, these bimodal expression patterns of *FT* are unique to photoperiods longer than 14 hours[99], indicating that a photoperiodic time measurement mechanism can generate this unique pattern. Interestingly, even in animals (in the *pars tuberalis* in the sheep pituitary), an early long-photoperiod responsive gene, *Eyes absent 3* (*Eya3*), which is involved in the induction of thyroid-stimulating hormone *β* (*TSHβ*) transcription—an essential hormone for seasonal gonad maturation—shows a similar long-day specific bimodal expression pattern with peaks in the morning and at the end of the day[100,101]. Therefore, potentially, this potentially suggests that animals may have also evolved a similar network architecture to measure long-day photoperiods.

To examine whether there is a light-sensitive phase for the morning *FT* induction within a day, circadian gating experiments using the R/FR ratio adjustments with different timing and duration were performed. The results indicated that the duration of the light treatment rather than the timing was important to determine the levels of morning *FT*, although some time-dependent sensitivity differences existed[99]. Because this response demonstrated clear far-red-light (FR)-duration dependency and also regulation by phyA [a photoreceptor controlling FR-high irradiation response (HIR)[102]], we concluded that FR-HIR controls the level of morning *FT* expression. Although it is different from the originally proposed hourglass mechanism based on R/FR reversibility and thermal reversion of phyB (Fig. 1a), the induction levels of morning *FT* expression can be interpreted as an hourglass mechanism because the morning *FT* levels highly correlate with the duration (but not so much the timing) of FR exposure perceived by phyA. phyA has a very slow thermal reversion rate[103], so that FR-activated phyA can structurally keep the light information throughout the night in long days. On the contrary, the afternoon peak level was purely controlled by the external coincidence mechanism that we already knew.

Classical physiological analyses in various long-day plants showed that they often required longer exposure of light. FR-enriched light was particularly effective for early flowering, and later these responses were considered to be FR-HIR responses controlled by phyA[5,104]. However, the *Arabidopsis phyA* mutant grown under white light (provided by cool white fluorescence tubes) in long days resembled wild-type plants, and it showed a delayed flowering phenotype only under short-day conditions with additional 8 hours of low-fluence FR enriched light extension[105], indicating the contribution of phyA on photoperiodic flowering regulation is limited to specific light conditions in *Arabidopsis*. Importantly, finding the bimodal expression pattern of *FT* and underlying FR-HIR mediated morning *FT* induction under natural R/FR conditions clearly demonstrated that *Arabidopsis* also controls flowering using phyA-mediated FR-HIR in sunlight, and it also has another mechanism that fits the external coincidence model (Fig. 2c). The previous observation of the lack of flowering phenotype in the *phyA* mutant under white light[105] is likely due to the scarcity of FR spectrum when using fluorescence tubes as a white light source. Although morning *FT* is controlled by FR-HIR, CO is still required, as *FT* expression is depressed throughout the day in the *co* mutant[98]. CO protein stability increases under natural R/FR conditions, and CO protein exists in the morning and afternoon, when *FT* expression peaks, although the presence of CO protein without the activation of the FR-HIR mechanism is not sufficient to induce *FT* in the morning[98,99]. How phyA mechanistically induces *FT* in the morning still remains largely unknown. However, several known photoperiodic flowering regulators, including GI, ZTL, and TARGET OF EAT 1 (TOE1), are involved in the regulation (Fig. 2c)[98,99,106]. GI and ZTL physically regulate CO stability, and TOE1 directly affects CO activity, indicating that one of the mechanisms of morning *FT* induction might be the direct control of CO protein stability and activity. In short days, CO protein is degraded throughout the day[89]. This photoperiod-dependent CO protein stability regulation might still be the basis of long-day specific induction of *FT* under natural light conditions. Interestingly, blue-light receptors, FKF1 and cry2, are more specific in regulating the afternoon peak of *FT*[98].

Similar to the FKF1-GI module, the involvement of phytochrome-mediated FR-HIR in the photoperiodic response was discovered in *Marchantia*. There is only one phytochrome (Mpphy) and PHYTOCHROME INTERACTING FACTOR (MpPIF) in *Marchantia*[107]. The only phytochrome possesses characteristics of both phyA and phyB in *Arabidopsis*, and the phytochrome FR HIR responses to induce gametangiophore formation[108]. Land plant phytochrome originates from a common ancestor in charophytic algae (freshwater green algae). The basal lineage of land plants, including liverwort (*Marchantia*), contains only one phytochrome[107]. Indeed, the expression profiles of *FT* homologs in other long-day plants, such as wheat, Medicago, strawberry, and hybrid aspen, also demonstrated similar bimodal expression patterns[109–112]. In addition, in the short-day plant soybean, the expression pattern of *E1* (the most important repressor of soybean florigen), shows a similar long-day specific bimodal expression pattern[113], indicating that the same mechanism was used to express the repressor in long days to make soybean a short-day plant. These observations indicate that the same hourglass (phyA FR-HIR) and coincidence (FKF1-GI module) mechanisms are likely utilized for these plants to respond to long-day conditions.

Twilight influence on biomass and flowering time

Recently, in another attempt to study more natural responses of plants growing at different latitudes in spring, the effects of simulated twilight at dawn and dusk on photoperiodic responses were studied under lab long-day conditions[114]. Introducing various durations of gradual changes in light intensity at dawn and dusk into growth conditions (without changing the total daily light energy) altered biomass and flowering time independently, compared to plants grown under a standard square-shaped light regime. For example, a 60-minute-long twilight treatment increased the median biomass

of 25-day-old *Arabidopsis* plants by 16%, while a 90-minute-long twilight had no significant effect, compared to control plants grown without twilight. Independently, flowering time was more delayed under the 90-minute-long twilight treatment than under the 60-minute-long (or shorter) ones, indicating that twilight duration affects plant development differently through multiple pathways. To modulate flowering time in response to twilight changes, known photoreceptors (phyB, phyD, phyE, and cry2), certain circadian clock components (CCA1, LHY, and GI), and FT are involved, further confirming the significance of light/circadian control of florigen expression under more natural conditions. Different photoreceptors (phys and crys), which have varying sensitivities to light intensity[115], are involved in the entrainment of the circadian clock[116]. Thus, intriguing questions are how and when plants reset their clock in response to gradually changing light, and how various photoreceptors coordinate the resetting to influence the expression patterns of clock and clock-associated output genes. In analyzing the effects of twilight on long-day-induced flowering, it would be informative to analyze how twilight conditions affect the bimodal expression pattern of *FT* in wild-type and clock mutant plants. The result may help understand the roles of the clock genes in twilight sensing. Answering these questions will further elucidate how plants recognize the beginning and the end of the day and how the changes in twilight regulate photoperiodic responses in natural environments.

Night break effects at a molecular level

Performing night break experiments has been instrumental in determining critical day length in plant photoperiodism investigations. Although there are limited reports regarding the molecular mechanisms underlying the night break effects, here I introduce what we have learned at the molecular level.

The effects of night breaks (1 hour of either red, far-red, or blue light exposure given around ZT16 in short days) on flowering time were studied in *Arabidopsis* photomorphogenesis mutants[117]. Far-red light exposure was the most effective on the acceleration of flowering, although the flowering time ended up being between long-day-grown and short-day-grown plants. Also, phytochrome chromophore biosynthesis mutants did not exhibit much response to the night break treatments. This observation is now understandable considering the contribution of phyA HIR for morning *FT* expression and phyA-dependent stabilization of CO protein around that time (Fig. 2b and c).

In wheat, a long-day plant, the night break effect was studied for flowering induction at the molecular level[118]. Night break treatments (1 hour of white light) were given at different times during the night in short days. Although the night break given in the middle of the night was the most effective, all night break treatments given at different times of night induced similar flowering earlier than in short day-grown plants in wheat. Unlike the classical example in short-day plants, repetitive night break treatments were more effective than the single night break treatment. The treatment immediately induced the expression of *PHOTOPERIOD 1* (*PPD1*) gene, a key positive regulator of wheat photoperiodic flowering that induces *FT1* expression. This induction is controlled by phyB and phyC photoreceptors, and the light effects are only partly R/FR photoreversible. Interestingly, the night break treatment that induced *PPD1* and *FT1* did not change the phases of the circadian clock genes. These observations indicate that the night break mechanism in wheat fits Bünning's hypothesis better than the external coincidence model.

The classical textbook examples of the night break effects—a single short-duration (minutes) treatment completely inhibits short-day induced flowering and the effect is R/FR reversible—were mainly obtained from short-day plants[5]. At least we have learned the molecular mechanisms of night-break effects in a short-day rice plant. Although repetitive 10-min light pulses per day were required to see a change in rice flowering time, the single exposure was enough to see the gene expression changes[119]. The night break clearly shows the inhibition of *FT*-homolog in rice *Heading day 3a* (*Hd3a*) expression, and the inhibitory effects show obvious time-dependency. The light given in the middle of the night (around ZT17 in 10-h light/14-h dark short days) is the most effective and this light signal is perceived mainly by phyB (with R/FR photoreversibility)[119,120]. This treatment also caused slight phase delays in some circadian clock-regulated genes[119], fitting the external coincidence model. At the beginning of our understanding of the photoperiodic flowering mechanism in rice, its similarity to the one in *Arabidopsis* was discussed. Soon, identification of various rice-specific mechanisms[121] lead to recognition that the photoperiodic time measurement mechanism in rice is different from the one in *Arabidopsis*[17].

Based on extensive night break experiments using either blue or red light pulses, rice has two light sensitive phases—one for blue light and the other for red light— to induce rice specific flowering activators and repressors, respectively (Fig. 3a)[122]. Blue light induces the expression of *Early heading date 1* (*Ehd1*) in an *Oryza sativa GI* (*OsGI*)-dependent manner. Blue light sensitivity spans a wider time window than red-light sensitivity. Red light absorbed mainly by the phyB photoreceptor induces *Ghd7* (*Grain number, plant height, and heading date 7*) expression, and the peak timing of red-light sensitivity coincides with blue-light sensitivity in long days but happens in the middle of the night in short days (Fig. 3a). Because Ghd7 protein represses the expression of *Ehd1* transcription, it has been proposed that *Ehd1* expression level is lower in long day because of the presence of Ghd7-dependent suppression (Fig. 3a, b). A recent work showed that phyB physically binds to Ghd7 protein and stabilizes it in long days[123]. Ghd7 stability is negatively regulated through a proteasome-dependent degradation mechanism by direct binding of OsGI, and phyB physically competes with OsGI binding to Ghd7, stabilizing it[123]. Ghd7 protein is unstable throughout the entire day in short days, releasing Ghd7-dependent repression. This facilitates the induction of *Ehd1*, a rice-specific transcriptional activator of florigens (*Hd3a* and *RICE FLOWERING LOCUS T 1: RFT1*) in short days (Fig. 3b)[122,124]. Thus, the photoperiodic time measurement mechanism is composed of two external coincidence regulations, which are independently controlled by red and blue light, respectively, but also have a hierarchal relationship (i.e., Ghd7 repressing *Ehd1*), which is altered by the internal coincidence mechanism of controlling the phases of these two light-induced regulations (Fig. 3a). Because of this hierarchy, the night break effect is mainly controlled by red light, which activates phyB-specific *Ghd7* induction and the Ghd7 stabilization pathway (Fig. 3c). It is noteworthy that the strength of night break effects correlates with red-light intensities[120].

In the rice photoperiodic flowering pathway, the *CO* homolog, *Hd1*, also plays a critical role as a direct regulator of *Hd3a* expression to control heading dates[125,126]. However, unlike CO, Hd1 protein is stable even in the dark, but changes its function from an activator to a repressor of florigen expression depending on photoperiod[127]. This photoperiodic conversion of Hd1 function is controlled by Ghd7. In long days, Ghd7 physically binds to Hd1 to convert the Hd1 complex into a repressor of both *Ehd1* and *Hd3a*[128]. Although the clear coincidence mechanism has not been directly described as related to Hd1-related regulation, to precisely control flowering time under specific photoperiods, the balance (timing of expression and levels and their interactions) among one transcriptional repressor (Ghd7) and two activators (Ehd1 and Hd1) of florigen (*Hd3a* and *RFT*) is crucial[129].

A metabolic daylength measurement mechanism that controls photoperiodic growth

Although photoperiodic changes in florigen homolog levels control various seasonal developments in many plant species[10,14,15,130], recent work revealed the presence of a different seasonal time measurement mechanism. The interplay between photosynthesis-derived sucrose/sugar metabolic signaling and the circadian clock regulates photoperiodic vegetative growth in long days and short days in *Arabidopsis*[131,132]. As some plants do not show photoperiodic flowering responses and the *CO-FT* photoperiodic flowering module is not mainly active in short days in *Arabidopsis*, the Joshua Gendron group has sought other photoperiodic mechanisms that possibly exist in short days by identifying genes with short-day specific expression from the comparison of transcriptome datasets of long-day and short-day grown plants[131]. The mutant of one of these genes, *PHLOEM PROTEIN 2 -A13* (*PP2-A13*), showed short-day specific defects in growth and flowering (i.e., lower biomass, leaf senescence initiation before bolting, bolting

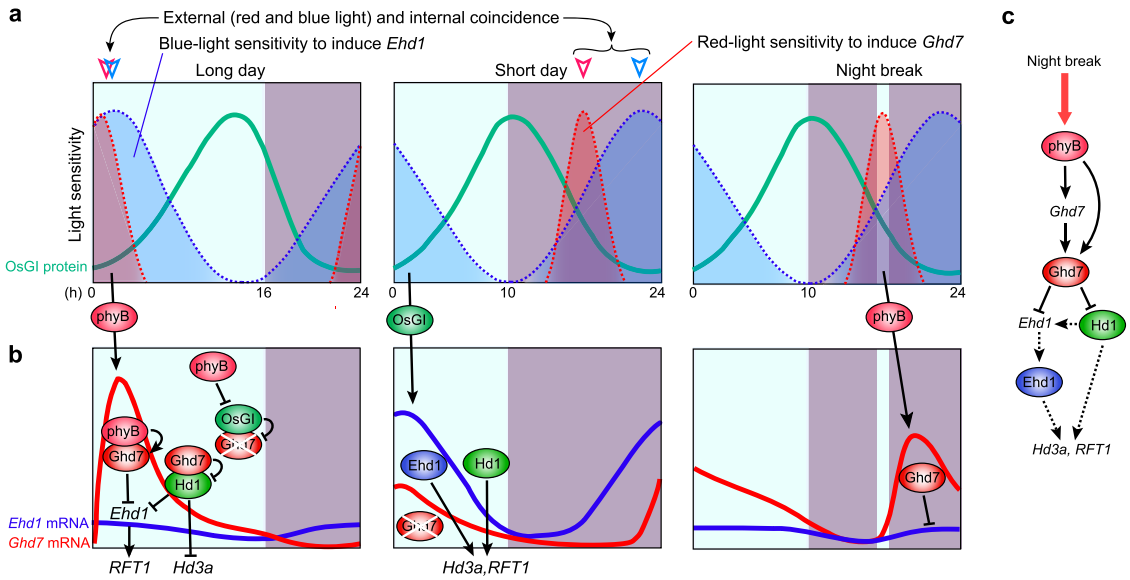


Fig. 3 | The photoperiodic time measurement mechanisms and night break effects in rice. a Photoperiodic flowering in rice is regulated by red and blue light. The light sensitivity for red and blue light oscillates throughout the day. Red light perceived by phyB induces *Ghd7* expression, while blue light induces *Ehd1* expression in an OsGI-dependent manner. The phases of maximum sensitivity of red and blue light coincide in long days, but not in short days. These mechanisms have features that fit both external and internal coincidence models. **b** At the molecular level, phyB signaling forms a feedforward loop in long days to induce *Ghd7* and stabilize Ghd7 protein by physical interaction in the morning. Ghd7 represses *Edh1* expression; therefore, even though blue light is present, the *Edh1* level is lower in long days than in short days. Ghd7 binds to Hd1 protein to convert Hd1 to a repressor of *Edh1* and *Hd3a*. OsGI binds to the Ghd7 protein to degrade it in the afternoon. In short days, the red-light sensitive window for *Ghd7* induction is in the middle of night, so the *Ghd7* level stays low. In addition, Ghd7 protein is actively degraded in short days. Without Ghd7, blue light can induce *Ehd1* in the morning. Both Ehd1 and Hd1 induce the expression of florigen genes (*Hd3a* and *RFT*). In short days, the night break treatment in the middle of the night induces *Ghd7* to repress flowering. (Note that the precise diel expression patterns of *Ghd7*and *Ehd1* vary among reports, possibly due to usage of different cultivars and growth conditions, and their patterns in the night break panel are predicted ones). **c** The summary of the night break cascade. PhyB-mediated Ghd7 activation is the key to the night break treatment.

delay, etc.)[131]. The expression pattern of *PP2-A13* shows a bimodal expression profile with a unique short-day specific early night peak (Fig. 4). The *PP2-A13* expression is repressed by photosynthesis-derived sucrose signaling during the long-day afternoon (around ZT8-16), and this photosynthesis/sucrose-dependent repression of *PP2-A13* is negatively gated by the circadian clock during the subjective night, so that sensitivity to photosynthesis-derived sucrose metabolic signaling is higher during the ZT8-16 time window than near dusk[131]. In this pathway, light information (its quality and quantity) is integrated through photosynthesis activity, and the presence of sucrose (or its derived sugars) is critical for controlling the transcription of *PP2-A13*, although the mechanisms of this sucrose signaling and *PP2-A13*-dependent growth regulation remain elusive.

The Gendron group further found that a similar photosynthesis/sucrose metabolic pathway also regulates vegetative growth in long days[132]. The gene *MYO-INOSITOL-1-PHOSPHATE SYNTHASE 1* (*MIPS1*) shows a long-day specific bimodal expression pattern with morning (at ZT4) and dusk (at ZT16) peaks (Fig. 4), and the *mips1* mutant grows slowly only under long-day conditions. Photosynthesis-derived sucrose signaling induces *MIPS1* expression in the long-day afternoon, and this regulation is independent of the photoperiodic flowering mechanism. Although the mechanism of *MIPS1* action in long-day specific growth regulation is unknown, it is proposed that the long-day afternoon expression of *MIPS1* is crucial for growth activation. Importantly, exposing plants to weak light, which is enough to entrain the circadian clock but lower than the photosynthesis compensation point, failed to induce *MIPS1* expression in the long-day afternoon. Therefore, Wang et al. proposed that plants may measure the duration of photosynthesis activity above the compensation point to control photoperiodic growth[132].

The photoperiodic regulation of both *PP2-A13* and *MIPS1* expression shares various similarities. Both are regulated by photosynthesis-derived sucrose metabolic signaling. The crucial changes in gene expression for both genes for the regulation of plant growth happen in the ZT8-16 window. Because of the circadian gating mechanism, this window has higher sensitivity to sucrose-mediated signaling for photoperiodic growth regulation than at other times of the day. Although it is merely one possibility, based on their findings, the photoperiodic expression of these genes might be controlled by the external coincidence mechanism between light signals processed through photosynthesis activity and clock-controlled sucrose/sugar signal sensitivity (Fig. 4). In this regulation, sucrose signaling could induce *MIPS1*, while it could repress *PP2-A13* (Fig. 4).

It has been well-characterized that plants precisely adjust starch degradation kinetics during nighttime through the circadian clock-associated mechanism, depending on photoperiods[42,133–136], so that they won't starve from a lack of sugar at the end of nights of varying lengths. Although starch accumulation and degradation kinetics show photoperiod-specific distinct patterns (Fig. 4), starch by itself doesn't seem to be essential for photoperiodic growth regulation, because photosynthesis/sucrose signaling still regulates *MIPS1* and *PP2-A13* in the starchless mutant (although the expression patterns of both genes are affected). In addition to photoperiodic adjustment of starch degradation rates, photosynthesis-derived sugar signals are known to regulate several developmental transitions. The photosynthetic glucose adjusts the timing of juvenile-to-adult leaf developmental transition (known as heteroblasty), which may affect the overall biomass of plants, by partly controlling the expression of microRNA156 (miR156)[137]. Trehalose-6-phosphate, a signaling intermediate of a sugar metabolic pathway[138], is involved in flowering regulation as an important factor for *FT* induction in leaves and miR156 repression at the shoot apex[139]. It is of great interest to investigate whether the photoperiodic growth mechanisms discussed here share metabolic signaling networks with either starch degradation kinetic regulation or other sugar-controlled developmental regulation. Also, because both *MIPS1* and *PP2-A13* genes showed photoperiod-specific bimodal expression patterns, similar to the *FT* bimodal expression pattern in long days, these genes may also be controlled by at least two light-dependent mechanisms, which may fit different day-length measurement mechanisms

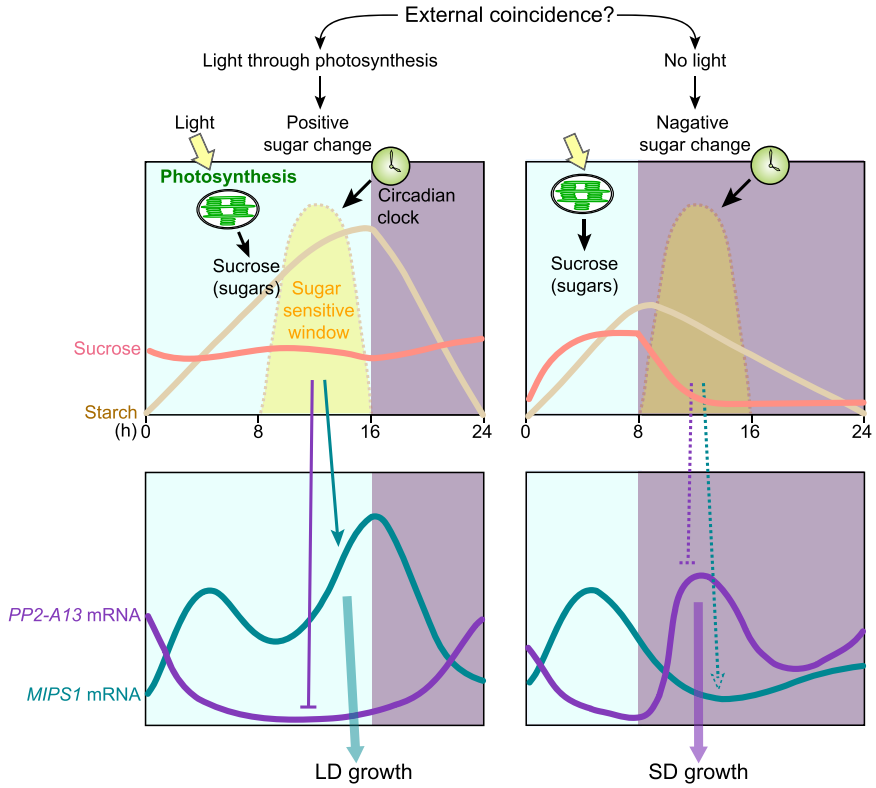


Fig. 4 | A possible model for the metabolic day length measurement mechanism for photoperiodic growth regulation. This diagram depicts one of the possibilities that may explain the expression patterns of *PP2-A13* and *MIPS1* genes in long days and short days. In this mechanism, the afternoon light information captured by photosynthesis (above the compensation point) is translated into sucrose (and/or derived sugar) changes. Within the ZT8-16 window, sugar is synthesized by photosynthesis in long days, but not in short days. Together with a higher sensitivity to sucrose/sugar during that time, the sugar changes (amount or kinetics?) induce the expression changes in *PP2-A13* and *MIPS1*. The sucrose/sugar signaling represses *PP2-A13* and induces *MIPS1* in long days. In short days, the pathway is not activated during the ZT8-16 period; therefore, the activation of *MIPS1* and the repression of *PP2-A13* do not happen. This helps plants to induce *PP2-A13* in the time window of short days. Afternoon (ZT8-16) expression of *MIPS1* and *PP2-A13* induces growth in long days and short days, respectively. The starch production and degradation are controlled by photoperiods, but the amounts of starch changes might not directly regulate photoperiodic growth. The photoperiodic accumulation patterns of starch and sucrose are based on ref.[136] and ref.[134], respectively.

discussed in this review. As we have just begun to learn about this new regulation, we anticipate learning more about how plants measure photoperiods using the photosynthesis-derived sucrose metabolic pathway.

Future perspective

The classical models have profoundly shaped our understanding of photoperiodism, guiding both experimental design and theoretical frameworks in plant photobiology and chronobiology. They provided testable predictions, conceptual clarity, and a basis for investigating seasonal responses across diverse plant taxa. However, as our molecular understanding of photoperiodic flowering mechanisms deepens, it becomes clear that molecular findings often reflect features from more than one model as discussed in this article. Finding that a photosynthesis-associated metabolic pathway controls photoperiodic growth widens our view of daylength-sensing mechanisms from interactions between conventional photoreceptors and transcriptional regulators. Our more comprehensive understanding of photoperiodic time measurement is still limited to several species, like *Arabidopsis* and rice. Classical physiological work as well as recent molecular biological work in photoperiodism using different plants clearly indicates the presence of some variations in the photoperiodic time measurement mechanisms, confirming the importance of studying multiple species. To understand the more species-specific mechanisms, classical models will guide our molecular biology experiments by providing physiological knowledge and theoretical framework related to their responses, but we no longer need to choose only one representative model to discuss possible underlying mechanisms as we now have tools and resources to explore the mechanisms in depth that control diversity in photoperiodism in various plants.

Data availability

No datasets were generated or analyzed during the current study.

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References

1. Garner, W. W. & Allard, H. A. Effect of the relative length of day and night and other factors of the environment on growth and reproduction in plants. *J. Agric. Res* **18**, 553–606 (1920).
2. Bünning, E. Open address: Biological clocks. *Cold Spring Harb. Symp. Quant. Biol.* **25**, 1–9 (1960).
3. Vince-Prue, D. *Photoperiodism in plants*. (McGraw-Hill Book Company, 1975).
4. Jabbur, M. L., Bratton, B. P. & Johnson, C. H. Bacteria can anticipate the seasons: Photoperiodism in cyanobacteria. *Science* **385**, 1105–1111 (2024).
5. Thomas, B. & Vince-Prue, D. *Photoperiodism in plants*. *Academic Press* (1996).
6. *Photoperiodism: the Biological Celender*. (Oxford University Press, 2010).
7. Withrow, R. W. in *Photoperiodism and Related Phenomena in Plants and Animals* (ed R. W. Withrow) 439-471 (Am Assoc Adv Sci, 1959).
8. Song, Y. H., Shim, J. S., Kinmonth-Schultz, H. A. & Imaizumi, T. Photoperiodic flowering: time measurement mechanisms in leaves. *Annu. Rev. Plant Biol.* **66**, 441–464 (2015).
9. Nakane, Y. & Yoshimura, T. Photoperiodic regulation of reproduction in vertebrates. *Annu. Rev. Anim. Biosci.* **7**, 173–194 (2019).
10. Gendron, J. M. & Staiger, D. New horizons in plant photoperiodism. *Annu. Rev. Plant Biol.* **74**, 481–509 (2023).
11. Lin, X., Liu, B., Weller, J. L., Abe, J. & Kong, F. Molecular mechanisms for the photoperiodic regulation of flowering in soybean. *J. Integr. Plant Biol.* **63**, 981–994 (2021).
12. Freytes, S. N., Canelo, M. & Cerdán, P. D. Regulation of flowering time: When and where?. *Curr. Opin. Plant Biol.* **63**, 102049 (2021).
13. Wang, X., Zhou, P., Huang, R., Zhang, J. & Ouyang, X. A daylength recognition model of photoperiodic flowering. *Front. Plant Sci.* **12**, 778515 (2021).

14. Osnato, M., Cota, I., Nebhnani, P., Cereijo, U. & Pelaz, S. Photoperiod control of plant growth: Flowering time genes beyond flowering. *Front. Plant. Sci.* **12**, 805635 (2021).
15. Su, C., Wang, Y., Yu, Y., He, Y. & Wang, L. Coordinative regulation of plants growth and development by light and circadian clock. *aBIOTECH* **2**, 176–189 (2021).
16. Takagi, H., Hempton, A. K. & Imaizumi, T. Photoperiodic flowering in *Arabidopsis*: Multilayered regulatory mechanisms of *CONSTANS* and the florigen *FLOWERING LOCUS T*. *Plant Commun.* **4**, 100552 (2023).
17. Vicentini, G. et al. Environmental control of rice flowering time. *Plant Commun.* **4**, 100610 (2023).
18. Wang, F., Han, T. & Chen, Z. J. Circadian and photoperiodic regulation of the vegetative to reproductive transition in plants. *Commun. Biol.* **7**, 579 (2024).
19. Hendricks, S. B. Rate of change of phytochrome as an essential factor determining photoperiodism in plants. *Cold Spring Harb. Symp. Quant. Biol.* **25**, 245–248 (1960).
20. Klose, C., Nagy, F. & Schafer, E. Thermal reversion of plant phytochromes. *Mol. Plant* **13**, 386–397 (2020).
21. Lees, A. D. Photoperiodic timing mechanisms in insects. *Nature* **210**, 986–989 (1966).
22. Lees, A. D. Photoperiodic time measurement in the aphid *Megoura viciae*. *J. Insect Physiol.* **19**, 2279–2316 (1973).
23. Hamner, K. C. & Bonner, J. Photoperiodism in relation to hormones as factors in floral initiation and develpment. *Bot. Gaz.* **100**, 388–431 (1938).
24. Borthwick, H. A., Hendricks, S. B. & Parker, M. W. The reaction controlling floral initiation. *Proc. Natl. Acad. Sci. USA* **38**, 929–934 (1952).
25. Carr, D. J. The photoperiodic behaviour of short-day plants. *Physiol. Plant* **5**, 70–84 (1952).
26. Nanda, K. K. & Hamner, K. C. Studies on the nature of the endogenous rhythm affecting photoperiodic response of Biloxi soybean. *Bot. Gaz.* **120**, 14–28 (1958).
27. Teets, N. M. & Meuti, M. E. Hello darkness, my old friend: A tutorial of Nanda-Hamner protocols. *J. Biol. Rhythms* **36**, 221–225 (2021).
28. Hamner, K. C. Photoperiodism and circadian rhythms. *Cold Spring Harb. Perspect. Biol.* **25**, 269–277 (1960).
29. Bünning, E. Circadian rhythms and the time measurement in photoperiodism. *Cold Spring Harb. Symp. Quant. Biol.* **25**, 249–256 (1960).
30. Saunders, D. S. Erwin Bünning and Tony Lees, two giants of chronobiology, and the problem of time measurement in insect photoperiodism. *J. Insect Physiol.* **51**, 599–608 (2005).
31. Pittendrigh, C. S. & Minis, D. H. The entrainment of circadian oscillations by light and their role as photoperiodic clocks. *Am. Nat.* **98**, 261–294 (1964).
32. Pittendrigh, C. S. Circadian surfaces and the diversity of possible roles of circadian organization in photoperiodic induction. *Proc. Natl. Acad. Sci. USA* **69**, 2734–2737 (1972).
33. Pittendrigh, C. S. Circadian rhythms and the circadian organization of living systems. *Cold Spring Harb. Symp. Quant. Biol.* **25**, 159–184 (1960).
34. Pittendrigh, C. S. & Daan, S. A functional analysis of circadian pacemakers in nocturnal rodents. V. Pacemaker structure: a clock for all seasons. *J. Comp. Physiol. A* **106**, 333–355 (1976).
35. Saunders, D. S. Thermoperiodic control of diapause in an insect: theory of internal coincidence. *Science* **181**, 358–360 (1973).
36. Stoleru, D., Peng, Y., Agosto, J. & Rosbash, M. Coupled oscillators control morning and evening locomotor behaviour of *Drosophila*. *Nature* **431**, 862–868 (2004).
37. Grima, B., Chelot, E., Xia, R. & Rouyer, F. Morning and evening peaks of activity rely on different clock neurons of the *Drosophila* brain. *Nature* **431**, 869–873 (2004).
38. Liang, X. et al. Morning and evening circadian pacemakers independently drive premotor centers via a specific dopamine relay. *Neuron* **102**, 843–857 (2019).
39. Yoshii, T., Saito, A. & Yokosako, T. A four-oscillator model of seasonally adapted morning and evening activities in *Drosophila melanogaster*. *J. Comp. Physiol. A* **210**, 527–534 (2024).
40. Takimoto, A. & Hamner, K. C. Effect of temperature and preconditioning on photoperiodic response of Pharbitis nil. *Plant Physiol.* **39**, 1024–1030 (1964).
41. McWatters, H. G., Bastow, R. M., Hall, A. & Millar, A. J. The *ELF3 zeitnehmer* regulates light signalling to the circadian clock. *Nature* **408**, 716–720 (2000).
42. Flis, A. et al. Photoperiod-dependent changes in the phase of core clock transcripts and global transcriptional outputs at dawn and dusk in *Arabidopsis*. *Plant Cell Environ.* **39**, 1955–1981 (2016).
43. Hayama, R., Agashe, B., Luley, E., King, R. & Coupland, G. 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 (2007).
44. Hayama, R., Mizoguchi, T. & Coupland, G. Differential effects of light-to-dark transitions on phase setting in circadian expression among clock-controlled genes in *Pharbitis nil*. *Plant Signal Behav.* **13**, e1473686 (2018).
45. Hicks, K. A. et al. Conditional circadian dysfunction of the *Arabidopsis early-flowering 3* mutant. *Science* **274**, 790–792 (1996).
46. Glossop, N. R. & Hardin, P. E. Central and peripheral circadian oscillator mechanisms in flies and mammals. *J. Cell Sci.* **115**, 3369–3377 (2002).
47. Takahashi, N., Hirata, Y., Aihara, K. & Mas, P. A hierarchical multi-oscillator network orchestrates the *Arabidopsis* circadian system. *Cell* **163**, 148–159 (2015).
48. Endo, M., Shimizu, H., Nohales, M. A., Araki, T. & Kay, S. A. Tissue-specific clocks in *Arabidopsis* show asymmetric coupling. *Nature* **515**, 419–422 (2014).
49. Suarez-Lopez, P. et al. *CONSTANS* mediates between the circadian clock and the control of flowering in *Arabidopsis*. *Nature* **410**, 1116–1120 (2001).
50. Yanovsky, M. J. & Kay, S. A. Molecular basis of seasonal time measurement in *Arabidopsis*. *Nature* **419**, 308–312 (2002).
51. Lin, X., Fang, C., Liu, B. & Kong, F. Natural variation and artificial selection of photoperiodic flowering genes and their applications in crop adaptation. *aBIOTECH* **2**, 156–169 (2021).
52. Wang, F., Li, S., Kong, F., Lin, X. & Lu, S. Altered regulation of flowering expands growth ranges and maximizes yields in major crops. *Front Plant Sci.* **14**, 1094411 (2023).
53. Rédei, G. P. Supervital mutants of *Arabidopsis*. *Genetics* **47**, 443–460 (1962).
54. Koornneef, M., Hanhart, C. J. & van der Veen, J. H. A genetic and physiological analysis of late flowering mutants in *Arabidopsis thaliana*. *Mol. Gen. Genet.* **229**, 57–66 (1991).
55. Fowler, S. et al. *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 (1999).
56. Nelson, D. C., Lasswell, J., Rogg, L. E., Cohen, M. A. & Bartel, B. *FKF1*, a clock-controlled gene that regulates the transition to flowering in *Arabidopsis*. *Cell* **101**, 331–340 (2000).
57. Christie, J. M. Phototropin blue-light receptors. *Annu. Rev. Plant Biol.* **58**, 21–45 (2007).
58. Suetsugu, N. & Wada, M. Evolution of three LOV blue light receptor families in green plants and photosynthetic stramenopiles: phototropin, ZTL/FKF1/LKP2 and aureochrome. *Plant Cell Physiol.* **54**, 8–23 (2013).
59. Brunner, M. & Kaldi, K. Interlocked feedback loops of the circadian clock of *Neurospora crassa*. *Mol. Microbiol.* **68**, 255–262 (2008).

60. Chen, C. H., Dunlap, J. C. & Loros, J. J. Neurospora illuminates fungal photoreception. *Fungal Genet. Biol.* **47**, 922–929 (2010).
61. Imaizumi, T., Tran, H. G., Swartz, T. E., Briggs, W. R. & Kay, S. A. FKF1 is essential for photoperiodic-specific light signalling in *Arabidopsis*. *Nature* **426**, 302–306 (2003).
62. Imaizumi, T., Schultz, T. F., Harmon, F. G., Ho, L. A. & Kay, S. A. FKF1 F-box protein mediates cyclic degradation of a repressor of *CONSTANS* in *Arabidopsis*. *Science* **309**, 293–297 (2005).
63. Fornara, F. et al. *Arabidopsis* DOF transcription factors act redundantly to reduce *CONSTANS* expression and are essential for a photoperiodic flowering response. *Dev. Cell* **17**, 75–86 (2009).
64. Yan, J. et al. FKF1 F-box protein promotes flowering in part by negatively regulating DELLA protein stability under long-day photoperiod in Arabidopsis. *J. Integr. Plant Biol.* **62**, 1717–1740 (2020).
65. Sawa, M., Nusinow, D. A., Kay, S. A. & Imaizumi, T. FKF1 and GIGANTEA complex formation is required for day-length measurement in *Arabidopsis*. *Science* **318**, 261–265 (2007).
66. Sawa, M., Kay, S. A. & Imaizumi, T. Photoperiodic flowering occurs under internal and external coincidence. *Plant Signal Behav.* **3**, 269–271 (2008).
67. Song, Y. H., Smith, R. W., To, B. J., Millar, A. J. & Imaizumi, T. FKF1 conveys timing information for CONSTANS stabilization in photoperiodic flowering. *Science* **336**, 1045–1049 (2012).
68. Baudry, A. et al. F-box proteins FKF1 and LKP2 act in concert with ZEITLUPE to control *Arabidopsis* clock progression. *Plant Cell* **22**, 606–622 (2010).
69. Hayama, R. et al. PSEUDO RESPONSE REGULATORs stabilize CONSTANS protein to promote flowering in response to day length. *EMBO J.* **36**, 904–918 (2017).
70. Lee, B. D. et al. The F-box protein FKF1 inhibits dimerization of COP1 in the control of photoperiodic flowering. *Nat. Commun.* **8**, 2259 (2017).
71. Wang, H., Ma, L. G., Li, J. M., Zhao, H. Y. & Deng, X. W. Direct interaction of *Arabidopsis* cryptochromes with COP1 in light control development. *Science* **294**, 154–158 (2001).
72. Liu, B., Zuo, Z., Liu, H., Liu, X. & Lin, C. *Arabidopsis* cryptochrome 1 interacts with SPA1 to suppress COP1 activity in response to blue light. *Genes Dev.* **25**, 1029–1034 (2011).
73. Zuo, Z., Liu, H., Liu, B., Liu, X. & Lin, C. Blue light-dependent interaction of CRY2 with SPA1 regulates COP1 activity and floral initiation in Arabidopsis. *Curr. Biol.* **21**, 841–847 (2011).
74. Rizzini, L. et al. Perception of UV-B by the *Arabidopsis* UVR8 protein. *Science* **332**, 103–106 (2011).
75. Sheerin, D. J. et al. Light-activated phytochrome A and B interact with members of the SPA family to promote photomorphogenesis in Arabidopsis by reorganizing the COP1/SPA complex. *Plant Cell* **27**, 189–201 (2015).
76. Hwang, D. Y. et al. GIGANTEA regulates the timing stabilization of CONSTANS by altering the interaction between FKF1 and ZEITLUPE. *Mol. Cells* **42**, 693–701 (2019).
77. Feke, A., Vanderwall, M., Liu, W. & Gendron, J. M. Functional domain studies uncover novel roles for the ZTL Kelch repeat domain in clock function. *PLoS One* **16**, e0235938 (2021).
78. Cho, S. W. et al. Disrupting FKF1 homodimerization increases *FT* transcript levels in the evening by enhancing CO stabilization. *Plant Cell Rep.* **43**, 121 (2024).
79. Zoltowski, B. D. & Crane, B. R. Light activation of the LOV protein vivid generates a rapidly exchanging dimer. *Biochemistry* **47**, 7012–7019 (2008).
80. Herrou, J. & Crosson, S. Function, structure and mechanism of bacterial photosensory LOV proteins. *Nat. Rev. Microbiol* **9**, 713–723 (2011).
81. Heintz, U. & Schlichting, I. Blue light-induced LOV domain dimerization enhances the affinity of Aureochrome 1a for its target DNA sequence. *Elife* **5**, e11860 (2016).
82. Kubota, A. et al. Co-option of a photoperiodic growth-phase transition system during land plant evolution. *Nat. Commun.* **5**, 3668 (2014).
83. Linde, A. M. et al. Early evolution of the land plant circadian clock. *N. Phytol.* **216**, 576–590 (2017).
84. Renau-Morata, B. et al. CDF transcription factors: plant regulators to deal with extreme environmental conditions. *J. Exp. Bot.* **71**, 3803–3815 (2020).
85. Kanesaka, Y., Inoue, K., Tomita, Y., Yamaoka, S. & Araki, T. Circadian clock does not play an essential role in daylength measurement for growth-phase transition in *Marchantia polymorpha*. *Front. Plant Sci.* **14**, 1275503 (2023).
86. Zhao, X. et al. A critical suppression feedback loop determines soybean photoperiod sensitivity. *Dev. Cell* **59**, 1750–1763 (2024).
87. Kloosterman, B. et al. Naturally occurring allele diversity allows potato cultivation in northern latitudes. *Nature* **495**, 246–250 (2013).
88. Shi, L. et al. Aging later but faster: how *StCDF1* regulates senescence in *Solanum tuberosum*. *N. Phytol.* **242**, 2541–2554 (2024).
89. Valverde, F. et al. Photoreceptor regulation of CONSTANS protein in photoperiodic flowering. *Science* **303**, 1003–1006 (2004).
90. Song, Y. H. et al. Distinct roles of FKF1, GIGANTEA, and ZEITLUPE proteins in the regulation of CONSTANS stability in *Arabidopsis* photoperiodic flowering. *Proc. Natl. Acad. Sci. USA* **111**, 17672–17677 (2014).
91. Kim, T. S. et al. HSP90 functions in the circadian clock through stabilization of the client F-box protein ZEITLUPE. *Proc. Natl. Acad. Sci. USA* **108**, 16843–16848 (2011).
92. Jang, S. et al. *Arabidopsis* COP1 shapes the temporal pattern of CO accumulation conferring a photoperiodic flowering response. *EMBO J.* **27**, 1277–1288 (2008).
93. Ranjan, A., Fiene, G., Fackendahl, P. & Hoecker, U. The Arabidopsis repressor of light signaling SPA1 acts in the phloem to regulate seedling de-etiolation, leaf expansion and flowering time. *Development* **138**, 1851–1862 (2011).
94. Arongaus, A. B. et al. *Arabidopsis* RUP2 represses UVR8-mediated flowering in noninductive photoperiods. *Genes Dev.* **32**, 1332–1343 (2018).
95. de Los Reyes, P. et al. CONSTANS alters the circadian clock in *Arabidopsis thaliana*. *Mol. Plant* **17**, 1204–1220 (2024).
96. Takada, S. & Goto, K. TERMINAL FLOWER2, an Arabidopsis homolog of HETEROCHROMATIN PROTEIN1, counteracts the activation of *FLOWERING LOCUS T* by CONSTANS in the vascular tissues of leaves to regulate flowering time. *Plant Cell* **15**, 2856–2865 (2003).
97. An, H. et al. CONSTANS acts in the phloem to regulate a systemic signal that induces photoperiodic flowering of *Arabidopsis*. *Development* **131**, 3615–3626 (2004).
98. Song, Y. H. et al. Molecular basis of flowering under natural long-day conditions in *Arabidopsis*. *Nat. Plants* **4**, 824–835 (2018).
99. Lee, N. et al. The *FLOWERING LOCUS T* gene expression is controlled by high-irradiance response and external coincidence mechanism in long days in *Arabidopsis*. *N. Phytol.* **239**, 208–221 (2023).
100. Dupre, S. M. et al. Identification of *Eya3* and *TAC1* as long-day signals in the sheep pituitary. *Curr. Biol.* **20**, 829–835 (2010).
101. Wood, S. H. et al. Circadian clock mechanism driving mammalian photoperiodism. *Nat. Commun.* **11**, 4291 (2020).
102. Shinomura, T., Uchida, K. & Furuya, M. Elementary processes of photoperception by phytochrome A for high-irradiance response of hypocotyl elongation in Arabidopsis. *Plant Physiol.* **122**, 147–156 (2000).
103. Burgie, E. S. et al. Differing biophysical properties underpin the unique signaling potentials within the plant phytochrome photoreceptor families. *Proc. Natl. Acad. Sci. USA* **118**, e2105649118 (2021).

104. Jackson, S. & Thomas, B. Photoreceptors and signals in the photoperiodic control of development. *Plant Cell Environ.* **20**, 790–795 (1997).
105. Johnson, E., Bradley, M., Harberd, N. P. & Whitelam, G. C. Photoresponses of light-grown *phyA* mutants of *Arabidopsis*. *Plant Physiol.* **105**, 141–149 (1994).
106. Kim, H. et al. Low temperature-mediated repression and far-red light-mediated induction determine morning *FLOWERING LOCUS T* expression levels. *J. Integr. Plant Biol.* **66**, 103–120 (2024).
107. Li, F. W. et al. Phytochrome diversity in green plants and the origin of canonical plant phytochromes. *Nat. Commun.* **6**, 7852 (2015).
108. Inoue, K., Nishihama, R., Araki, T. & Kohchi, T. Reproductive induction is a far-red high irradiance response that is mediated by phytochrome and PHYTOCHROME INTERACTING FACTOR in *Marchantia polymorpha*. *Plant Cell Physiol.* **60**, 1136–1145 (2019).
109. Laurie, R. E. et al. The Medicago *FLOWERING LOCUS T* homolog, *MtFTa1*, is a key regulator of flowering time. *Plant Physiol.* **156**, 2207–2224 (2011).
110. Chen, A. et al. PHYTOCHROME C plays a major role in the acceleration of wheat flowering under long-day photoperiod. *Proc. Natl. Acad. Sci. USA* **111**, 10037–10044 (2014).
111. Kurokura, T., Samad, S., Koskela, E., Mouhu, K. & Hytonen, T. *Fragaria vesca* CONSTANS controls photoperiodic flowering and vegetative development. *J. Exp. Bot.* **68**, 4839–4850 (2017).
112. Ding, J., Zhang, B., Li, Y., Andre, D. & Nilsson, O. Phytochrome B and PHYTOCHROME INTERACTING FACTOR8 modulate seasonal growth in trees. *N. Phytol.* **232**, 2339–2352 (2021).
113. Xu, M. et al. The soybean-specific maturity gene E1 family of floral repressors controls night-break responses through down-regulation of *FLOWERING LOCUS T* orthologs. *Plant Physiol.* **168**, 1735–1746 (2015).
114. Mehta, D. et al. Twilight length alters growth and flowering time in Arabidopsis via *LHY/CCA1*. *Sci. Adv.* **10**, eadl3199 (2024).
115. Legris, M., Ince, Y. C. & Fankhauser, C. Molecular mechanisms underlying phytochrome-controlled morphogenesis in plants. *Nat. Commun.* **10**, 5219 (2019).
116. Somers, D. E., Devlin, P. F. & Kay, S. A. Phytochromes and cryptochromes in the entrainment of the *Arabidopsis* circadian clock. *Science* **282**, 1488–1490 (1998).
117. Goto, N., Kumagai, T. & Koornneef, M. Flowering responses to light-breaks in photomorphogenic mutants of *Arabidopsis thaliana*, a long-day plant. *Physiol. Plant* **83**, 209–215 (1991).
118. Pearce, S. et al. Night-break experiments shed light on the Photoperiod1-mediated flowering. *Plant Physiol.* **174**, 1139–1150 (2017).
119. Ishikawa, R. et al. Suppression of the floral activator *Hd3a* is the principal cause of the night break effect in rice. *Plant Cell* **17**, 3326–3336 (2005).
120. Ishikawa, R., Shinomura, T., Takano, M. & Shimamoto, K. Phytochrome dependent quantitative control of *Hd3a* transcription is the basis of the night break effect in rice flowering. *Genes Genet Syst.* **84**, 179–184 (2009).
121. Hayama, R. & Coupland, G. The molecular basis of diversity in the photoperiodic flowering responses of Arabidopsis and rice. *Plant Physiol.* **135**, 677–684 (2004).
122. Itoh, H., Nonoue, Y., Yano, M. & Izawa, T. A pair of floral regulators sets critical day length for *Hd3a* florigen expression in rice. *Nat. Genet.* **42**, 635–638 (2010).
123. Zheng, T. et al. Post-transcriptional regulation of Ghd7 protein stability by phytochrome and OsGI in photoperiodic control of flowering in rice. *N. Phytol.* **224**, 306–320 (2019).
124. Komiya, R., Yokoi, S. & Shimamoto, K. A gene network for long-day flowering activates *RFT1* encoding a mobile flowering signal in rice. *Development* **136**, 3443–3450 (2009).
125. Yano, M. et al. *Hd1*, a major photoperiod sensitivity quantitative trait locus in rice, is closely related to the Arabidopsis flowering time gene *CONSTANS*. *Plant Cell* **12**, 2473–2484 (2000).
126. Kojima, S. et al. *Hd3a*, a rice ortholog of the *Arabidopsis FT* gene, promotes transition to flowering downstream of *Hd1* under short-day conditions. *Plant Cell Physiol.* **43**, 1096–1105 (2002).
127. Ishikawa, R. et al. Phytochrome B regulates *Heading date 1* (*Hd1*)-mediated expression of rice florigen *Hd3a* and critical day length in rice. *Mol. Genet. Genomics* **285**, 461–470 (2011).
128. Nemoto, Y., Nonoue, Y., Yano, M. & Izawa, T. *Hd1*,a *CONSTANS* ortholog in rice, functions as an *Ehd1* repressor through interaction with monocot-specific CCT-domain protein Ghd7. *Plant J.* **86**, 221–233 (2016).
129. Zong, W. et al. Strong photoperiod sensitivity is controlled by cooperation and competition among Hd1, Ghd7 and DTH8 in rice heading. *N. Phytol.* **229**, 1635–1649 (2021).
130. Pin, P. A. & Nilsson, O. The multifaceted roles of FLOWERING LOCUS T in plant development. *Plant Cell Environ.* **35**, 1742–1755 (2012).
131. Liu, W. et al. A metabolic daylength measurement system mediates winter photoperiodism in plants. *Dev. Cell* **56**, 2501–2515 (2021).
132. Wang, Q., Liu, W., Leung, C. C., Tarte, D. A. & Gendron, J. M. Plants distinguish different photoperiods to independently control seasonal flowering and growth. *Science* **383**, eadg9196 (2024).
133. Graf, A., Schlereth, A., Stitt, M. & Smith, A. M. Circadian control of carbohydrate availability for growth in plants at night. *Proc. Natl. Acad. Sci. USA* **107**, 9458–9463 (2010).
134. Sulpice, R. et al. *Arabidopsis* coordinates the diurnal regulation of carbon allocation and growth across a wide range of photoperiods. *Mol. Plant* **7**, 137–155 (2014).
135. Seki, M. et al. Adjustment of the Arabidopsis circadian oscillator by sugar signalling dictates the regulation of starch metabolism. *Sci. Rep.* **7**, 8305 (2017).
136. Alexandre Moraes, T. et al. The circadian clock mutant *lhy cca1 elf3* paces starch mobilization to dawn despite severely disrupted circadian clock function. *Plant Physiol.* **189**, 2332–2356 (2022).
137. Yang, L., Xu, M., Koo, Y., He, J. & Poethig, R. S. Sugar promotes vegetative phase change in *Arabidopsis thaliana* by repressing the expression of *MIR156A* and *MIR156C*. *Elife* **2**, e00260 (2013).
138. Lunn, J. E. et al. Sugar-induced increases in trehalose 6-phosphate are correlated with redox activation of ADPglucose pyrophosphorylase and higher rates of starch synthesis in *Arabidopsis thaliana*. *Biochem J.* **397**, 139–148 (2006).
139. Wahl, V. et al. Regulation of flowering by trehalose-6-phosphate signaling in *Arabidopsis thaliana*. *Science* **339**, 704–707 (2013).

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Competing interests

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