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Review article

Regulation of PIF4-mediated thermosensory growth

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ABSTRACT

Ambient temperature has profound impacts on almost every aspect of plant growth and development, including seed germination, stem and petiole elongation, leaf movement, stomata development, flowering, and pathogen defense. Although the signal transduction pathways underlying plant responses to extreme cold and heat temperatures have been well studied, our understanding, at the molecular level, of how plants adjust phenotypic plasticity in response to nonstressful ambient temperature is still rudimentary. This review summarizes studies related to PHYTOCHROME-INTERACTING FACTOR 4 (PIF4), the cardinal regulator of thermoresponsive growth in the model dicotyledonous plant *Arabidopsis thaliana*, emphasizing recent progress in the light-quality- and photoperiod-dependent regulation of PIF4-mediated thermomorphogenesis.

1. Introduction

Temperature fluctuation exceeding the optimal range for yield formation due to global climate change is a major factor leading to yield variability and thus a great threat to food security in many regions of the world [1–3]. For many crop species, the range of about 10 − 30 °C is considered non-stressful and yet temperature variations within this range have dramatic influences on many aspects of plant growth and development [4]. Moderate increases in ambient temperature promote thermomorphogenetic changes including exaggerated stem elongation and early flowering [5], which could potentially increase the risk of lodging and affect the biomass/seed yield [6,7].

The thermo-induced architectural changes are believed to be the consequences of massive transcriptomic reprogramming triggered by temperature elevations, as indicated by early studies in the model dicotyledonous plant *Arabidopsis thaliana* [8–11]. This conclusion was corroborated by the discovery that ambient temperature modulates transcript abundance mainly by regulating mRNA synthesis rather than the decay of temperature-responsive transcripts [12]. A major effect of this transcriptomic reprogramming is to stimulate the accumulation of the growth-promoting phytohormone auxin by enhancing the expression of genes involved in auxin syntheses, such as *YUCCA8* (*YUC8*), and auxin signaling, such as *INDOLE-3-ACETIC ACID INDUCIBLE 19* & *29* (*IAA19* & *IAA29*) [13–16].

A central transcriptional regulator of plant thermal response is PHYTOCHROME-INTERACTING FACTOR 4 (PIF4), which induces the expression of the above-mentioned auxin-related genes in response to moderately increased temperature [13–16]. PIF4 belongs to the Subfamily 15 of the basic Helix-Loop-Helix (bHLH) superfamily. The eight-member PIF transcription factor family was first identified as key players in transducing light signals perceived by the red (R)/far-red (FR) photoreceptors phytochromes (phys) [17]. Extensive studies during the past two decades demonstrate that PIFs are integrators of light and various environmental and developmental signals [17–19]. Although PIF proteins show apparent functional redundancy within the family in multiple morphogenetic responses, such as seed germination, seedling de-etiolation, shade avoidance, and photoperiodic growth [18,19], thermomorphogenesis is mediated primarily through PIF4 [10,20–22].

As an early ambient temperature signaling component, PIF4 is regulated at multiple levels, including transcription, post-translational modification, DNA-binding ability, transcriptional activity, and protein stability (Fig. 1). Furthermore, recent reports revealed distinct temperature-signaling mechanisms under different light conditions and photoperiod regimes, indicating more complex regulation of PIF4-mediated thermal responses by light quality, day length, and the circadian clock [20,23,24]. In this review, I first overview the effects of light quality and photoperiod on PIF4-mediated thermoresponsive growth and then discuss more generic thermal regulation of PIF4 at different levels in *Arabidopsis thaliana*. For detailed information about other ambient temperature responses in Arabidopsis and other species, I refer the readers to other excellent reviews published recently [25–27].

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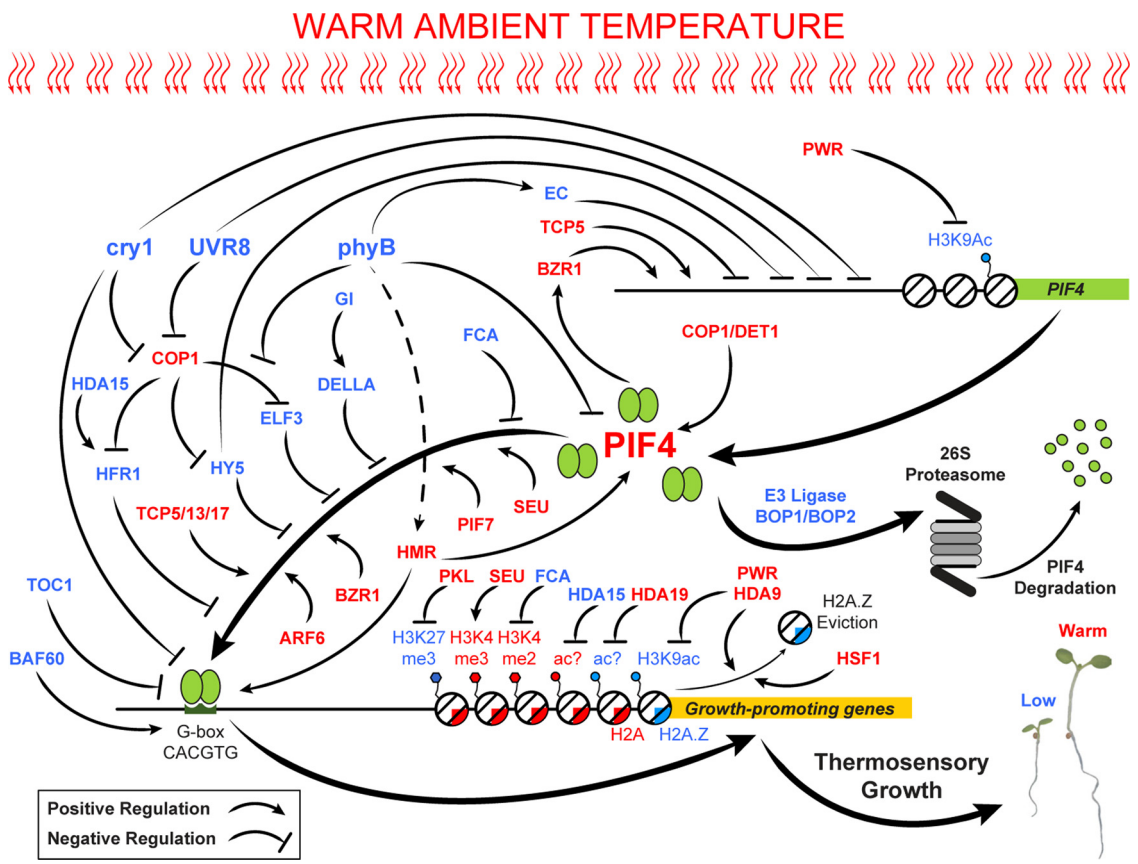

Fig. 1. Regulation of PIF4-mediated thermosensory growth. As a central regulator of thermomorphogenesis, PIF4 is targeted by various factors and modulated at different levels. PIF4 proteins are depicted as homodimers (two green ovals). Red- and blue-colored factors specify positive and negative regulators of PIF4-mediated thermal responses, respectively. The dashed line suggests that how phyB regulates HMR in ambient temperature response is not clear. The question marks by ac (acetylation) above illustrations of nucleosomes mean undetermined types of histone acetylation (H3K14ac, H3K9ac, and maybe others) [61]. PIF4, PHYTOCHROME-INTERACTING FACTOR 4; phyB, phytochrome B; cry1, cryptochrome 1; UVR8, UV RESISTANCE LOCUS 8; EC, EVENING COMPLEX; COP1, CONSTITUTIVE PHOTOMORPHOGENIC 1; DET1, DE-ETIOLATED 1; HFR1, LONG HYPOCOTYL IN FAR-RED 1; TCP5/13/17, TEOSINTE BRANCHED 1/CYCLOIDEA/PCF 5/13/17; HY5, ELONGATED HYPOCOTYL 5; ELF3, EARLY FLOWERING 3; GI, GIGANTEA; FCA, FLOWERING CONTROL LOCUS A; TOC1, TIMING OF CAB EXPRESSION 1; BAF60, BRG1/BRM-ASSOCIATED FACTOR 60; ARF6, AUXIN-RESPONSE FACTOR 6; BZR1, BRASSINAZOLE-RESISTANT 1; HMR, HEMERA; PIF7, PHYTOCHROME-INTERACTING FACTOR 7; SEU, SEUSS; PKL, PICKLE; HDA9/15/19, HISTONE DEACETYLASE 9/15/19; PWR, POWERDRESS; HSF1, HEAT SHOCK FACTOR 1; BOP1/2, BLADE-ON-PETIOLE 1/2; ac, acetylation; me, methylation (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

2. PIF4 is targeted by multiple photoreceptors during thermal response

Plant morphology is shaped by different families of photoreceptors that sense distinct light spectra [28]. In Arabidopsis, the R/FR photoreceptor phyB, the blue/ultraviolet-A (UV-A) light receptor cryptochrome 1 (cry1), and the UV-B receptor UV RESISTANCE LOCUS 8 (UVR8) have been reported to regulate either PIF4 activity or PIF4 abundance or both in warm ambient temperature [15,29–32].

phyB was proposed to be an ambient temperature sensor based on its biochemical property and chromatin-association nature [29,30]. phyB exists as two photo-interconvertible isoforms in plant cells: an R-absorbing inactive Pr form and an FR-absorbing active Pfr form, and thus the percentage of the Pfr form of phyB is regulated by the R/FR ratio [33]. Besides, the Pfr form of phyB also spontaneously reverts back to Pr in a light-independent but temperature-dependent reaction called dark reversion or thermal reversion [34]. Consequently, temperature elevation reduces the abundance of biologically active phyB by increasing the rates of thermal reversion [30]. Besides, phyB is associated with the promoters of temperature-responsive PIF target genes in a temperature-dependent way [29]. Given the fact that phyB physically interacts with PIF4 [35], it may modulate thermal responses by regulating PIF4 activity on these shared targets. phyB also has a dramatic effect on *PIF4* transcript levels in a warm environment [29] but this regulation is photoperiod-dependent and will be elaborated in the next section.

Blue light has a strong inhibitory effect on thermoresponsive hypocotyl elongation via cry1 [15,31]. Although cry1 also represses the transcription of *PIF4*, it doesn't affect PIF4 protein abundance at warm ambient temperature [15]. Instead, cry1 inhibits PIF4 activity on thermoresponsive genes involved in auxin biosynthesis and signaling through two mechanisms [15]. First, like phyB, cry1 directly interacts with PIF4 to restrict its transcriptional activity. Secondly, cry1 stabilizes LONG HYPOCOTYL IN FAR-RED 1 (HFR1), an atypical PIF-like bHLH transcription factor, by inhibiting the E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1). In turn, HFR1 prevents PIF4 from binding to target genes by forming non-DNA-binding heterodimers with PIF4 [31,36]. It is worth mentioning that the inhibitory role of cry1 in thermomorphogenesis is determined by light intensity as well as seedling's development stage [20].

Similar to blue light, low dose UV-B light also attenuates thermomorphogenesis by regulating PIF4 [32]. The UV-B receptor UVR8 does not interact with PIF4 [37], but it regulates both *PIF4* transcription and PIF4 activity in a COP1-dependent manner [32]. On one hand, UVR8 controls PIF4 abundance by inhibiting *PIF4* transcript levels in both cooler and warmer environments. Although it may also promote PIF4

protein degradation, as seen at a lower temperature, this effect is attenuated when the temperature increases. On the other hand, UVR8 inhibits COP1-mediated HFR1 degradation by sequestering COP1 and highly accumulated HFR1 heterodimerizes with PIF4 to restrict PIF4 transcriptional activity on thermoresponsive genes.

Furthermore, ELONGATED HYPOCOTYL 5 (HY5), a bZIP transcription factor that is also targeted by COP1 for degradation, regulates thermomorphogenesis by controlling both *PIF4* transcription and PIF4 activity [23,38,39]. Some conclusions mentioned above were drawn from studies carried out in monochromatic lights. How HY5 is regulated by each photoreceptor at warm temperatures and how crosstalks among different light signaling pathways in the natural white light condition affect PIF4-mediated thermal response are still obscure.

3. Photoperiodic control of PIF4-mediated thermoresponsive growth

Thermomorphogenesis occurs in all photoperiodic regimes but at distinct times [23]. The contribution of PIF4 to thermomorphogenesis has been observed in all photoperiodic regimes [23]. Consistent with the photoperiodic control of the thermoresponsive hypocotyl growth, *PIF4* expression was induced by warm daytime temperatures in the long-day (LD) condition but warm nighttime temperatures under the short-day (SD) regime [23,24]. These data indicate a strong causal relationship between the thermo-induced *PIF4* expression and the photoperiod-dependent thermomorphogenetic hypocotyl elongation.

In the SD condition, the induction of *PIF4* transcripts is gated by the evening complex (EC) of the circadian clock [40]. The EC, composed of the products of three circadian clock genes, *EARLY FLOWERING 3, 4* (*ELF3* and *ELF4*), and the MYB transcription factor *LUX ARRYTHMO* (*LUX*), binds to the promoter of *PIF4* and represses its expression in the early evening [40,41]. On the other hand, ELF3 also directly interacts with PIF4 and prevents it from activating growth-related target genes [42]. As the levels of *ELF3*, *ELF4*, and *LUX* decrease through the night, *PIF4* transcription and PIF4 transcriptional activity are derepressed and the accumulation of functionally active PIF4 promotes hypocotyl growth at the end of the night [40]. Interestingly, phyB physically interacts with ELF3 both in vitro and in vivo [43,44], which helps stabilize ELF3 and assists it in blocking PIF4 transcriptional activity [42]. phyB also associates with the EC on the *PIF4* promoter to maintain the low *PIF4* level in the early night under SD conditions [29,41]. In response to temperature elevation, the accelerated dark-reversion rate of phyB reduces the steady-state level of the active Pfr form, leading to decreased activity of the EC in the early night [29,30,41]. Consequently, the *PIF4* level accumulates more rapidly during the night and promotes hypocotyl growth throughout the night [23,41].

Despite the fact that ELF3 regulates both *PIF4* expression and PIF4 transcriptional activity [41,42], its thermoresponsive function is restricted to SD conditions [23]. When grown under neutral day or LD conditions, the *elf3* loss-of-function mutant sustained warm temperature sensitivity [23]. Given that the timing of *PIF4* induction by the elevated temperatures in LD is distinct from that in SD conditions [23], an LD-specific mechanism must exist to regulate PIF4-mediated thermomorphogenesis. Because phyB's dark-reversion rate depends on ambient temperature but not the light environment, phyB might be able to sense the temperature during daytime in the LD conditions. This hypothesis is supported by a recent study showing that the hypocotyl growth of the *phyB* null mutant is insensitive to both continuous R light (Rc) and LD conditions [20].

Although the circadian gating mechanism of thermo-induced *PIF4* accumulation under the LD condition is not known, a recent study demonstrated that GIGANTEA (GI), a core clock protein, attenuates thermomorphogenesis under the LD regime by inhibiting PIF4 function [45]. Under LDs, GI thermostabilizes the DELLA protein, REPRESSOR OF *ga1-3* (RGA), which in turn inhibits PIF4 from binding to auxin-related targets (e.g. *YUC8*) and thus restricts hypocotyl growth. Interestingly, GI mainly restricts PIF4 activity at night but plays a minor role during daytime in LDs. The thermomorphogenetic function of GI is more prominent in LDs than SDs because its abundance remains constantly low in the SD conditions. Without the protection of sufficient GI proteins, RGA is readily degraded in SDs and consequently, thermoactivated PIF4 promotes hypocotyl growth by inducing *YUC8* expression.

To date, at least three factors have been found to be responsible for the daytime temperature sensing by regulating PIF4. The first factor is HEMERA (HMR), which was originally identified as a key component in the phyB-mediated light signaling pathway [46–48]. HMR plays a more prominent role in daytime thermosensing under Rc and LD conditions as a transcriptional coactivator of PIF4 [20]. HMR physically interacts with PIF4 and induces the expression of auxin-related, growth-promoting PIF4 target genes through its C-terminal transcriptional activation domain (TAD) [20,46]. Interestingly, HMR does not regulate *PIF4* transcription. Instead, it facilitates thermoresponsive PIF4 accumulation and its TAD is indispensable for PIF4 protein stability at a warm temperature [20].

The other two daytime thermosensory regulators downstream of phyB are DE-ETIOLATED 1 (DET1) and COP1. Unlike HMR, DET1 and COP1 regulate PIF4 at both transcriptional and post-translational levels. On one hand, COP1 and DET1 suppress the accumulation of HY5, which not only inhibits the transcription of *PIF4* but also competes with PIF4 on binding to growth-promoting PIF4 targets [23,38,39]. However, this layer of regulation seems to be independent of the photoperiod because DET1 and COP1 promote HY5 degradation and *PIF4* transcription in both LD and SD conditions [38,39]. This is consistent with the observation that DET1 and COP1 are required for thermomorphogenesis in both LD and SD regimes [23,38]. As mentioned in the previous section, COP1 also suppresses HFR1 accumulation and thus prevents it from heterodimerizing with PIF4 and inhibiting PIF4 transcriptional activity on thermoresponsive genes [15,32,36,49–51]. However, the photoperiodic control of HFR1 by COP1 is not clear. It is worth mentioning that ELF3 is also a substrate of COP1 [42,52]. Therefore, COP1 may also regulate *PIF4* transcription and PIF4 transcriptional activity by mediating the ubiquitination and degradation of ELF3 under the SD condition. On the other hand, DET1 and COP1 promote thermosensory growth by stabilizing PIF4 protein [38]. When *PIF4* expression was driven by the strong constitutive cauliflower mosaic virus promoter (CaMV 35S) in the *cop1* or *det1* mutant background, the *PIF4* transcript level was induced drastically but PIF4 protein failed to accumulate [38].

The thermomorphogenetic studies during the past decade showed a great deal of variability in experimental conditions, including light quality (white light vs. monochromatic light), light intensity, photoperiodic regime, growth medium (with vs. without sugar), seedling developmental stage, as well as duration and amplitude of the warm temperature treatment. Inevitably, these variations lead to some data inconsistency and occasionally even contradictory conclusions. Therefore, we are still far from getting a whole picture of how ambient temperature regulates PIF4 in natural light and temperature conditions. In the following sections, I will summarize recent models on thermal regulation of PIF4 in a generic way, mentioning but not emphasizing the limitation of each model (Fig. 1).

4. Thermal regulation of *PIF4* transcription

As discussed above, the thermo-induced expression of *PIF4* is gated by the circadian clock and shows distinct patterns in different photoperiodic regimes [16,23]. Many negative regulators of *PIF4* transcription, including all three types of photoreceptors – phyB, cry1, and UVR8, have been described. Warm temperatures promote the thermal reversion of phyB, weakening its restriction on *PIF4* transcription in the early night during the SD condition [29,40,41]. UVR8 and cry1 may inhibit *PIF4* accumulation by antagonizing COP1-mediated HY5

degradation [15,32,39,53]. However, how this is achieved in different photoperiodic regimes is largely unknown.

On the other hand, very few positive regulators of *PIF4* transcription were identified. It is still not clear how *PIF4* expression is activated by elevated temperatures under different photoperiodic regimes. The BR-regulated transcription factor BRASSINAZOLE-RESISTANT 1 (BZR1) may contribute to this process. At elevated temperatures, BZR1 is preferentially localized to the nucleus, where it binds to the *PIF4* promoter and activates *PIF4* transcription [54]. As PIF4-mediated thermomorphogenesis is brassinosteroid dependent, the BZR1 seems to enhance or accelerate the temperature response by acting in a feedforward loop on *PIF4* expression [54]. It would be interesting to study whether the thermo-induced nuclear translocation of BZR1 or its accessibility to the *PIF4* promoter is gated by the circadian clock. Three TEOSINTE BRANCHED 1/CYCLOIDEA/PCF (TCP) transcription factors, TCP5/13/17, may also contribute to promoting thermoresponsive *PIF4* accumulation [55]. The expression of *PIF4* at an elevated temperature (28 °C) was greatly downregulated in the *tcp5tcp13tcp17* triple mutant. Warm temperatures promote TCP5 accumulation at both transcriptional and post-transcriptional levels. Moreover, TCP5 directly binds to the *PIF4* promoter to activate its transcription.

The thermo-induced expression of *PIF4* may also require histone deacetylation [56]. Elevated temperatures greatly reduce the H3K9 acetylation level at the +1 nucleosome of *PIF4*, which correlates with the enhanced expression of *PIF4* [56]. It was suggested that POWERDRESS (PWR), an interacting partner of HISTONE DEACETYLASE 9 (HDA9), is required for the H3K9 deacetylation and induction of *PIF4* upon temperature increase [56]. However, a recent study showed that HDA9 may not be involved in the regulation of *PIF4* transcription at a warm temperature [57]. Hence, the epigenetic control of thermoresponsive *PIF4* expression is still ambiguous.

5. Regulation of PIF4 activity at elevated temperatures

The binding of PIF4 to auxin-related, thermoresponsive genes (e.g. *YUC8*, *IAA19*, and *IAA29*) and activation of their expression is an early and key step in the thermo-induced growth response. This seemingly straightforward process is regulated by multiple mechanisms, including control of chromatin accessibility, regulation of PIF4 DNA-binding ability, and modulation of PIF4 transactivation activity.

5.1. Chromatin accessibility of thermoresponsive PIF4 target genes

By far, no study has shown that PIF4 functions as a pioneer transcription factor that can directly bind to condensed chromatin and recruit other transcription factors and/or histone modifiers. Instead, accumulating evidence indicates that warm ambient temperatures regulate the accessibility of the genomic regions of PIF4 targets through epigenetic factors such as histone variants, histone modifiers, and chromatin remodelers.

The histone variant H2A.Z is by far the most studied factor that affects the thermodynamics of chromatin structure in plants. The incorporation and deposition of H2A.Z at PIF4-targeted loci significantly affects the thermoresponsive expression of both flowering genes such as *FLOWERING LOCUS T* (*FT*) and growth-promoting genes including those involved in auxin biosynthesis and signaling (e.g. *YUC8*, *IAA19*, and *IAA29*) [11,22,56,58–60]. Although the effects of H2A.Z on thermomorphogenesis show species- and organ-specificity [58,59], it is well demonstrated that H2A.Z depletion at the thermoresponsive genes allows binding and transcriptional activation by PIF4 upon temperature increases in Arabidopsis [11,22,57]. H2A.Z nucleosomes seem not to be intrinsically temperature-responsive [60]. The net eviction of H2A.Z from nucleosomes associated with temperature-responsive genes is regulated by at least two factors. The first one belongs to the HSFA1 clade of Arabidopsis HEAT SHOCK FACTORS (HSFs) [60]. Most HSFs are essential for heat stress responses, while the HSFA1 clade positively regulates the warm ambient temperature transcriptome by rapidly and dynamically evicting H2A.Z nucleosomes at numerous target genes. How many of these thermoresponsive genes are shared with those regulated by PIF4 remains to be studied. The second factor is a chromatin-modifying enzyme called HDA9, which is stabilized by warm temperatures and promotes H2A.Z deposition at the auxin biosynthetic gene *YUC8* by enhancing histone deacetylation at the *YUC8* locus [57]. Consequently, activation of *YUC8* by PIF4 promotes the accumulation of auxin and thermomorphogenesis. The function of HDA9 requires the SANT domain-containing protein PWR, which also plays a crucial role in facilitating thermomorphogenesis [56,57]. Besides HDA9, two other histone deacetylases HDA19 and HDA15 also contribute to the thermomorphogenetic control [61]. Interestingly, while HDA9 and HDA19 promote thermo-induced hypocotyl growth, HDA15 functions as a negative regulator of this process. HDA15 physically interacts with HFR1, the latter of which is known to inhibit PIF4 activity by forming heterodimers [31]. Thus, it was hypothesized that HDA15 could stabilize HFR1 or HFR1 could recruit HDA15 to PIF4 target genes to epigenetically repress thermo-induced hypocotyl elongation [61]. However, how three histone deacetylases playing opposite roles in thermomorphogenesis coordinate to regulate thermoresponsive genes is still unknown.

Besides histone deacetylation, histone methylation may also play an important role in regulating the expression of temperature-sensitive genes. Warm ambient temperatures moderately increase H3K4 dimethylation (H3K4me2) on the promoter of *YUC8*, but this promotion is restricted by an RNA-binding protein FLOWERING CONTROL LOCUS A (FCA) [62]. The transcription regulator SEU, however, positively regulates thermomorphogenesis by inducing H3K4me3 in the chromatin regions of *YUC8* and *IAA19* [63]. Elevated temperatures also decrease the level of H3K27me3 on *IAA19* and *IAA29*, which relies on the function of a chromatin remodeling factor called PICKLE (PKL) [64]. Although the levels of these histone methylation changes correlate with the expression of temperature-responsive PIF4 target genes, the writers and erasers for these modifications and their thermomorphogenetic roles are largely unknown. It has been shown that several H3K27me3 demethylases in the JUMONJI family play key roles in regulating temperature- and photoperiod-regulated flowering and transgenerational memory of heat stress responses [65–68]. Therefore, it would be interesting to study whether these histone demethylases are directly involved in thermoresponsive growth by regulating PIF4 target genes.

In addition to the deposition of histone variants and changes in histone modifications, chromatin-based transcriptional regulation also relies on ATP-dependent chromatin remodeling complexes (CRCs). The above-mentioned PKL belongs to the ATPase CHD3 subfamily and usually promotes H3K27me3 [69,70]. However, in the case of thermomorphogenesis PKL inhibits the level of H3K27me3 on temperature-sensitive PIF4 target genes [64]. Another potential CRC component for ambient temperature responses is the BRG1/BRM-ASSOCIATED FACTOR 60 (BAF60) subunit of the SWI/SNF subfamily [71]. BAF60 preferentially targets G-box motifs and acts antagonistically to PIF4 on the expression of hypocotyl elongation regulatory genes. Some of these genes, such as *IAA19*, are well-documented thermo-inducible PIF4 targets. Moreover, knocking down *BAF60* by RNAi leads to highly elongated hypocotyls at 28 °C. All these data indicate that the SWI/SNF CRC may regulate the thermo-induced hypocotyl elongation by controlling the chromatin accessibility of PIF4-targeted growth-promoting genes at warm temperatures.

5.2. Control of PIF4 DNA-binding ability in warm temperatures

A number of transcription regulators have either antagonistic or synergistic effects on PIF4 activity by controlling its DNA-binding affinity. Positive regulators, such as AUXIN-RESPONSE FACTOR 6 (ARF6), BZR1, SEUSS (SEU), physically interact with PIF4 to promote

its association with the promoters of growth-promoting, thermo-responsive genes [63,72]. Interestingly, the DNA-binding ability of SEU may not rely on PIF4, suggesting sequential recruitments of SEU and PIF4 to their common targets in response to temperature changes [63]. Recently, PIF7 was reported to be another PIF member that regulates thermoresponsive growth in Arabidopsis [73]. The elevated temperatures rapidly enhance the accumulation of PIF7, which promotes the expression of auxin biosynthesis and signaling genes by binding to their promoters. These auxin-related genes largely overlap with those PIF4-targeted thermoresponsive genes. Because PIF7 physically interacts with PIF4, it is highly possible that these two PIF factors enhance each other's DNA-binding affinity by forming heterodimers. Similarly, TCP5/13/17 also physically interact with PIF4 and promote the expression of thermoresponsive PIF4 targets by directly binding to their promoters [55,74]. Although not shown in these two publications, it is possible that TCP5/13/17 might enhance PIF4 activity by facilitating its association with the common target genes. Negative regulators restrict the DNA-binding ability of PIF4 either by directly binding PIF4 or by competing with PIF4 on the same target promoters or both. Some representative negative regulators are HFR1, HY5, ELF3, FCA, and the key repressors of GA-responsive growth DELLAs [36,38,42,62,75,76]. It is noteworthy that besides its function in promoting PIF phosphorylation and degradation, phyB also inhibits binding of PIF1 and PIF3 to their target promoters through its N-terminal photosensory module [77]. Therefore, it is also possible that phyB restricts the association of PIF4 with auxin-related genes and warmer temperature alleviates this restriction by enhancing the thermal reversion rate of phyB.

5.3. Control of PIF4 transactivation activity in warm temperatures

Both positive and negative regulators of PIF4 transactivation activity have been identified. In the neutral day condition, the activity of PIF4 is gated by the circadian clock component TIMING OF CAB EXPRESSION 1 (TOC1) [16]. Expression of *TOC1* is maintained at a low level during the early day and accumulates towards dusk, when it physically interacts with PIF4 to restrict the thermoresponsive expression of auxin-related genes, such as *YUC8*, *IAA19*, and *IAA29* in the evening. The binding of TOC1 to PIF4 suppresses PIF4 transcriptional activity without interfering with its DNA binding affinity. Interestingly, EC recruits HDA9 to repress *TOC1* expression during the night by promoting H3 deacetylation at the *TOC1* locus [78]. Given that EC and HDA9 play opposite roles on PIF4-mediated thermal response [41,57,61], the thermodynamic regulation of PIF4 activity by EC, HDA9, and TOC1 under diurnal conditions needs to be re-investigated.

HMR, on the other hand, functions as a transcription co-activator of PIF4 through its C-terminal TAD [20,46]. However, it is not clear whether the transactivation activity of PIF4 is still needed for the activation of auxin-related thermoresponsive genes. HMR interacts with the Active phyB Binding (APB) domain of PIF4, a region overlapping with one of the two identified TADs of PIF4 [20,46,79]. Hence, the effect of the HMR-PIF4 interaction on the transactivation activity of PIF4 needs to be investigated.

PIF4 is known to be regulated post-translationally by phosphorylation modification. While light-triggered PIF4 phosphorylation destabilizes PIF4 protein (discussed below) [80,81], warm temperature leads to the accumulation of hyper-phosphorylated PIF4 forms [31]. Phosphorylation of transcription factors can potentially regulate not only their stability but also transactivation activity, and phosphorylation at different sites may lead to distinct regulation [82]. It seems that PIF4 can be phosphorylated by more than one kinase [80]. Given the correlation between accumulation of hyper-phosphorylated PIF4 and strong activation of PIF4-targeted growth-promoting genes at a warm temperature, it is possible that the thermo-induced PIF4 phosphorylation may enhance its transcriptional activity. Therefore, it would be interesting to see whether TOC1 and HMR regulate the phosphorylation modification of PIF4 in response to temperature elevations.

6. PIF4 stability under warm temperatures

Both HMR and DET1/COP1 are required for thermosensory PIF4 accumulation, but their functions may be distinct from each other. HMR's TAD is mainly required for promoting PIF4 accumulation during temperature elevation and plays a minor role at a lower ambient temperature [20]. In contrast, DET1/COP1-mediated PIF4 stability control is not restricted to warm temperature. It seems that the overall PIF4 protein accumulation at any given growth temperature requires DET1 and COP1 [38,83]. PIF4 degradation is mediated by BLADE-ON-PETIOLE (BOP) 1 and 2, which are substrate recognition subunits in CULLIN3-based E3 ubiquitin ligases [84]. Similar to DET/COP1, BOPs regulate PIF4 protein abundance at both cooler (22 °C) and warmer (28 °C) temperatures. Hence, DET1/COP1 and BOP1/2 might serve as more general regulators of PIF4 accumulation but play antagonistic roles. Nevertheless, this does not rule out the possibility that HMR protects PIF4 from being degraded by BOP-mediated ubiquitin-proteasome pathway through physical interactions at warm temperatures.

As mentioned in the previous section, PIF4 protein stability is regulated by phosphorylation in the light [80,81]. Although PIF4 phosphorylation is not required for BOP2-mediated PIF4 ubiquitination in vitro [84], it doesn't eliminate the possibility that phosphorylation of PIF4 could enhance its interaction with BOP and thus subsequent ubiquitination and degradation. Therefore, it would be interesting to test which form of PIF4 (phosphorylated, dephosphorylated, or both) is protected by HMR and COP1/DET1, especially when facing temperature increases.

7. Future perspectives

The bHLH transcription factor PIF4 plays a central role in thermoresponsive growth. More and more ambient temperature signaling components are discovered to regulate thermosensing and response in a PIF4-dependent way. Nonetheless, upon temperature elevations how these factors coordinate systematically in regulating PIF4-mediated thermal response remains to be elucidated. Recent studies on light-quality- and day-length-dependent control of thermomorphogenesis further complicate the network. We are still at a very early stage in understanding the interactions and crosstalks among these PIF4-oriented regulators under different light conditions and photoperiod regimes. Furthermore, epidermal PIF4 is both sufficient and required for thermoresponsive growth, although *PIF4* is expressed in all aerial tissues [85]. Therefore, we need to re-evaluate the regulation of PIF4 by these thermal signaling components in a tissue-specific way. Future thermomorphogenetic studies incorporating ecophysiological diversity and tissue specificity in Arabidopsis and crop plants will greatly assist us in developing climate-smart crops with high seed or biomass yield.

Declaration of Competing Interest

None.

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