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Invited Review

Emerging Hubs in Plant Light and Temperature Signaling†

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ABSTRACT

Due to their nature as sessile organisms, plants must accurately sense their surroundings and then translate this information into efficient acclimation responses to maximize development. Light and temperature are two major stimuli that provide immediate cues regarding energy availability, daylength, proximity of other species and seasonal changes. Both cues are sensed by complex systems and the integration of these signals is of very high value to properly respond to environmental changes without being disguised by random changes. For instance a cold day has a different significance if it occurs during the illuminated phase of the day or during the night, or when days are shortening during the fall instead of a long-day in spring. Here, we summarize recent advances in the nature of signaling components that operate as connectors of light and temperature signaling, with emphasis on the emerging hubs. Despite the nature of the thermosensors is still in its infancy compared to an important body of knowledge about plant sensory photoreceptors, the interaction of both types of signaling will not only bring clues of how plants integrate environmental information, but also will help in leading research in the nature of the thermosensors themselves.

INTRODUCTION

For sessile organisms, the degree of fitness is heavily dependent on how well these individuals grow and adapt to environmental changes and how timely they make developmental transitions such as germination and flowering. Rapid acclimation to daily variations plus a precise estimation of seasonal changes become very important mechanisms of survival. The perception of ambient light and temperature changes are hence of primary importance.

Globally, plant populations show well-adapted physiological responses that allow them to develop in extreme thermal and lighting conditions ranging from very hot arid lands, high altitude lands, to cold tundra. But plants also evolved to respond to common and less stressful ambient temperature shifts (12–28°C), which result in morphological, anatomical and metabolic adaptations, enabling plants to improve their fitness. Some light conditions elicit very similar changes (1). Since both signals regulate common plant responses, it is not surprising they share signaling elements. How are then light and temperature signaling integrated?

Light signals are perceived and transduced by a set of chromoproteins known as sensory photoreceptors, which are highly conserved in different plant species (2). The phytochromes are among the most important sensory photoreceptors in plants (3), which may be found in two spectrophotometrically distinct and interconvertible forms: the red light (R ~660 nm) absorbing form (*Pr*) which is also inactive, and the far-red light (FR ~730 nm) absorbing form (*Pfr*) which is active. Their spectral properties are due to a linear tetrapyrrole chromophore, the phytochromobilin. Plants also possess three families of UV-A/Blue absorbing photoreceptors: the cryptochromes, the phototropins and the members of the Zeitlupe family (4–6); and the UVR8 photoreceptor which absorbs UV-B light (7,8). Thirteen photoreceptors were found in Arabidopsis, but the total number of photoreceptors varies among species due to gene duplication within each family.

In early photomorphogenic studies, action spectra helped in hypothesizing the existence of photoreceptors (9). On the contrary, plant responses to temperature did not aid in hypothesizing about the nature of the thermoreceptor(s). Temperature perception could be as complex as the number of biochemical reactions that take part inside a cell and defining a "thermosensor" has remained a challenge. Calcium channels in the plasma membrane are among the first identified components of a putative thermosensor (10). These channels respond to both cold and heat changes and trigger a fast Ca^{2+} influx into the cytosol (11). In bacteria, membrane fluidity is a key element in temperature perception (12,13). Finka and Goloubinoff (14,15) have performed experiments on transgenic *P. patens* and theorized that changes in the plasma membrane fluidity due to temperature shifts can be perceived by Cyclic Nucleotide Gated Ion Channels (CNGCs). These channels permeate Ca^{2+} and cause the activation of Calcium/Calmodulin-dependent kinases (16). The cytosolic heterotrimeric G-protein of the ARF family have a proposed mechanism activating the guanylate cyclase, producing cGMP and thus providing CNGCs with a substrate to function, but how G-Protein senses temperature itself is still not clear. A recent report analyzing proteomic signatures highlights numerous light and temperature responses associated with cAMP, fueling the theory of a possible adenylate cyclase with roles as a temperature sensor in plants (17).

In addition to membranes and membrane proteins as potential thermosensors, recent reports point to chromatin-related pro-

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cesses as possible thermosensors. A model was proposed, where H2AZ containing nucleosomes provide thermosensory information to coordinate the response of the transcriptome to ambient temperature. When temperature increases, H2AZ decreases at +1 nucleosomes, allowing an increase in the expression of temperature-responsive genes, such as *HSP70* and *FLOWERING LOCUS T* (*FT*) (18,19). This model requires H2AZ behaving as a repressor of transcription. However, it is not straightforward to reconcile this H2AZ role with more recent findings. First, the decrease in H2AZ also occurs in genes that do not respond to temperature (19). Second, recent findings show that *in vivo* (insect cells), +1 nucleosomes form a barrier for RNA polymerase II; which stalls more frequently at 8–13 bp within the first nucleosome (20). Furthermore, RNA polymerase II stalling decreases as H2AZ increases in +1 nucleosomes and H2A.Z reduces the nucleosome barrier to RNAPII, leading to increased elongation (20). Given that there is evidence in Arabidopsis that the presence of H2AZ correlates with transcription (21,22), the role of H2AZ in reducing the +1 nucleosome barrier to RNA polymerase II might also be conserved in plants. Therefore, we cannot rule out the possibility that plants may use H2AZ as a temperature compensation mechanism for transcription. In this way, increasing H2AZ in +1 nucleosomes at lower temperatures (19), may compensate for the increase in the energetic barrier RNA polymerase II has to overcome when reaching the +1 nucleosome at lower temperatures.

Finally, we also cannot dismiss the possibility that temperature effects on the activity of the photoreceptors themselves (23–25), including dark reversion (26), to be partly responsible for triggering physiological responses.

Having introduced putative temperature sensing mechanisms, we will proceed to analyze the cross talk between light and temperature responses and its impact in plant architecture and phase transitions.

INTERACTIONS BETWEEN LIGHT AND TEMPERATURE IN THE CONTROL OF GERMINATION

Pioneering experiments performed on lettuce seeds by Takaki & Zaia (27) suggested that light and temperature act at common points to promote germination. These initial observations have now, at least in some cases, molecular explanations.

Seed dormancy prevents viviparity and ensures germination only after certain key conditions have been perceived. Whether seeds enter dormancy or germinate depends on the balance between two hormones: abscisic acid (ABA), which promotes dormancy (28); and gibberellic acid (GA) which helps to break dormancy and promotes germination (29). This balance is influenced by various light and thermal *stimuli* (Fig. 1a).

In some plants, imbibition and appropriate light conditions are not enough for seeds to break dormancy; they also need a minimal cold exposure after imbibition. This process is called stratification (30). In Arabidopsis, phytochromes are the sole photoreceptors that promote germination (31). Using Arabidopsis mutants deficient in different phytochromes it was shown that the hierarchy of their action is influenced by temperature: phyB promotes germination across a wide range of temperatures, whereas phyA is more important at warmer temperatures and phyE at cooler temperatures (32). More recent reports have established that phytochrome signaling and light interactions are

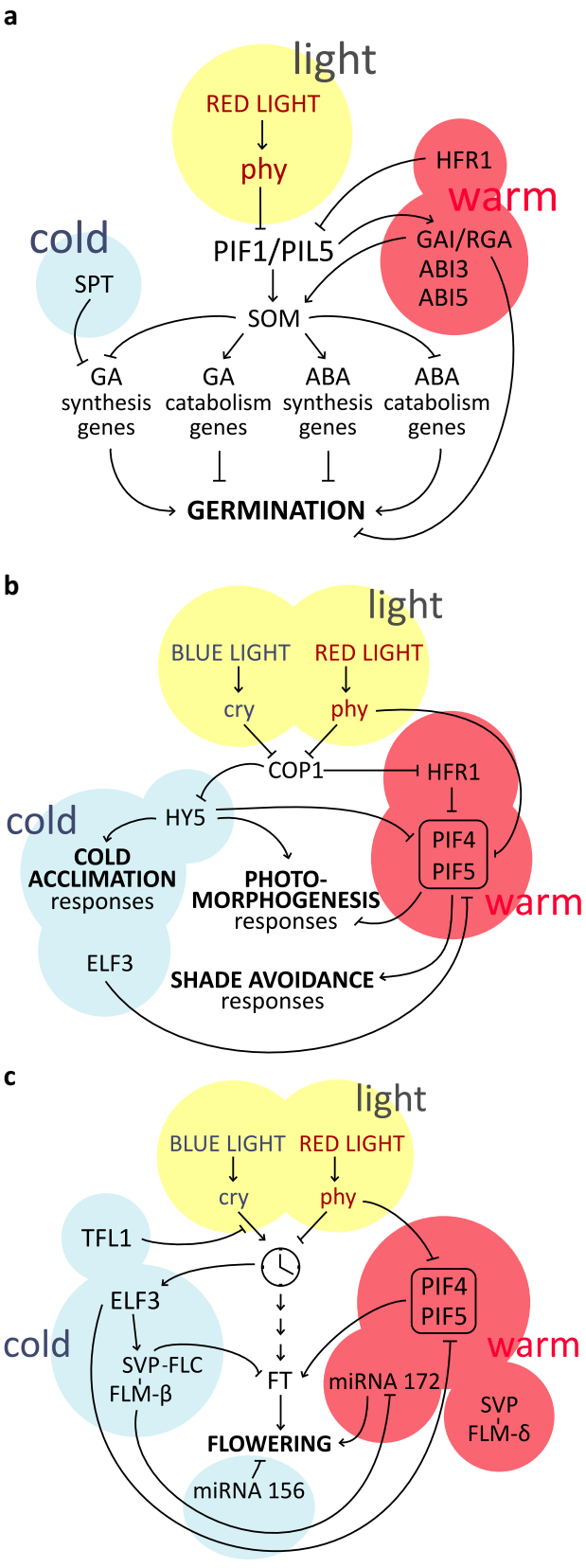

Figure 1. Simplified signaling pathways for Germination (a), Shade Avoidance, Cold Acclimation and Photomorphogenesis responses (b) and Flowering (c) in Arabidopsis, highlighting genes involved in light (yellow circles), and temperature cross talk (blue and red circles for genes involved in low and warm temperatures, respectively).

also important during seed maturation. Maturation under cool temperatures promoted dormancy and in this case phyB and phyD were required to break this cool-induced dormancy (33). In addition, effects of temperature in maternal tissue during seed maturation were more effective than the photoperiod, another very important environmental clue. In a more recent report, it was shown that *FT* controls seed dormancy in response to temperatures affecting maternal tissue (34). *FT* expression decreases in siliques of plants that were exposed to lower temperatures (16°C *vs* 22°C), leading to higher mRNA expression of phenylpropanoid pathway genes, higher procyanidin and tannin levels in the seed coat and lower permeability, leading ultimately to lower germination rates (34,35). Given that during flowering induction the *FT* gene is the target of several flowering pathways, including the light quality pathway perceived by phytochromes (36–38), it would be interesting to know if *FT* is also integrating light and temperature signals in the maternal tissue during seed development.

Phytochromes transduce light signaling by binding to and promoting the degradation of a small subset of transcription factors that belong to the basic helix-loop-helix family (bHLH). Seven of these transcription factors bind phytochromes in a R/FR reversible manner and thus were named PHYTOCHROME INTERACTING FACTORS (PIF) or PHYTOCHROME INTERACTING FACTOR3-LIKE (PIL): PIF3, the founding member (39), PIF4, PIF5 (also known as PIL6), PIF1 (also known as PIL5), PIF6 (also PIL2), PIF7 and PIF8 (also UNE10) (40).

These transcription factors act mainly as repressors of phytochrome-promoted responses. PIF1 has been identified to be an inhibitor of germination (41). It reduces GA content by repressing GA synthesis genes (*GA3ox1* and *GA3ox2*), and by activating GA catabolic genes (*GA2ox2*) (41,42). On the other hand, PIF1 increases ABA content by activating the expression of ABA synthesis genes (*ABA1*, *NCED6* and *NCED9*) and repressing the ABA catabolic gene *CYP707A2* (42,43). Therefore, PIF1 favors the GA/ABA balance toward ABA and this balance is then reversed once red light activates phytochrome which in turn promotes the degradation of PIF1 (42). However, PIF1 does not regulate GA and ABA metabolism directly (44), but indirectly through the action of its direct target SOMNUS (SOM), a CCCH-type zinc finger nuclear protein (45). In addition to regulating ABA and GA metabolism, PIF1 also regulates GA signaling. It promotes the transcription of *GA INSENSITIVE* (*GAI*) and *REPRESSOR OF GA* (*RGA*), two of the five DELLA genes which encode repressors of GA responses and are themselves degraded upon binding to the GA containing GIDs (GIBBERELLIN INSENSITIVE DWARF), the GA-receptors (46). PIF1 also binds to the promoters and induces the transcription of positive ABA signaling regulators: the transcription factors ABA INSENSITIVE 3 (ABI3) and ABA INSENSITIVE 5 (ABI5) (44). PIF1 and ABI3 interact and also regulate SOM mRNA expression in a collaborative manner (47). High temperatures (around 30°C in Arabidopsis) inhibit germination, mainly by activating ABA synthesis (48). Here, is precisely a point of connection between light and temperature signaling: ABI3, ABI5 and DELLA proteins act together to activate *SOM* expression in response to higher temperatures (49). Therefore, light and temperature share the same target, *SOM,* in addition to the aforementioned facts, that PIF1 also targets *ABI3*, *ABI5* and *DELLAs* (*GAI* and *RGA*) promoters (43,44). However, they play opposite roles, whereas light promotes germination through the degradation of PIF1, high temperature inhibits germination by increasing SOM levels. This antagonistic role between both signals is a clear sign of how germination in supraoptimal thermal conditions can be downregulated even in the presence of proper light requirements.

Another important interaction point of light and temperature are the GA metabolism genes. *AtGA3ox1* and *AtGA3ox2* expression increases in response to light perceived by phytochromes through the action of PIF1, and conversely, *AtGA2ox2*, involved in GA catabolism, decreases (43,44,50). Of these genes, *AtGA3ox1* and *AtGA3ox2* are regulated similarly by low temperatures (around 5°C) during imbibition (51). Thus, at least these two GA metabolism genes are convergence points between light and temperature signals. Another transcription factor, SPATULA (SPT), (also a bHLH), was shown to repress the expression of *AtGA3ox1* and *AtGA3ox2* and to be necessary for the stratification requirement. On the other hand, PIF1 represses *AtGA3ox1* and *AtGA3ox2* in the dark and is necessary for the light requirement. Hence, the light and stratification requirement for germination are due, at least in part, to the action of both SPT and PIF1 on *AtGA3ox1* and *AtGA3ox2* expression, although, indirectly by PIF1 (43,52).

LIGHT AND TEMPERATURE SIGNALING INTERACTIONS DURING SHADE AVOIDANCE

Common factors act during early seedling emergence and shade avoidance responses

When seedlings emerge, skotomorphogenesis is the default developmental program: seedlings elongate to reach soil surface with closed apical hook and cotyledons to protect the apical meristem during emergence from soil. Once seedlings reach light, the photomorphogenic program is triggered by sensory photoreceptors; the phytochromes and cryptochromes play prominent roles during this seedling stage. Once seedlings green-up, the changes in light quality are important to monitor the presence of plant neighbors, which may produce shading (actual competitors) or may compete for the light resource in the future [reviewed by (53)]. As photosynthetic tissues absorb light in the UV-Red region of the spectrum, the proportion of FR light reflected or transmitted by plant tissues is increased in the shade or in the presence of neighbors, even without actually shading. The relative increase in FR light reverts phytochrome to its inactive form, Pr. As a result, stems and petioles elongate and leaves bend upward (hyponasty). These responses are important to avoid shade, even before actual shading, and are known collectively as Shade Avoidance Responses (SAR). phyB is the most important photoreceptor mediating shade responses; *phyB* mutants display a constitutive Shade Avoidance Syndrome (SAS). The factors regulating seedling photomorphogenesis and the responses to shade overlap; some of the factors that promote skotomorphogenic development also induce the SAR. Among these factors are the PIF transcription factors, which activate elongation of hypocotyls and stems (54), and senescence (55). Phytochromes, mainly phyB and to a lesser extent phyA, antagonize the action of PIFs by preventing them from binding to their targets (56), and promoting their phosphorylation and degradation by the proteasome. For instance phyB induces the phosphorylation and degradation of PIF3 (57–59), PIF4 and PIF5 (60), and phyA and phyB promote the phosphorylation and degradation of PIF1. Conversely, the PIFs also regulate the activity of phytochromes by promoting their destruction (61). This way,

phytochromes inhibit the responses to shade by inactivating PIFs. Further downstream, the PIFs function as connectors of light, hormone and temperature signaling [reviewed by (62)]. Both auxin biosynthesis and transport are necessary to induce hypocotyl, stems and petioles elongation in response to shading conditions (63–66). The PIFs connect light signaling with hormone signaling in a direct way: PIF4 and PIF5 bind to regulatory regions and directly regulate the transcription of *YUCCA* genes encoding auxin biosynthesis enzymes and *IAA* genes involved in auxin signaling (67). *LONG HYPOCOTYL IN FR LIGHT1* (*HFR1*) was also found among the direct targets of PIF4 and PIF5. HFR1 is shade induced and it is involved in part of a feedback mechanism that prevents excessive SAS responses, by forming nonfunctional heterodimers with PIF4 and PIF5 (68). PIF7, which is more stable than PIF4 and PIF5 and inactivated by phosphorylation, also activates *YUCCA* genes to rapidly induce auxin synthesis in response to shade (69).

Responses to shade and temperature have common grounds: PIF4 as a hub

Higher temperatures (27–30°C for Arabidopsis) trigger changes in plant architecture that are similar to the SAS, including elongation of hypocotyls and stems and accelerated flowering (1,70) (Fig. 1b,c). Elongation responses that occur during exposure to higher temperatures are also dependent on increased auxin levels (1,71). These auxin-dependent responses to temperature are mediated by (71,72), and seem to involve increased binding of PIF4 to the promoters of genes involved in auxin biosynthesis, *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1* (*TAA1*), *CYP79B2* and *YUCCA8* and *IAA* genes involved in auxin signaling (67,73,74). As a result, PIF4 is a node for temperature and light signaling integration.

PIF4 activity is temperature dependent

PIF4 and PIF5 are regulated by the circadian clock at the mRNA level and their expression rises during mid-night time and the light phase to decrease again in the evening and early night. Protein levels are low in the light phase due to phytochrome-enhanced PIF degradation. This way, PIF4 and PIF5 highest activity peaks during late night triggering hypocotyl elongation (75). Hypocotyl elongation is dependent on photoperiod, hypocotyls are taller under Short Days (SD) than under Long Days (LD). This effect depends on PIF4 and PIF5, as *pif4 pif5* double mutants are equally short in both SD and LD and the peak of *PIF4* expression in WT plants occurs just before dawn only under SD, but not LD (76). When temperature increases, from 22 to 28°C, the maximum level of *PIF4* mRNA also occurs before dawn in LD, and higher PIF4 protein levels cause increased elongation and auxin-related genes expression (76,77). Under continuous light, the expression of both PIF4 and PIF5 mRNAs where shown to increase after a shift from 20 to 29°C and the increase in hypocotyl length at warmer temperatures observed in WT plants almost disappeared in *pif4 pif5* double mutants (71,72,74,78).

More recent studies demonstrated that PIF4 is regulated at the protein level by both light and temperature (79). As mentioned above, phytochromes promote phosphorylation and degradation of PIF4. On the contrary, higher temperatures promote the accumulation of hyper-phosphorylated forms of PIF4 (79). To this temperature regulation of PIF4, the same authors added another twist. They found that HFR1, which inhibits PIF4 by forming a bHLH heterodimer incapable of binding DNA (68), is also elevated under warm temperatures restraining excessive elongation (79). However, increased PIF4 protein stability at warmer temperatures was not found in all conditions, including continuous light (71), therefore other mechanisms may explain the increase in PIF4 DNA binding activity at warmer temperatures (18).

ELF3 plays an important role in temperature and light signaling cross talk

EARLY FLOWERING 3 (ELF3) is involved in both the regulation of flowering time and hypocotyl elongation (80,81). The ELF3 protein seems to work as an adaptor protein (82). Within the circadian clock, ELF3 forms part of the Evening Complex (EC) together with ELF4 and the transcription factor LUXARRHYTHMO (LUX). The EC represses the expression of *PIF4* and *PIF5* in the early evening; when the EC decreases, it relieves their expression late in the night period (83). *PIF4* expression increases in *elf3* mutants mainly during the night as so do PIF4 gene targets (76). However, the involvement of ELF3 in temperature responses seems to be more direct than anticipated for its role in the EC.

ELF3 was originally proposed to be involved in ambient temperature responses after *elf3* mutants being less sensitive to the delay in flowering time imposed by lower temperatures (16°C) in comparison with optimal temperatures (23°C), and the *elf3* transcriptome being similar to the transcriptome of WT plants grown at lower temperatures (84). Afterward, *elf3* mutants were shown to be insensitive to warm temperature pulses, showing a constitutive high expression of clock-related genes *PRR7*, *PRR9* and *GI* and also *PIF4* (85). Furthermore, the expression of several clock genes and *PIF4* and *PIF5* were studied in short days at 22 and 28°C in the EC mutants, including *elf3*. It was determined that EC represses the expression of *PIF4* and *PIF5* in a temperature-dependent manner, explaining the hypocotyl response to warm temperature (86,87). More interestingly, the binding of the EC components ELF3 and ELF4 to the PIF4 promoter decreased at higher temperatures providing a molecular mechanism for transducing temperature signaling to hypocotyl growth through ELF3 (86,87). When the temperature effect on the binding activity of the EC to the *PIF4* promoter was introduced in a mathematical model, the temperature effect on hypocotyl growth was recapitulated (88).

The role of ELF3 in signaling temperature was proposed to be independent of the EC complex because the *lux* mutants still responded to temperature (86,87), but we cannot rule out a redundant role for the LUX homolog NOX. It would be interesting to study the response to temperature in *lux nox* double mutants. Finally, an EC independent role for ELF3 was recently shown as ELF3 directly binds to PIF4, inhibiting its DNA binding activity and thus preventing hypocotyl elongation (83,89).

HY5 antagonizes PIF4 in the control of temperature responses

As mentioned above, the PIF proteins are mainly promoters of skotomorphogenesis. On the contrary, other transcription factors like the bZIP transcription factor LONG HYPOCOTYL 5 (HY5) are promoters of photomorphogenesis (90,91). In the dark, HY5 is degraded by the proteasome in a CONSTITUTIVE PHOTO-

MORPHOGENIC 1 (COP1) dependent fashion (92); in the light, HY5 is stabilized to promote deetiolation (93,94). Interestingly, HY5 is also temperature-regulated, but unlike PIF4, its mRNA transcription increases at lower temperatures and the HY5 protein is also stabilized by low temperatures (78,95).

During deetiolation, the accumulation of photosynthetic pigments is highly dependent on HY5. During this process, moderate low temperatures (17°C) and red light also stabilize HY5, which promote the synthesis of chlorophyll and carotenoids pigments. Loss of HY5 function causes a lack of red-light induction of these photosynthetic pigments at lower temperatures (17°C compared to 27°C). Hence, HY5 is another hub for temperature and light signaling integration (96). To evaluate the molecular mechanisms of HY5 temperature-dependent regulation of pigment synthesis, the promoters of synthesis genes were evaluated for HY5 binding *in vivo*. HY5 binding to these promoters was more evident at lower temperatures and antagonized the binding of PIF1 (PIF1) and PIF4 to the same binding motifs. Therefore, HY5 promotes the expression of these genes at lower temperatures, whereas the PIFs repress them at higher temperatures, competing for the same cis-acting elements (96).

Connections between HY5 and PIF4 do not end on competition for promoter binding. For instance HY5 negatively regulates *PIF4* mRNA expression and its effects are more evident for the hypocotyl response at higher temperatures. The *hy5* hypocotyls are taller than WT hypocotyls in warm temperatures, but this hypersensitive response is suppressed by PIF mutations (78).

LIGHT AND TEMPERATURE INTEGRATION TO REGULATE FLOWERING TIME

Light regulates flowering in two main ways. Plants may respond to light quality signals like those that trigger the SAR (36,38) or to photoperiods, which are informative of seasonal changes (97). Plants that flower when days are increasing in length are known as LD plants and plants that flower when days are shortening are known as SD plants. Temperature also affects flowering in two main ways. Some cultivars of wheat and barley, for instance or some Arabidopsis accessions, require a prolonged exposure to low, but nonfreezing temperatures, to properly respond to photoperiodic signals (increase in day-length) once spring approaches. This effect of long exposures to cold is known as vernalization, a mechanism to ensure overwintering and was reviewed elsewhere (98,99). But temperatures around optimal growth temperatures (12–28°C in Arabidopsis) also modulate flowering time; in Arabidopsis, lower temperatures inhibit flowering whereas higher temperatures promote it. The genetic analysis of the flowering response to ambient temperature led to the proposition of a thermosensory pathway (100). Since its original proposition, the thermosensory pathway was believed to interact with light signaling, since mutations in phyA and cry2 modified the response to ambient temperature (100). However, this pathway seems to be more complex than originally anticipated. Genetic evidence suggests that at least two pathways take part in the thermosensory pathway (84). In this section, we review the interactions between light signaling and the thermosensory pathway (Fig. 1c).

Light and temperature integration at the level of PEBP genes

The different pathways that regulate flowering, the vernalization pathway, the photoperiod pathway, the thermosensory pathway, the autonomous, age and the hormone pathways are integrated at the transcription level of a small group of genes. For this reason, these genes are known as "flowering pathway integrator genes". *FT* and the transcription factor genes *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*) and *LEAFY* (*LFY*) are the most studied "integrator" genes. Once the expression of these genes is activated, flowering is induced. The *FT* gene encodes the FT protein that works as a moving signal and is conserved among angiosperms. FT is generated in the leaf-phloem tissue and acts in the meristem to promote the floral transition (101–105).

The *FT* gene is a convergence point for light and ambient temperature signaling since it is regulated by both light (light quality and photoperiod) and temperature (36–38,97,100,106–108). Consistently, the flowering time of *ft* mutants is less sensitive to ambient temperature (106,109). The regulation of *FT* expression in response to both photoperiod and temperature has been successfully modeled by adding FT repressors with temperature-dependent activity to existing circadian clock models, therefore relatively simple gene circuits can explain the integration of photoperiod and temperature signals into flowering (88).

FT belongs to a small family of proteins with homology to phosphatidylethanolamine-binding proteins (PEBPs) [reviewed by (110)]. The role of the six Arabidopsis PEBP genes in temperature-dependent flowering was thoroughly studied and it was shown that *FT*, *TWIN SISTER OF FT* (*TSF*) and *TERMINAL FLOWER 1* (*TFL1*) play the most important roles (109). The number of genes within this family differs among species. However, they can be found essentially in two main groups. The *FT*-like genes that promote flowering and the *TLF1*-like genes that repress flowering (111). *TSF1* is the closest *FT* homolog in Arabidopsis; it also responds to light and photoperiod cues (112), and plays roles in temperature signaling (109) functioning as another convergence point for light and temperature pathways, although with a less prominent role in comparison with *FT*.

TFL1 has a prominent role in temperature signaling. The transcriptome of *tfl1* mutant strongly suggests that TFL1 is a positive regulator of responses to lower (16°C) temperatures (84). The loss of TFL1 causes earlier flowering at 16°C than at 23°C (84,109,113). Conversely, the overexpression of TFL1 has stronger effects at 22°C than at 16°C, mimicking the effect of lower temperatures and saturating its effects (113). These facts suggest a role for TFL1 as a repressor of flowering under lower temperatures. The intersection with light signaling is not evident, but it is interesting that, genetically, TFL1 acts downstream cryptochromes cry1 and cry2 as a negative regulator of their signaling in SD, conditions where the photoperiod pathway is inactive (114). Interestingly, in strawberry, *TFL1* is temperature-regulated and sets the photoperiodic requirements (115). It is possible then that the role of TFL1 in the intersection of photoperiodic and temperature signaling may be at least partially conserved in the Rosaceae. Finally, as is the case for *ft*, *tfl1* mutants show some degree of sensitiveness to temperature. However, when combined with an *elf3* mutation, this sensitivity almost disappears, suggesting that *elf3* and *tfl1* affect two different pathways (84).

Transcription factors as convergence points

The thermosensory pathway works with a suite of transcription factors and their regulators to control the flowering response. Among these regulators, microRNAs play important roles; miR172 and miR156 are responsive to temperature and also

Table 1. Key genes implicated in the different plant development pathways regulated by light and temperature. Genes are ordered in the table in descending hierarchy in the signal pathway. Genes with a signal relay function are underlined. ¨\¨ separates different genes.

	Gene/s	Environmental Input	Pathway Role	Role on effector	Effector	References
Germination	phyA/phyB	Light	Promoter	Protein degradation	*PIF1/PIL5*	(32, 33)
	HFR1	Warm temp.	Promoter	Nonfunctional heterodimer formation	*PIF1/PIL5*	(134)
	PIF1/PIL5	Light/Warm temp.	Repressor	Transcriptional activation	*SOM/GAI/RGA*	(41–44)
	SPT	Cold temp.	Repressor	Transcriptional repressor	GA synthesis genes	(52)
	SOM	Light/Warm temp.	Repressor	Transcriptional repression	GA synthesis/ABA degradation genes	(45,47)
			Repressor	Transcriptional activation	GA degradation/ABA synthesis genes	
	GAI/RGA	Warm temp.	Repressor	Transcriptional repression	*SOM*	(43,44,49)
	ABI3	Warm temp.	Repressor	Transcriptional repression	*SOM*	(43,44,49)
	ABI5	Warm temp.	Repressor	Transcriptional repression	*SOM*	(43,44,49)
Photomorphogenesis-shade avoidance	phyB	Light	Promoter	Protein degradation	*COP1 PIF1/PIF3/ PIF4/PIF5*	(53,56,79,92)
	cry1	Light	Promoter	Inhibition of protein degradation	*COP1*	(53,79)
	COP1	Light	Repressor	Protein degradation	*HFR1/HY5*	(53,92)
	HY5/HYH	Cold temp.	Promoter	Transcriptional activation	Cold Acclimation genes	(78,90–96)
		Cold temp.	Promoter	Transcriptional repression	*PIF1/PIF4*	
	ELF3	Cold temp.	Promoter	Inhibition of DNA binding	*PIF4/PIF5*	(80,81,86–89)
	HFR1	Light/Warm temp.	Promoter	Nonfunctional heterodimer formation	*PIF4/PIF5*	(68,79)
	PIF4	Light/Warm temp.	Repressor	Transcriptional activation	*IAA*/YUCCA genes	(67,71–78)
	PIF5	Light/Warm temp.	Repressor	Transcriptional activation	*IAA*/YUCCA genes	
Flowering	phyB	Light	Repressor	Protein degradation	Flowering time genes; *PIF4/PIF5*	(56,60,84)
	phyA	Light		Protein stabilization	Flowering time genes	(56,100)
	cry2	Light	Promoter	Protein stabilization	Flowering time genes; Cryptochrome interacting genes	(100)
	PIF4	Warm temp.	Promoter	Transcriptional activation	*FT*	(18,71,79,132)
	PIF5	Warm temp.	Promoter	Transcriptional activation	*FT*	(72)
	TFL1	Light/Cold temp.	Repressor	Transcriptional repression	Meristem Identity genes	(84,109,113–115)
	ELF3	Cold temp.	Repressor	Protein stabilization	SVP/FLM/FLC complex/PIF4/PIF5	(84,88,130,131)
	SVP -FLMβ-FLC	Light/Cold temp.	Repressor	Transcriptional repression	FT/miRNA172	(70,127,130,131)
	SVP - FLMδ	Warm temp.	Promoter	Dominant negative	*FT*	(70,126,127,130,131)
	miRNA172	Warm temp.	Promoter	Gene silencing	Flowering repressors TOE2/SMZ	(116,123)
	miRNA156	Cold temp.	Repressor	Gene silencing	*FT*	(117,118)
	FT	Cold temp.	Repressor	Transcriptional activation	Flowering genes	(88,110,111)

regulate flowering (116–120). The miR156 targets the mRNA of *SQUAMOSA PROMOTER BINDING PROTEIN-LIKE 3* (*SPL3*). microRNA-resistant versions of *SPL3* lead to an increase in *FT* expression in leaves and temperature-independent flowering. A similar phenotype was obtained by reducing miR156 activity via target mimicry, confirming the important role of miR156 in temperature signaling (118). SPL3 activates *FT* and *SEP3* (119), which participate in a positive feedback loop to induce flowering (121,122). The miR172 shows an opposite effect to miR156, its overexpression produces a temperature independent early flowering, decreasing temperature sensitivity; among its targets, flowering repressors *TARGET OF EAT 2* (*TOE2*) and *SCHLAFMUTZE* (*SMZ*) are downregulated at 23°C in comparison with 16°C (120,123). The responsiveness of microRNAs to temperature suggests that the sensing mechanism acts upstream these microRNAs. SHORT VEGETATIVE PHASE (SVP), a MADS box transcription factor, negatively regulates miR172 (116).

SVP is one of the main regulators within the thermosensory pathway. *svp* mutants are early flowering and insensitive to lower temperatures and SVP directly binds to the promoter of *FT* (106). Furthermore, SVP forms complexes with other MADS box transcription factors, including FLOWERING LOCUS C (FLC) and FLOWERING LOCUS M (FLM), and also activates *SOC1* (124). Breakthrough discoveries occurred recently, when it was determined how SVP acts together with FLM in the thermosensory pathway (117,125,126). *FLM* and *SVP* genes encode repressors of flowering which were known to interact genetically in the same pathway (127). Natural variation experiments uncovered *FLM* as a modulator of the sensitivity of flowering to temperature (70). More recently it was shown that the ratio between

two splice variants of *FLM* transcripts, *FLM*-β and *FLM*-δ depend on temperature, resulting in predominant SVP–FLM-β complexes at low temperatures that repress flowering, and predominant SVP–FLM-δ complexes at higher temperatures, which act as dominant-negative factors, activating flowering (126). Furthermore, it was also shown that SVP is degraded by the proteasome at higher temperatures, relieving flowering from repression (125,128).

How is then the thermosensory pathway connected to light signaling? LATE ELONGATED HYPOCOTYL (LHY) and CIRCADIAN CLOCK–ASSOCIATED1 (CCA1) are two MYB transcription factors that work as part of the circadian clock, specifically in the morning loop (129). *cca1 lhy* double mutants flower earlier than WT plants in both LD and SD. However, under continuous light *cca1 lhy* double mutants flower late and this specific late flowering phenotype is suppressed by mutations in *SVP* or in *ELF3* (130,131). It was determined that in these conditions, SVP protein levels accumulate and require ELF3. SVP levels are high in ELF3 overexpressors and the peak of SVP abundance is delayed in *elf3* mutants (131). Given the roles of ELF3 in light signaling and clock function and its role in the flowering response to ambient temperature (84), ELF3 emerges as a point of connection between light signaling and temperature signaling in the regulation of flowering. Both *elf3* and *svp* mutants have similar temperature-hyposensitive flowering phenotypes (84,106).

ELF3 and PIF4 as general convergence points

As presented above, ELF3 represents a connection point between light and temperature signaling during early seedling development and shade avoidance and also during flowering induction. As mentioned in previous sections, ELF3 binds to PIF4 and prevents its transcriptional activity (89). On the other hand, *pif4* mutants display opposite phenotypes to *elf3* mutants, they are late flowering in SD at higher temperatures (27°C compared to 22°C) (18). PIF4 promotes flowering under higher temperatures by directly binding to the *FT* promoter. Although the protein levels of PIF4 may not increase with temperature in all conditions (18,71,132), the binding of PIF4 to the *FT* promoter is dependent on temperature and low temperature suppress the early flowering of PIF4 overexpressors (18). Overexpression of PIF4 inhibits the late flowering of ELF3 overexpressors at 22°C (89). It would be interesting to know, using transgenic lines with different levels of overexpression and measuring flowering time at different temperatures if the ELF3 and PIF4 interaction varies with temperature and extends the model proposed by Salome-Prat and col (89) to the promotion of flowering (133).

CONCLUSION

Despite efforts to identify dedicated plant thermosensors, their nature has been elusive. Nevertheless, significant advances were made in understanding how light and temperature signaling interact, and some of the common partners identified (Table 1). In the near future, it will be interesting to know how they relate to each other. Are all of them part of the same pathway? Are different pathways working in different conditions of, for instance, photoperiod and temperature? How are the MADS box transcription factors FLM, SVP and FLC related to HY5, PIF4, ELF3 and TFL1? Within the range 16–28°C that has been extensively used, are the mechanisms underlying responses to lower temperatures similar to those underlying responses to higher temperatures? Genetic dissection of the action of these components will be extremely useful to isolate and study single temperature-responsive pathways that interact with light signaling.

On the other hand, it is difficult to explain this whole set of results if we think that only a small set of thermosensors are involved. It is more likely that a diverse set of temperature-dependent biochemical processes are in place to detect subtle changes in temperature, including membrane fluidity, transcription, mRNA processing and splicing, chromatin dynamics, DNA replication, circadian clock function, microRNA biogenesis and activity, etc. Being these processes general, one of the challenges is to determine which changes are compensatory, like the temperature compensation mechanisms of the circadian clock, and which ones are truly part of a temperature perception and signaling mechanism.

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