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REVIEW

Tissue-specific regulation of flowering by photoreceptors

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Abstract Plants use various kinds of environmental signals to adjust the timing of the transition from the vegetative to reproductive phase (flowering). Since flowering at the appropriate time is crucial for plant reproductive strategy, several kinds of photoreceptors are deployed to sense environmental light conditions. In this review, we will update our current understanding of light signaling pathways in flowering regulation, especially, in which tissue do photoreceptors regulate flowering in response to light quality and photoperiod. Since light signaling is also integrated into other flowering pathways, we also introduce recent progress on how photoreceptors are involved in tissue-specific thermosensation and the gibberellin pathway. Finally, we discuss the importance of cell-type-specific analyses for future plant studies.

Keywords Photoperiod · Light quality · Temperature · Gibberellin · Tissue-specific regulation · Phytochrome · Cryptochrome · Day length

Abbreviations

SAR	Shade avoidance response
LD	Long day
SD	Short day

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Introduction

Plants utilize sunlight not only as an energy source via photosynthesis but also as an information source for surrounding conditions. Being sessile, plants employ photoreceptors to perceive environmental light signals and regulate physiological responses to adapt to the changing surroundings. Among photoreceptor-regulated physiological responses, the vegetative to reproductive phase transition (referred to as flowering) is crucial because the appropriate timing of flowering directly leads to reproductive success. Environmental light signals contain many kinds of information, such as photoperiod, light intensity, light direction, and light quality. Of these light signals, light quality and photoperiod especially are widely used in flowering regulation.

Light quality usually refers to the red:far-red light ratio. Since red light is predominantly absorbed by chlorophylls, and far-red light passes through most leaves, the red:far-red light ratio under a vegetation canopy decreases (<1) compared to direct sunlight (around 1.2) (Fig. 1). In response to low light quality, such as under a vegetation canopy, flowering is promoted by the 'shade avoidance response (SAR)', which was first described systematically by Sachs in 1863 [1, 2]. SAR is a kind of emergency response to escape from shade; therefore, it is usually associated with long petioles, erect and pale (low chlorophyll content) leaves, and early flowering. Another informative light signal for flowering regulation is the photoperiod, or the day length. Except for polar and equatorial regions, the photoperiod undergoes seasonal oscillations; in contrast to the light quality pathway, therefore, the photoperiod pathway is a mechanism for regular seasonal flowering, which was first reported by Garner and Allard in 1923 [3]. Plants that flower under

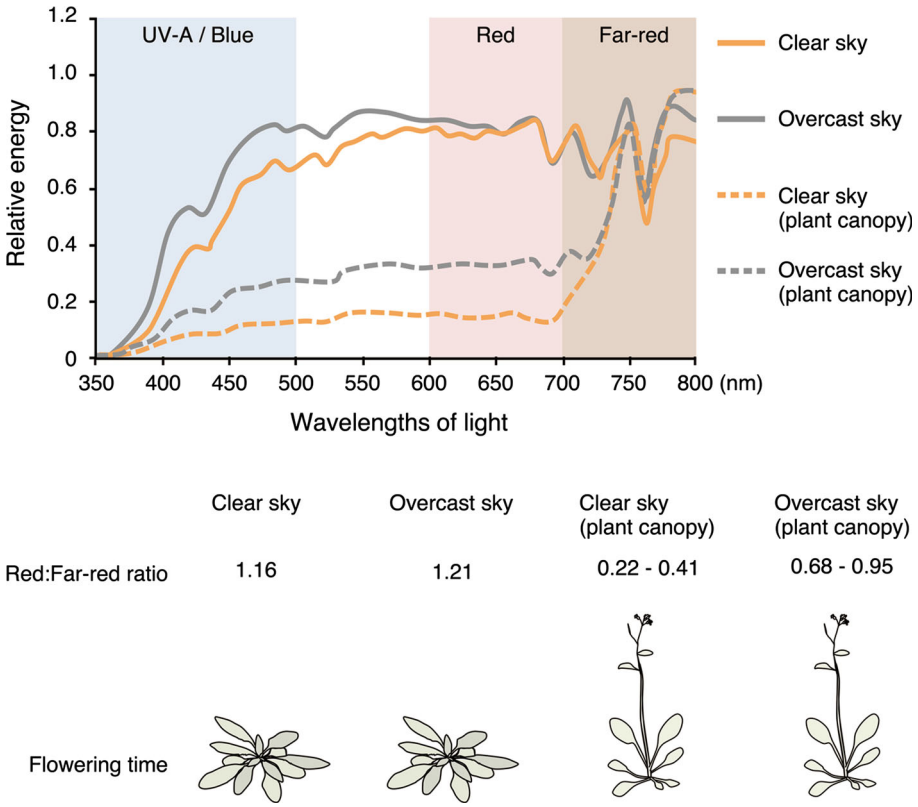

Fig. 1 Light quality-mediated flowering regulation. When plants are under a vegetation canopy, or when it is overcast, plants cannot perceive enough sunlight. However, plants need to distinguish between an enduring vegetation canopy and a temporal cloudy day. Since blue and red light is predominantly absorbed by chlorophyll, sunlight that passes through the vegetation canopy contains more *far-red light* than *blue* or *red light*. On the other hand, under overcast conditions, clouds reflect a large portion of sunlight, resulting in a small increase of blue light but very little change in the *red*:*far-red* ratio [116]. Therefore, only when plants are under a vegetation canopy will they initiate SAR and induce flowering. An increase of the *blue light* ratio may not affect flowering in this case, because flowering time was not affected over a wide range of *blue light* intensities (25–164 μmol m^{-2} s^{-1}) [117]

long day (LD) and short day (SD) are referred to as LD plants and SD plants, respectively.

The model plant *Arabidopsis thaliana*, a facultative LD plant, has provided a wealth of data elucidating the molecular basis of light signaling, as well as genetic pathways. Although large numbers of light signaling studies have focused on early photomorphogenesis, much less attention has been paid to flowering regulation by photoreceptors. Since some light signaling mutants manifest only a flowering phenotype and others display only photomorphogenic phenotypes, the light signaling pathways appear to diverge at an early step. Intriguingly, several studies have clearly demonstrated that flowering is regulated by light signaling pathways in specific tissues, although photoreceptors are expressed in almost all cell types [4–6]. Therefore, it is important to marshal data about the various light signaling factors and the responsible tissues that are involved in flowering regulation in *Arabidopsis*. Moreover, recent progress makes it clear that light signaling is also integrated into many other flowering pathways, especially the thermosensory and gibberellin pathways. In this review we highlight the molecular mechanisms operative in photoreceptor-mediated flowering regulation. We also discuss recent studies on tissue-specific functions of circadian clocks that are tightly involved in light and temperature signaling, and the regulation of flowering.

Light signal transduction in the light quality pathway

A low red:far-red light ratio induces SAR-associated photomorphogenesis and early flowering. phyB plays a major role in this response. A subfamily of basic helix–loop–helix (bHLH) proteins, phytochrome-interacting factors (PIFs), directly interact with the Pfr form of phyB [7, 8]. The PIF transcription factors contribute to photomorphogenic processes, including hypocotyl elongation, chloroplast development, and seed germination [9–11]. In flowering regulation, overexpression of PIF4 or PIF5 causes early flowering [12, 13]. Moreover, genome-wide association studies demonstrated that PIF4 is associated with variation

in flowering time [14]. However, a quadruple mutant deficient in *pif1*, *pif3*, *pif4,* and *pif5*, or any of the respective single mutants, displayed no clear differences in flowering time [9], suggesting highly redundant functions of PIFs.

Another factor, early flowering 3 (ELF3), is crucial for light quality signaling. Quantitative trait loci (QTL) analysis in recombinant inbred lines of *Arabidopsis* identified *ELF3* as the most likely candidate gene affecting the shade avoidance response [15]. *ELF3* is a circadian clock gene, and *elf3* mutants show long hypocotyls, early flowering, and impaired responsiveness to red light, much like a *phyB* mutant [16, 17]. Consistent with this notion, phyB, ELF3, and PIF4 interact both genetically and physically [18–20]. Although the circadian clock gates rapid SAR [21], a recent study shows that the ELF3–PIF4 interaction is independent of the ELF3–ELF4–LUX complex (evening complex) [20, 22]. How a circadian clock protein functions in the SAR is still unclear because the mechanisms for light input to the circadian clock system are not well understood.

A transcriptional mediator complex subunit, phytochrome and flowering time 1 (PFT1), has been reported as a key factor that mediates phyB-to-FT signaling [23]. Recent studies, however, show that PFT1 has pleiotropic functions, including plant hormone signaling, sulfate assimilation, iron homeostasis, and reactive oxygen species (ROS) distribution [24–26]. Therefore, PFT1 appears to be a more general transcriptional mediator, rather than a specific factor for the light quality pathway.

Plant photoreceptors for flowering regulation

In *Arabidopsis*, five classes of photoreceptors have been discovered. Phytochromes (phyA to phyE) are major red and far-red light photoreceptors [27]. Phytochromes can be classified as light-labile type I (phyA) and light-stable type II (phyB to phyE) [28, 29]. Type II phytochromes are responsible for classic red:far-red photoreversible physiological responses. Cryptochromes (cry1 and cry2) are ultraviolet-A (UV-A) and blue light photoreceptors [30]. Similar to the phytochromes, cry1 is light stable and cry2 is light labile [31, 32]. In *Arabidopsis*, cry3 and cry-dash appear to be more like photolyase than bona fide blue light photoreceptors, although some cry-dash orthologs have been shown to possess cryptochrome activities in other organisms [33–37]. Phototropins (phot1 and phot2) are also UV-A and blue light receptors involved in phototropism. Zeitlupe (ZTL), flavin-binding, kelch repeat, F-box 1 (FKF1), and lov kelch protein 2 (LKP2) have an F-box domain and a Kelch repeat domain [38]. These have not been classified as canonical photoreceptors but nevertheless have been demonstrated to be blue light photoreceptors [39]. UV resistance locus 8 (UVR8) is the latest identified photoreceptor, which absorbs UV-B [40].

Among these photoreceptor family members, phytochromes, cryptochromes, and ZTL/FKF1/LKP2 are three major photoreceptors that are implicated in regulating the timing of flowering. Deploying such a variety of photoreceptors may be beneficial for plants to optimize their adaptation, since both blue and red/far-red light are needed for the precise estimation of surrounding light conditions and seasons (see detail below). Signals from these photoreceptors are integrated into the expression of flowering locus T (FT) protein, which is also known to be a flowering hormone, or florigen [41]. FT protein is tissue specifically synthesized in the leaf vascular phloem companion cells, and it moves through the phloem to the shoot apical meristem. At the shoot apical meristem, FT functions as a part of the florigen activating complex (FAC) and promotes transcription of floral initiation genes [42].

In contrast to the leaf vascular bundle-specific *FT* expression, photoreceptors that regulate flowering are expressed in almost all cell types. Although traditional GUS staining showed striped patterns of phytochrome expression, promoter::LUC or promoter::GFP assays are more consistent with ubiquitous expression of phytochromes [4, 43–45]. Cryptochromes and ZTL/FKF1/LKP2 are also similar to the phytochromes and are expressed ubiquitously in plants [5, 45, 46]. Taken together, plant photoreceptors are expressed uniformly, and therefore the downstream mechanisms for vascular *FT* regulation should be tissue specific.

Tissue-specific regulation of the light quality pathway

Given the aforementioned collection of regulatory genes, it was possible to identify a particular tissue where SAR is regulated. A study using a phyB-GFP enhancer trap line revealed that phyB in mesophyll regulates flowering in the light quality pathway [4]. Interestingly, phyB-GFP that was expressed only in vasculature, epidermis, or root did not complement the early flowering phenotype of the parental *phyB* mutant, implying a particular importance of mesophyll for the light quality pathway. Although mesophyll expression of *ELF3*, *ELF4*, *LUX*, and *PFT1* can be observed by tissue-specific microarray analysis, the tissue-specific functions of other factors that are implicated in SAR have not yet been tested [47]. Therefore, how mesophyllic phyB regulates vascular *FT* expression is still unclear. Some reports have demonstrated that proteins can be transported from mesophyll to vasculature through plasmodesmata, suggesting intertissue communication between these two tissues [48]. SAR is associated with chronic reduction in the amount of photosynthesis, and it is critically distinguished from cloud cover (Fig. 1). Therefore, mesophyllic regulation of flowering associated with SAR seems to be

biologically reasonable to achieve tight coupling of SAR regulation with photosynthesis.

Light signal transduction in the photoperiod pathway

In many plant species including *Arabidopsis*, constans (CO) and its direct target FT are crucial, especially in the photoperiod pathway [46, 49, 50]. When CO and FT functions are perturbed, plants cannot sense seasonal cues, and flowering time under a floral inductive condition will be the same as in the non-inductive condition. In *Arabidopsis*, *co* and *ft* mutants show late flowering both under LD and SD conditions [51]. On the other hand, *CO* and *FT* overexpressing lines exhibit a dramatically early flowering phenotype independent of day length [52–54]. Therefore, regulation of *CO* and *FT* gene transcription and protein stability are major control mechanisms that may be impacted by photoreceptor input.

CO expression

Although *CO* expression is regulated by flowering BHLH 1(FBH1), FBH2, FBH3, and FBH4 [55], FKF1 also regulates photoperiod-dependent flowering. The expression levels of *FKF1* and *gigantea* (*GI*) are oscillatory, and the respective proteins accumulate in the evening [56]. Accumulated FKF1 and GI interact with each other, forming a complex in a blue light-dependent manner. The FKF1–GI complex degrades cycling DOF factor 1 (CDF1), a transcriptional repressor of *CO*, in the evening [57]; therefore, blue light-dependent *CO* transcriptional activation can be observed at the end of the day. ZTL and LKP2 also regulate *CO* transcription. In contrast to FKF1, overexpression of *ZTL* or *LKP2* strongly suppresses *CO* expression and results in late flowering [58, 59]. However, there are at least two explanations for why these overexpressing lines show suppressed *CO* transcription. One explanation is that when three or more factors are required for a functional complex, knockout or overexpression leads to a perturbed stoichiometric balance of the components, resulting in impaired function. This is compatible with the notion that ZTL and LKP2 interact with FKF1 in yeast and in vitro [60]. Another possibility might involve the destabilization of circadian clock proteins timing of cab expression 1 (TOC1) and pseudo response regulator 5 (PRR5). ZTL has been shown to destabilize the TOC1 and PRR5 proteins. Flowering time in a *toc1 prr5* double mutant line is as late as in the *ZTL* overexpression lines [61], and these two clock genes control *CO* expression, probably through transcriptional and posttranscriptional regulation of CDFs [62–67]. PFT1, a phyB signaling component, also shows a slightly suppressed *CO* transcription level in the *pft1* mutant [23, 68, 69].

CO protein stability

In addition to *CO* transcriptional regulation, photoreceptors also control CO protein stability. Under LD conditions, CO protein expressed in the morning is degraded, whereas CO protein expressed in the evening is stabilized [70–72]. To achieve such a dynamic regulation of CO protein concentration, at least two different types of ubiquitin ligases are involved. An E3 ubiquitin ligase complex, comprising constitutive photomorphogenic 1 (COP1) and suppressor of phytochrome A (SPA1), triggers degradation of CO protein in the night period [73, 74]. As the day progresses, photoactivated cry2 directly binds SPA1 and inhibits the formation of the COP1-SPA1 complex [75]. Similar to the cry2 mechanism, phyA and cry1 also inhibit the COP1–SPA1 complex [76–79]. Physiological studies have indicated that phyA and cry2 are day-length sensors [80, 81], suggesting that COP1–SPA1-mediated CO protein degradation is a node in the regulatory network controlling photoperiodic flowering. FKF1 also stabilizes CO in the afternoon [82]. In addition, CO protein in the morning is degraded by a COP1-independent pathway that is activated by phyB [74]. Another ubiquitin ligase, high expression of osmotically responsive gene 1 (HOS1), is a good candidate for phyB-dependent CO protein degradation in the morning [71]. Phytochrome-dependent late flowering (PHL) also stabilizes CO protein in the afternoon [83]; however, the mechanism is largely unknown. These combinations of transcriptional and post-translational regulation lead to a transient accumulation of CO protein at the end of LD.

FT expression

Transcription of *FT* is mainly regulated by CO protein, which accumulates at the end of LD. Some other mechanisms to activate *FT* transcription are also involved. Cryptochrome interacting basic helix–loop–helix1 (CIB1) interacts with cry2 in a blue light-dependent manner, and the cry2–CIB1 complex interacts in vivo with DNA elements in chromatin associated with the *FT* promoter [84]. Furthermore, ZTL and LKP2, but not FKF1, are required for the accumulation of CIB1 protein in response to blue light [85]. GI and FKF1 also regulate *FT* expression in a *CO*-independent manner [72, 86]. Although GI shares the same binding region in the *FT* promoter as short vegetative phase (SVP), a repressor of *FT* expression, whether FKF1 is involved in this response is largely unknown [86]. GI has been considered to be a phyB signaling component [87]. Recently, an interaction between phyB and GI has been demonstrated both in vivo and by yeast two-hybrid analysis, implying the involvement of red light in *FT* expression [19].

Tissue-specific regulation of the photoperiod pathway

In contrast to the light quality pathway, the importance of vasculature (phloem companion cells) in photoperiodic flowering has been demonstrated. Phloem companion cell-specific expression of cry2-GFP driven by a vasculature-specific *SUC2* promoter was sufficient to complement the late flowering phenotype of a *cry2* mutant [5]. Tissue-specific expression of cry2-GFP in other tissues (mesophyll, epidermis, shoot apical meristem, and root) did not complement the late flowering phenotype at all. Although it is not yet clear which specific tissue is essential for the other day-length sensor, phyA, to regulate flowering, COP1 and SPA1, which are also involved in phyA signaling, are known to regulate flowering only through the vasculature [6, 74]. Therefore, the importance of vasculature in the photoperiod pathway has been clearly demonstrated. Vasculature-specific or vasculature-enriched expression of transcription factors such as CDF1, CO, and FT [57, 88] can explain why vasculature is important for the photoperiod pathway. Since a genetic interaction between phyB and phyA/cry2 has been demonstrated [80, 81], and since CO protein degradation is antagonistically regulated by phyB and phyA/cryptochromes [70], the signals from mesophyll phyB and vascular phyA/cry2 are integrated via the regulation of CO protein stability in the vasculature.

Consistent with vasculature-specific functions of the photoperiod pathway, the circadian clock in vasculature also regulates flowering in the photoperiod pathway [47, 89]. Perturbation of circadian clock in mesophyll epidermis, shoot apical meristem, hypocotyl, and root did not affect photoperiod pathway at all. Together, these observations suggest that there is a clear assignment of roles in light signaling pathways.

However, too many factors seem to be identified as vascular-enriched in the photoperiod pathway. In general, vascular-enriched expression patterns determined by GUS staining assay need to deal cautiously, because recent tissue-specific microarray analyses have demonstrated that expression levels of these photoreceptors are almost the same in mesophyll and vasculature [47]. An apparent discordance of spatial expression patterns may stem from the cell size and cell density of vascular cells: these small and tightly aligned cells will show relatively strong GUS staining even if the *GUS* expression levels are almost the same as in other tissues. To support our view, GUS staining was more intense in the vasculature even in 35S::GUS or equivalent transgenic lines [90, 91].

Light signals integrated into other pathways

In addition to the light signals, temperature and gibberellin also affect flowering. Temperature varies widely from hour-to-hour, day-to-day and season-to-season. Since plants are heterothermal organisms, appropriate responses to the large variation in temperature are crucial for plant growth regulation. In flowering, low temperature (2–10 °C, depending on the plant species) and intermediate temperature (12–27 °C, also referred to as ambient temperature) pathways have been extensively studied [92, 93]. Gibberellins are required for the normal growth of plants through the promotion of cell division and cell elongation. In addition to that, they promote flowering, especially under non-inductive light conditions [94]. Recent work revealed that signals from photoreceptors are also integrated into both temperature and gibberellin pathways.

Vernalization pathway

Plant flowering induction potentiated by low temperature is referred to as 'vernalization', and this response is beneficial for detecting a winter season preceding spring. A MADS-box transcription factor, flowering locus C (FLC) is an inhibitor of flowering activators, and it has a crucial role in the vernalization mechanism [95, 96]. Low temperature suppresses FLC accumulation and this leads to an increase in expression of FT and other flowering-related genes [97]. Light signaling is integrated into this vernalization pathway. The subgroup VIII-2 of NAC proteins encoding *vascular plant one-zinc finger1* (*VOZ1*) and *VOZ2* are direct phyB-interacting factors, and the *voz1 voz2* double mutant displays a late flowering phenotype. The *FLC* expression level in the double mutant is suppressed independent of vernalization [98, 99]. Consistent with the notion of crosstalk between regulatory mechanisms, *phyB* single mutants, *phyA phyB phyD* triple mutants, and *pft1* single mutants all show a slight elevation of *FLC* expression at 22 °C [68, 100]. In addition, sensitivity to red light reduced (SRR1), a protein involved in phyB signaling and circadian clock regulation, enhances *FLC* expression, and the mutant shows early flowering. These observations suggest that phytochrome signaling is integrated into the vernalization pathway and fine-tunes *FLC* expression [101, 102]. Interestingly, a feedback mechanism from FLC to light signaling was also observed. *CRY2* expression levels were decreased when both functional *FRIGIDA* and *FLC* alleles were present, but increased when vernalization was applied. These results indicate that *CRY2* expression is suppressed in response to the *FLC* expression levels [103], but little is known about the detailed mechanism.

Since *FLC* has been shown to function in the leaves of vegetative plants to repress *FT* expression in the companion cells of the phloem [104], photoreceptors may also function in vasculature in the vernalization pathway. Support for this idea comes from the finding that a phyB-interacting protein, VOZ1, is expressed only in vascular phloem [98].

Intermediate temperature pathway

In addition to the vernalization pathway, the intermediate temperature pathway also has a tight link with the photoreceptor-mediated light signaling pathway. Halliday and colleagues focused on the crosstalk between photoreceptors and ambient temperature [100]. As mentioned above, *phyB* mutants show early flowering at 22 °C, an optimal temperature for *Arabidopsis* growth. However, when plants were grown at 16 °C the *phyB* mutants and wild type had similar flowering times, indicating that phyB regulates flowering efficiently at 22 °C but not at 16 °C. At 16 °C, phyE plays a major role in flowering regulation. In the *phyA, phyB, phyD* triple mutant background, a *phyE* mutation manifests a significant early flowering phenotype at 16 °C [100]. Furthermore, *cry1*, *cry2,* and a *phyA cry2* double mutant all show severe late flowering at 16 °C, suggesting that major phytochromes and cryptochromes regulate flowering in a thermosensory pathway [101]. However, a *terminal flower1* (*tfl1*) mutation abolished the temperature response in cryptochrome mutants, but not in a *phyB* mutant. By contrast, an *elf3* mutation can suppress the temperature response in a *phyB* mutant, suggesting that there are at least two or more pathways that integrate light signals into the thermosensory pathway [102].

High ambient temperature causes not only early flowering but also other SAR-like features, such as long hypocotyls and petioles [103]. PIF4 is a good candidate that links photoreceptors and ambient temperature signaling, because a PIF4 overexpressing line displays a SAR-like phenotype, including early flowering and long hypocotyls [104]. Indeed, PIF4 protein accumulation is negatively regulated by the Pfr form of phyB [105, 106]. There is, however, another FVE- and FCA-mediated ambient temperature pathway, and genetic interactions between these genes and photoreceptors have been demonstrated [103].

We do not know yet if temperature sensing and integration into light signaling occur in a specific tissue. Circadian clock studies have demonstrated that there are two different circadian clocks: *catalase 3* (*CAT3*)::*luciferase* (*LUC*) and *chlorophyll A/B-binding protein 2* (*CAB2*)::LUC oscillation is affected by temperature cycles and light/dark cycles, respectively [107]. From gene expression patterns of *CAT3* and *CAB2*, Michael et al. hypothesized that a temperature-sensitive circadian clock may exist in the epidermis. This hypothesis appears to be convincing, because epidermis is located in the outer layers, where fluctuations of air temperature are easily detected. Indeed, such a tissue-specific function of the circadian clock in *Arabidopsis* has recently been described, and this epidermal clock is highly important for processing the intermediate temperature signal [89]. Interestingly, however, the epidermal clock regulates cell elongation including hypocotyl and petiole elongation but does not affect flowering time at all. Instead of the epidermal clock, vasculature clocks are involved in intermediate temperature-dependent flowering. This result does not directly mean that the photoreceptors that are involved in the intermediate temperature flowering pathway necessarily function in the vasculature, but FVE::GUS and FCA::GUS lines displayed a vascular-enriched GUS staining pattern, suggesting the importance of vasculature for intermediate temperature signaling [108, 109]. Future studies will reveal which tissue(s) express the photoreceptors that regulate flowering in response to intermediate temperatures.

Gibberellin pathway

SRR1 plays an important role in the regulation of the circadian clock and phyB signaling [110]. SRR1 stimulates various *FT* transcription repressors including CDF1, tempranillo (TEM1 and TEM2) and FLC, and suppresses flowering under non-inductive SD conditions. Although TEM1 and TEM2 are known as antagonists of CO binding to the *FT* promoter [111], these also repress GA3OXIDASE1 (GA3OX1) and GA3OX2 in the gibberellin biosynthesis pathway [112]. Furthermore, key negative regulators in gibberellin signaling, DELLA proteins, are crucial inhibitors of PIF4 transcription activity. DELLAs interact with PIF4 and impede its DNA-binding ability [113]. Therefore, light signaling and gibberellin signaling are integrated via control of PIF4-mediated transcription activity.

Gibberellin-mediated flowering is also regulated in specific sites. In the leaf vasculature, DELLA proteins regulate *FT* expression under LD condition, independent of CO and GI functions [116]. Furthermore, gibberellin signaling promotes flowering independently of photoperiod through the regulation of *squamosa promoter-binding protein-like* (*SPL*) genes in both leaves and shoot apical meristem [114, 115].

Perspective

As documented in the present review, phytochromes, cryptochromes, and ZTL/FKF1/LKP2 family proteins regulate flowering through multiple factors in various pathways and various specific tissues (Fig. 2). Although many factors are involved in both photomorphogenesis and flowering regulation, some are involved only in flowering regulation; therefore, future detailed tissue-specific studies should dissect these two closely related but different pathways. Also, some flowering pathways utilize the same factor as a key regulator. For example, PIF4 has crucial functions both in the light quality and intermediate

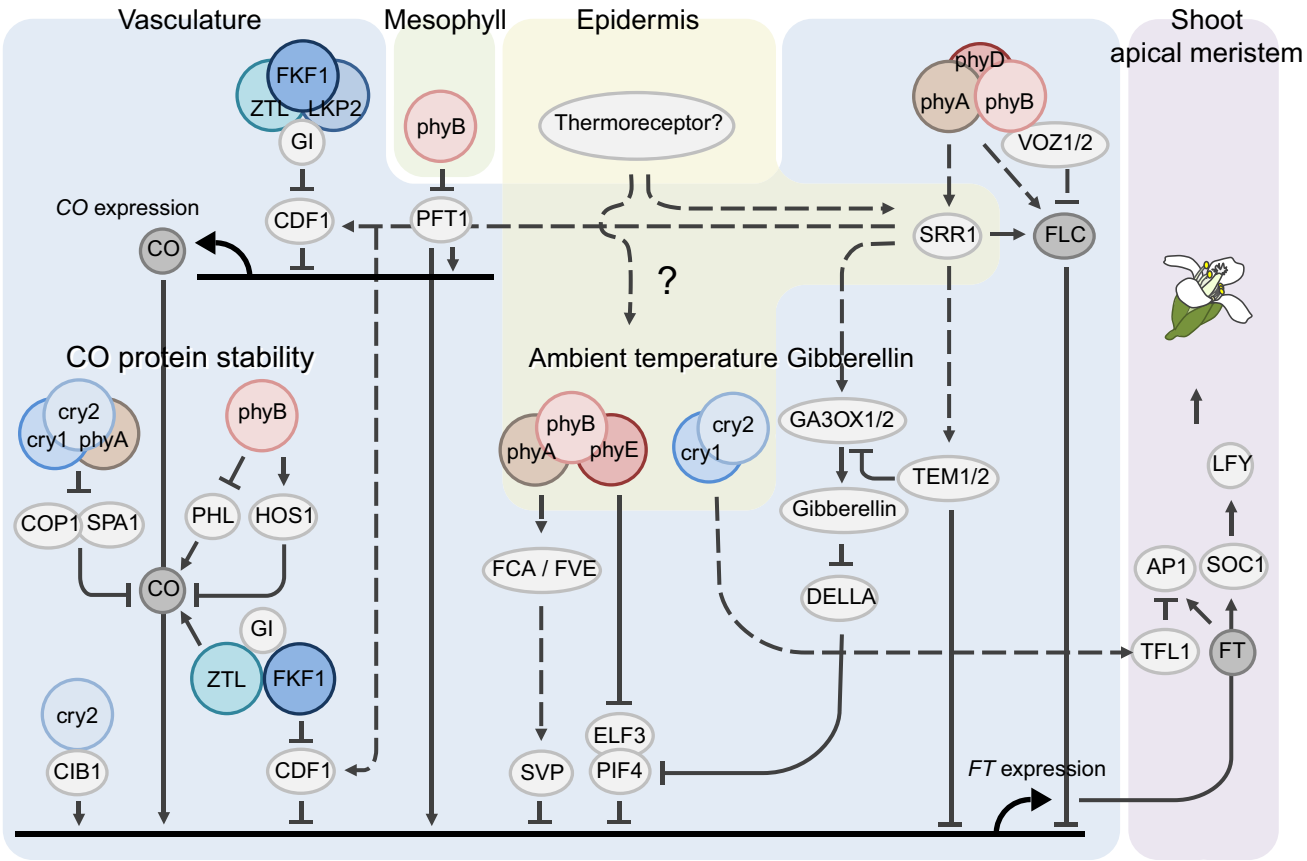

Fig. 2 Phytochromes, cryptochromes, and ZTL/FKF1/LKP2 tissue specifically regulate multiple flowering pathways. Direct and indirect transcriptional regulation of *CO*, *FT*, and *FLC,* and post-translational regulation of CO protein by photoreceptors and tissues in which the signaling pathway is processed. The *red/far-red* photoreceptors are depicted in *red*, *blue light* photoreceptors in *blue*. Intermediate temperature signaling may be initiated from a hypothetical thermoreceptor in epidermis. Photoreceptors in unknown tissue(s) might crosstalk to the intermediate temperature signaling pathway and FCA/FVE, and then downstream factors regulate *FT* expression in the vasculature. In the light quality pathway, phyB in mesophyll regulates *FT* expression in the vasculature through unknown inter-tissue signaling. The photoperiod pathway, including CO expression, CO protein stabilization, and *FT* expression, is regulated in the vasculature. Tissue-specific analyses have demonstrated that at least cry2, SPA1, and COP1 in vasculature have dominant roles in the photoperiod pathway, even though these genes are expressed in most cell types. The vernalization and gibberellin pathways may also function in vasculature. The regulation of CO, FT and FLC (depicted in *gray*) in vasculature is especially crucial for flowering regulation by photoreceptors. *Solid* and *dashed lines* indicate direct and indirect regulation, respectively

temperature pathways. It is conceivable that tissue-specific expression or activity of a photoreceptor signaling molecule may prevent miscommunication among different pathways in response to different light signals, and as such may serve as a precise means to regulate flowering, thus optimizing a plant's adaptation to its surroundings. Identification of the principle tissue for light quality or temperature sensing would aid in deciphering each signaling pathway and how photoreceptors are involved in it. As a case in point, the discovery of vasculature-specific *FT* expression led to a breakthrough in the elucidation of photoperiodic flowering mechanisms.

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