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Molecular and genetic control of plant thermomorphogenesis

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Temperature is a major factor governing the distribution and seasonal behaviour of plants. Being sessile, plants are highly responsive to small differences in temperature and adjust their growth and development accordingly. The suite of morphological and architectural changes induced by high ambient temperatures, below the heat-stress range, is collectively called thermomorphogenesis. Understanding the molecular genetic circuitries underlying thermomorphogenesis is particularly relevant in the context of climate change, as this knowledge will be key to rational breeding for thermo-tolerant crop varieties. Until recently, the fundamental mechanisms of temperature perception and signalling remained unknown. Our understanding of temperature signalling is now progressing, mainly by exploiting the model plant *Arabidopsis thaliana*. The transcription factor PHYTOCHROME INTERACTING FACTOR 4 (PIF4) has emerged as a critical player in regulating phytohormone levels and their activity. To control thermomorphogenesis, multiple regulatory circuits are in place to modulate PIF4 levels, activity and downstream mechanisms. Thermomorphogenesis is integrally governed by various light signalling pathways, the circadian clock, epigenetic mechanisms and chromatin-level regulation. In this Review, we summarize recent progress in the field and discuss how the emerging knowledge in *Arabidopsis* may be transferred to relevant crop systems.

The year 2015 is on track to surpass 2014 as the warmest year ever recorded since systematic temperature measurements began more than a century ago[1]. In fact, the 10 warmest years on record all occurred after 1998. The fifth report of the Intergovernmental Panel on Climate Change[2] projects an increase of 0.8–4.8 °C in global mean surface temperature within the twenty-first century. Such figures are alarming as it is expected that this will strongly affect plant distribution and survival, and therefore threaten biodiversity[3–11]. Some studies already indicate that plant species unable to adjust flowering time in response to temperature are disappearing from certain environments[5], and species tend to shift to higher altitudes and latitudes[12].

Likewise, crop productivity will probably suffer greatly from global warming, while food production is required to increase significantly to sustain a growing and more demanding world population[9,13–15]. A meta-analysis summarizing more than 1,700 studies on the effects of climate change and adaptations on crop yields revealed consensus that in the second half of this century, climate warming is likely to have a negative effect on yields of important staple crops[13].

Breeding for crop-level adaptations to cope with high temperatures could potentially reverse this negative trend[9,13–15]. In several plant species, mechanisms have evolved to adapt growth and morphology to stimulate mitigation of warmth through enhanced evaporative cooling, increased convection and direct avoidance of heat flux from the Sun[16–20]. If understood, the underlying molecular processes of these so-called thermomorphogenesis responses could be attractive breeding targets for improving crops to withstand climate warming.

Although abundant literature is available on how plants tolerate extreme heat stress (reviewed in refs 9,21), we are only beginning to understand the molecular mechanisms underlying thermomorphogenesis in response to moderately increased temperatures. A key breakthrough was the identification of the bHLH (basic helix–loop–helix) transcription factor PHYTOCHROME INTERACTING FACTOR 4 (PIF4) as a central regulator of ambient temperature signalling in *Arabidopsis*[22]. Recent findings have implicated important roles for light signalling pathways, the circadian clock[23–28], auxin[22,29–31] and other phytohormones[31–34] in PIF4-mediated temperature-induced growth. Furthermore, epigenetic mechanisms appear at the nexus of induction[35] and attenuation[36] of growth acclimation in response to high ambient temperatures.

Here we discuss and integrate recent findings on the molecular networks driving thermomorphogenic adaptations. We will highlight missing links and suggest how the knowledge on *Arabidopsis* could be transferred to crops. In addition to thermomorphogenesis, adaptation to high ambient temperature also involves physiological processes such as photosynthetic acclimation, respiration and changes in carbon balance. For discussions of these topics as well as on phenological changes including premature flowering, we refer the reader to reviews elsewhere[20,37–39].

Growth and developmental effects of high temperature

To the best of our knowledge, the term 'thermomorphogenesis' was coined by Erwin and colleagues[16], in analogy to photomorphogenesis (light-mediated growth), to describe the effects of temperature on plant morphology. Here, we will define it as the suite

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Figure 1 | **Typical thermomorphogenesis phenotypes of *Arabidopsis thaliana* plants. a**,**b**, Drawings of thermomorphogenic phenotypes at the young seedling stage (**a**) and the vegetative stage (**b**). Temperature-induced elongation growth of hypocotyls and petioles, and hyponasty of petioles and leaves, occur in both seedlings and vegetative plants, resulting in an open rosette structure that favours leaf-cooling capacity.

of morphological changes that together are likely to contribute to adaptive growth acclimation to otherwise detrimental high ambient temperature conditions.

Elongation of the hypocotyl is one of the earliest thermomorphogenic effects seen in seedlings across *Arabidopsis* accessions in response to high ambient temperature[22–36,40–50] (Fig. 1a and Table 1). It has been suggested that hypocotyl elongation moves the sensitive meristematic and photosynthetically active tissues away from heat-absorbing soil and may promote cooling by allowing better access to moving air[31].

Rosette leaves and cotyledons exhibit marked petiole elongation upon sensing of high ambient temperatures[17–20,22,23,28,30,35,36,41,45,50] and move upwards, a process called hyponastic growth[18–20,22,36,45,51–54] (Fig. 1a,b and Table 1). It has been argued that hyponasty reduces direct heat flux from the Sun and, again, allows a cooling breeze to reach the leaves[17–20]. Together with petiole elongation, hyponasty results in an open rosette structure. Plants grown at high ambient temperature exhibiting these phenotypes showed greater transpiration rates and had cooler leaves than their cool-grown counterparts, when both groups were subjected to high temperature conditions[17]. These data suggest that thermomorphogenic adaptations may contribute to high temperature mitigation by enhancing leaf evaporative cooling[17,18]. This idea was supported by mathematical models, which predicted that a combination of petiole elongation and hyponastic growth may operate in concert to sufficiently separate leaves from both the soil and each other to assure optimal transpiration and leaf cooling under well-watered conditions[17,18]. In addition, plants grown at high temperature have fewer stomata and develop smaller and thinner leaves (Table 1)[17,28,45,53,54]. These phenotypes may further assist cooling by reducing boundary-layer thickness, which stimulates heat dissipation by evaporation and convection[17–20].

PIF4 is a hub in ambient-temperature signalling

Changes in plant morphology initiated by high ambient temperature and by vegetation shade are very similar[55], indicating the possibility of shared signalling elements. This idea led to the identification of the bHLH transcription factor PIF4 as a key regulator of thermomorphogenic phenotypes including hyponasty, hypocotyl and petiole elongation[22,29,30,32,56,57]. As discussed below, PIF4 (and to a lesser extent PIF5) performs its pivotal function in high-temperature signalling by orchestrating transcriptional changes that subsequently trigger primarily phytohormone-induced elongation responses.

Shortly after shifting plants to high ambient temperature, a notable increase in *PIF4* transcript has been observed, triggering thermomorphogenesis[22,30,32]. But thermomorphogenesis needs to be precisely timed and restrained, in order, for example, to balance elongation growth against biomass production[58]. A complex circuitry of PIF4 regulation is therefore at play that includes gene expression, epigenetic regulation, protein stability, protein sequestration, promoter access and promoter competition (Fig. 2). This tight control of PIF4 activity and other coordinating factors is indispensable for the integration of various environmental signals into plant morphogenesis and growth control.

Transcriptional regulation of *PIF4*. Expression of *PIF4* itself is rhythmic and tightly regulated by the circadian clock (Fig. 2a)[59–62]. The clock regulates the rhythmic expression of *PIF4* and *PIF5* through repression by the 'evening complex' (EC), consisting of the proteins EARLY FLOWERING 3 (ELF3), ELF4 and LUX ARRHYTHMO (LUX)[59,62]. Transcription profiles of core clock genes show temperature-induced alterations in extended dark periods[25]. In diurnal conditions, however, clock gene expression is largely robust over a wide range of ambient temperatures. This temperature compensation seems to be primarily maintained through the clock components LATE ELONGATED HYPOCOTYL (LHY) and GIGANTEA (GI)[63]. It is possible that clock and temperature information are transmitted to PIF4 directly through ELF3, as the ability of ELF3 to bind target genes is attenuated at 27 °C (ref. 26). Interestingly, two recent studies indicated that genetic variation in *ELF3* explains a large part of natural variation in temperature-induced *PIF4* expression and elongation growth among *Arabidopsis* accessions[26,28]. When the EC peaks in the early night, *PIF4* expression is suppressed[64,65]. Reduction of EC as the night progresses then leads to a rise in *PIF4* levels. Post-dawn decrease of *PIF4* levels, however, suggests the involvement of other transcriptional repressors. As an additional level of regulatory control, ELF3 can also directly bind to PIF4 protein[66].

In the light, *PIF4* restriction probably involves a similar repression mechanism facilitated at least partially by the bZIP transcription factor LONG HYPOCOTYL 5 (HY5)[67–69] (Fig. 2a). *hy5* mutants grown at standard growth temperatures (20 °C) show increased *PIF4* expression at midday and a transiently increased expression in response to elevated temperature[41]. Genome-wide ChIP analyses have identified *PIF4* promoters as HY5 targets[70],

Table 1 | Thermomorphogenesis in *Arabidopsis thaliana*

Trait	Effect	Range (°C)	Ref.	*Arabidopsis* accessions used
Hypocotyl elongation	∧	17–27	43	Col-0
	∧	16–24	45	Col-0, Bay-0, C24, Cvi-0, Got-7, Rrs-7, Sha, Ws-2
	∧	20–28	28,29,33,41	Col-0, Ws-2, Ler, Rrs-7, Bay-0, Sha, Sf-2, Zu-0
	∧	20–29	31,32,42,44	Col-0, Ler (+20 from ref. 42*)
	∧	22–27	26,35	Col-0 (+19 from ref. 26†)
	∧	22–28	22–25,46	Col-0
	∧	22–29	30,47,48	Col-0, Ws-2, Ler
	∧ / = / ∨	23–27	49,50	Col-0 (∧), Sij-4 (=) (+139 from ref. 50‡)
	∧	23–28	36	Col-0
	∧	Various§	40	Estland
Petiole elongation	∧	16–24	45	Col-0, Bay-0, C24, Cvi-0, Sha, Ws-2
	∧	20–28	28,41	Col-0, Rrs-7, Bay-0, Sha
	∧	22–27	35	Col-0
	∧	22–28	17,22,23	Col-0
	∧	23–28	37	Col-0
	∧	22–29	30	Col-0
Hyponastic growth	∧	16–24	45	Col-0, Bay-0, C24, Cvi-0, Got-7, Rrs-7, Sha, Ws-2
	∧	Various‖	52	Col-0, Ws-2, Ler
	∧	22–28	22	Col-0
	∧	23–28	36	Col-0
	∧	20–30	51,53¶,54	Col-0, Ler, An-1, Cvi-0
Stomatal density	∨	22–28	17	Col-0
	∨	20–30	53¶,54	Col-0, Ler, An-1, Cvi-0
Leaf area	∧	20–28	28	Bay-0, Sha
	∨	22–28	17	Col-0
Leaf thickness	∨	22–28	17	Col-0
	∨ / =	20–30	53¶,54	Col-0, Ler, An-1, Cvi-0
Specific leaf area ($cm^2 g^{-1}$)	∧ / =	20–30	53¶,54	Col-0, Ler, An-1, Cvi-0
Ratio of blade length/total leaf length	∨ / =	20–30	53¶,54	Col-0, Ler, An-1, Cvi-0
Root elongation	∧	16–24	45	Col-0, Bay-0, C24, Cvi-0, Got-7, Rrs-7, Sha, Ws-2
	∧	23–29	100	Col-0

Table shows typical phenotypes associated with thermomorphogenesis; the effect direction (increase, ∧; decrease, ∨; or equal, =); the temperature treatment that was commenced in the experiments; and the accessions used in the respective studies. *Delker *et al.*[42] used 20 accessions, which all elongated. †Box *et al.*[26] used 19 accessions, which all elongated. ‡Sanchez-Bermejo[50] used 139 accessions, with the majority of accessions displaying elongation. §Orbovic and Poff[40] shifted plants between various temperatures. ‖Van Zanten *et al.*[52] used a range between 20 and 42 °C. ¶Effects derived from Vile *et al.*[53] are based on averages of 10 accessions; each accession showed the same trend.

and a temperature-insensitive quadruple *pif* mutant suppressed the temperature-hypersensitivity of *hy5* mutants[41]. Interestingly, HY5 protein is less abundant at higher temperatures[69], which presumably dampens HY5 control of PIF4 in warm conditions. Thus, temperature-dependent transcriptional release of *PIF4* by reducing HY5 levels, probably through the DE-ETIOLATED 1 (DET1)–CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) regulatory cascade[41], may represent a mechanism to control *PIF4* transcript levels in a light- and temperature-dependent manner.

Post-translational regulation of PIF4 protein levels. In addition to control at the transcriptional level, PIF4 is also subjected to post-translational control. PIF4 interacts with several proteins, which can affect its activity or stability. The name-giving interaction with phytochrome B (phyB) in the light, for example, results in phosphorylation and subsequent ubiquitination followed by proteasomal degradation of PIFs[71] (Fig. 2b). The kinase BRASSINOSTEROID-INSENSITIVE 2 (BIN2) has also been shown to phosphorylate PIF4 preferentially in the light, restricting the daytime impact of PIF4 by depleting protein levels[72]. As high temperature triggers accumulation of phosphorylated PIF4 in red and blue light, however, light-mediated phosphorylation does not necessarily result in degradation of the protein[58]. Possibly, differential phosphorylation patterns by independent kinases occur in response to distinct stimuli, resulting in different fates of the protein.

Recently, interaction of PIFs with DET1, a repressor of photomorphogenesis, has been shown to stabilize PIFs and counteract their degradation[73,74] (Fig. 2b). Whether this process directly contributes to the regulation of PIF activity in response to elevated temperatures remains to be elucidated. However, *det1* mutants are impaired in temperature-induced hypocotyl elongation[41], which could very well indicate a dual role of DET1 in temperature-dependent PIF regulation through direct interaction/stabilization, and also DET1-COP1-mediated HY5 degradation.

Interaction with other proteins can also sequester free PIF4 protein, preventing its DNA-binding and downstream transcriptional regulation[48,58,75]. Among these, LONG HYPOCOTYL IN FAR-RED 1 (HFR1), which accumulates in a CRYPTOCHROME 1 (CRY1)-dependent manner, acts as a negative regulator in temperature responses under monochromatic blue light[58]. This process may also

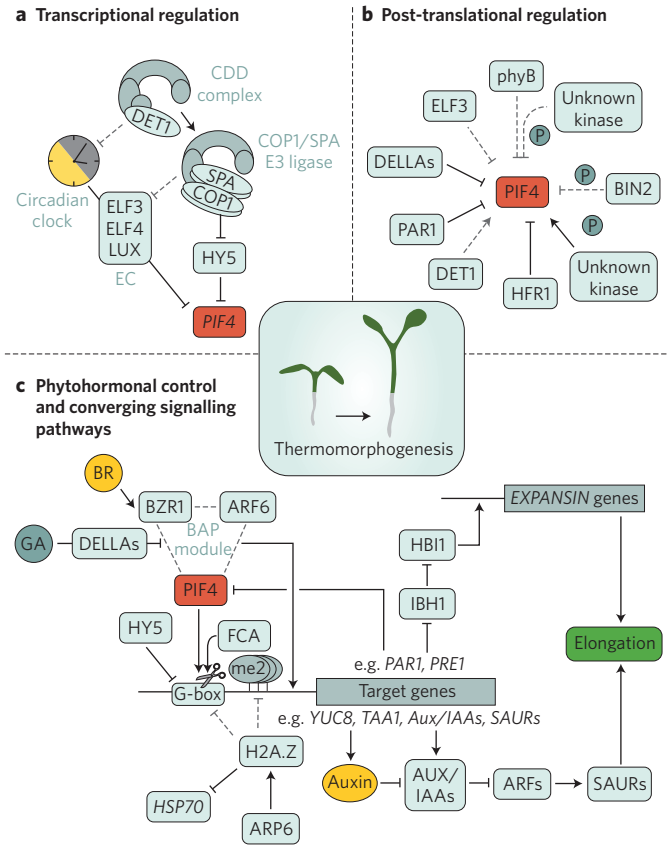

Figure 2 | Simplified model of the central role of PIF4 in the molecular genetic circuitries underlying thermomorphogenesis. a, In darkness, transcriptional regulation of *PIF4* involves gating by means of the evening complex (EC) of the circadian clock. In the light, transcriptional repression by HY5 is relieved by the COP1-SPA E3 ubiquitin ligase and the COP10–DDB1–DET1 (CDD) complex. **b**, PIF4 post-translational regulation contributing to temperature signalling involves phosphorylation by an as-yet unidentified kinase and sequestering of free PIF4. It remains to be established whether other PIF4-interactors/modifiers known from the light signalling context contribute to temperature signalling. **c**, PIF4 mediates transcriptional regulation of its target genes through binding to G-box promoter elements. This regulation is counteracted by HY5, which competes for the same binding sites. In addition, FCA can attenuate PIF4–G-box binding by removing H3K4Me2 chromatin marks. Further chromatin modifications such as eviction of H2A.Z-containing nucleosomes have been shown to contribute to thermomorphogenesis. However, whether this process directly affects PIF4-target genes remains to be established. Elongation growth is subsequently triggered by PIF4-mediated induction of auxin biosynthesis and auxin signalling, resulting in SAUR-mediated elongation growth, and by a cascade involving PAR1, PRE1, IBH1 and HBI1, ultimately resulting in the induction of *EXPANSIN* genes. Both downstream pathways involve feedback regulations and, at least partially, the transcription factors BZR1 and ARF6 (BAP module). Other phytohormones are involved in thermomorphogenesis, with brassinosteroids (BR) and gibberellic acid (GA) having an essential or permissive signalling function, respectively, involving the DELLA repressor proteins. **a**–**c**, Mechanisms with demonstrated relevance in temperature signalling are depicted by solid black lines; connections known from other biological processes that may potentially contribute to temperature signalling are shown as dashed grey lines.

contribute to PIF4 regulation in blue-light-rich white light conditions (Fig. 2b).

In addition, PIF4 access to target promoters seems to be under tight control as well. Here, competition for mutual regulatory DNA-binding sites can occur among PIF4 and HY5, which differentially affects the transcriptional activity of target genes[69]. As increasing temperatures result in decreased HY5 and increased PIF4 protein levels[22,32,69], the alteration in protein ratios can quantitatively affect target gene expression levels.

PIF4-mediated regulation of phytohormones

Phytohormone biosynthesis and signalling genes represent prominent PIF4 targets[32], thereby connecting PIF4 activity with the long-known essential role of phytohormones in thermomorphogenesis[31] (Fig. 2c).

Auxin and auxin signalling are required and sufficient for PIF4-mediated hypocotyl elongation induced by high temperature, and for other thermomorphogenic responses[29–32]. At high ambient temperatures, free indole-3-acetic acid (IAA) levels in aerial tissues are increased[29–31]. This is probably caused by temperature-mediated binding of PIF4 to promoters, and subsequent activation of auxin biosynthesis genes such as *YUCCA 8* (*YUC8*), *CYTOCHROME P450 FAMILY 79B* (*CYP79B*) and *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1* (*TAA1*)[29,30] (Fig. 2c). In support of this, IAA levels do not increase at high ambient temperatures in *pif4* mutants[29–31].

Increased intracellular auxin levels initiate gene expression changes by means of the TRANSPORT INHIBITOR 1/AUXIN SIGNALING F-BOX proteins (TIR1/AFBs) signalling pathway[77]. Auxin binding by a co-receptor complex formed by TIR1/AFBs and members of the AUXIN/INDOLE-3-ACETIC ACID (AUX/IAA) protein family results in the subsequent degradation of AUX/IAAs and the initiation of transcriptional auxin responses[78]. Accordingly, mutants defective in one or more of the partially redundant TIR1/AFBs show reduced temperature-induced hypocotyl elongation[31,41].

Among the temperature-inducible auxin response genes are the *SMALL AUXIN UP RNA 19–24* (*SAUR19–24*) and *SAUR61–68* subfamilies[29,32]. Several members of this gene family have been shown to regulate elongation growth, probably by increasing H^+-ATPase activity at the plasma membrane[79–81]. Accordingly, the overexpression of stabilized GFP-SAUR19 rescues the thermomorphogenic hypocotyl elongation defect of the *pif4* mutant[29]. Besides SAURs, EXPANSIN cell-wall-loosening enzymes directly affect cell elongation, and, interestingly, temperature-induced expression of an *EXPANSIN* gene was positively correlated with heat tolerance in the grass *Agrostis scabra*[82]. Furthermore, *EXPANSIN* expression in response to light and GA has been shown to depend on PIF4[75], which makes it likely that temperature control of *EXPANSIN*s also requires PIF4[44].

In addition to auxin, brassinosteroids (BR) and gibberellins (GA) play crucial roles in high-temperature-induced hypocotyl elongation[22,31–34,48,83,84] (Fig. 2c). The transcription factor *BRASSINAZOLE RESISTANT 1* (*BZR1*), for instance, is involved in the regulation of temperature-induced hypocotyl elongation in a PIF-dependent manner and directly interacts with PIF4[33]. Furthermore, the *det2-1* BR biosynthesis mutant displays defects in thermomorphogenic responses[31], and pharmacological inhibition of BR signalling inhibits temperature-induced growth[32]. Consistent with the currently understood molecular mechanism for synergistic interaction of auxin and BRs, a highly active BR pathway might sensitize seedlings for the temperature-induced increase in auxin levels[32]. This might be mediated through the regulation of transcription factor activity. PIF4 and BZR1 directly interact with AUXIN RESPONSE FACTOR 6 (ARF6) and enhance its binding to promoters. Accordingly, the BZR1/ARF6/PIF4 (BAP) module synergistically regulates many shared target genes that may ultimately trigger elongation growth[34,84] (Fig. 2c). But it remains unclear whether ARF6 has a role in thermomorphogenesis, and the exact role of BR also requires further investigation.

Gibberellin (GA) presence leads to degradation of growth-repressive DELLA proteins that inhibit PIF action in light signalling[75,85]. Moreover, Stavang and colleagues[32] demonstrated a rapid

up-regulation of the major GA biosynthesis genes *AtGA20ox1* and *AtGA3ox1* in *Arabidopsis* seedlings subjected to elevated temperatures, whereas the prominent catabolism gene *AtGA2ox1* was down-regulated. Consistent with these observations, detailed mutant analyses showed that both GA biosynthesis and signalling are required for the promotion of thermomorphogenesis[32]. This suggests that the GA pathway is more active at high ambient temperatures, putatively as a result of increased GA levels and release of DELLA-dependent PIF4 sequestering. In contrast to auxin, however, the GA pathway seems insufficient to induce thermomorphogenesis, because quintuple *della* mutant seedlings still show a partial hypocotyl elongation response[22,32]. Interestingly, GA-mediated cell elongation requires BRs, auxin, BZR1 and PIF4, and it was shown[34,83] that DELLA growth repressors directly interact with BZR1 and ARF6. GA presence releases DELLA-mediated repression of BZR1 and ARF6 to allow BAP-module function and subsequent induction of hypocotyl elongation[34,83] (Fig. 2c). Hence, GA seems permissive, rather than regulatory, by modulation of PIF4 activity.

Multiple signalling pathways converge at PIF4

Tight regulation of PIF4 and its downstream auxin biosynthesis and signalling targets is required to assure that cooling capacity is achieved, while physiological imbalance and exaggerated elongation growth is prevented. Therefore, several signal transduction pathways converge on PIF4 in addition to the (post-)transcriptional regulatory mechanisms discussed above.

One such pathway involves feedback regulation by *AUX/IAA* auxin signalling genes (Fig. 2c). Various *AUX/IAA*s (for example, *IAA4* and *IAA29*) are induced under high ambient temperatures in a PIF4-dependent manner[22,31]. Auxin-mediated degradation of AUX/IAAs and subsequent release of ARF transcription factors is essential for thermomorphogenesis. Yet, the TIR1/AFB-independent direct and rapid induction of the genes encoding AUX/IAA transcriptional repressors by PIF4 also provides the possibility of a fast and timely attenuation of the auxin stimulus when auxin levels decrease. Consistent with this idea, gain-of-function mutations in several *AUX/IAAs* (for example, *IAA3/SHY2* and *IAA19/MSG2)* can suppress PIF4-mediated hypocotyl elongation at high temperatures[30,47].

A recent study described the involvement of epigenetic silencing of the auxin biosynthesis gene *YUC8* to attenuate thermomorphogenesis[36]. Mutants in the RNA-binding protein FLOWERING TIME CONTROL PROTEIN A (FCA) exhibited increased PIF4 binding to the *YUC8* promoter (Fig. 2c). Accordingly, *fca* mutants displayed increased auxin levels and exhibited enhanced hypocotyl and petiole elongation as well as hyponasty under both control and elevated temperatures[36]. Furthermore, enhanced levels of the activating epigenetic histone mark H3K4me2 on chromatin of the *YUC8* promoter were observed at high temperatures, which was further stimulated in the *fca* mutant background[36]. Taken together, the results suggest that PIF4 binds to the *YUC8* promoter and stimulates auxin biosynthesis driving thermomorphogenesis shortly after high temperature sensing, followed by PIF4-mediated recruitment of FCA. This leads to removal of activating H3K4me2 marks and subsequent dissociation of PIF4 from the *YUC8* locus, resulting in attenuation of thermomorphogenesis[36].

Additional regulation of PIF4 may be conferred through HLH factors (Fig. 2c). The non DNA-binding HLH factor PHYTOCHROME RAPIDLY REGULATED 1 (PAR1) attenuates high-temperature-mediated elongation responses through direct inactivation of PIF4[48], resulting in decreased high-temperature-induced hypocotyl elongation[48]. Furthermore, the BAP module stimulates the expression of another non-DNA-binding HLH factor *PACLOBUTRAZOL RESISTANCE 1* (*PRE1*)[34,83]. PRE1 acts as a positive regulator of thermomorphogenesis as part of a module of three HLH/bHLH factors, together with ILI1 BINDING BHLH1 (IBH1) and HOMOLOG OF BEE2 INTERACTING WITH IBH1 (HBI1)[34,44,83]. Sequestration of IBH1 by PRE1 enables the binding of HBI1 to the promoters of *EXPANSIN* genes[44], promoting cell-wall loosening and hypocotyl elongation (Fig. 2c). Consistent with this model, high-temperature-induced hypocotyl elongation is severely reduced in transgenic lines displaying reduced PRE1/HBI1 or enhanced IBH1 levels[34,44,83].

Modelling-based integration of light, circadian and temperature signals in the control of thermomorphogenesis. The studies outlined above illustrate that PIF4 associates with a number of proteins that collectively integrate multiple environmental and endogenous stimuli to control thermomorphogenesis. Although we already have detailed knowledge of some molecular events, we are still some way from understanding how the network operates at a whole system level. When striving to do this, lab-to-lab variation in experimental regimes, and limited access to quantitative data, can provide additional obstacles. Thus, linking new and published data to gain a comprehensive understanding of thermoregulation is not a trivial process. Despite these constraints, mathematical modelling has emerged as a valuable approach to learning how complex biological systems work. Modelling provides a formal means to consolidate knowledge, challenge our current understanding and derive new and experimentally testable hypotheses. Recently, a combination of modelling and experimental approaches was successfully applied to address the complex regulatory circuitry underlying morphogenesis by connecting the circadian clock, light and temperature to identify new regulators and interconnections and to explain regulatory switches in response to multiple conflicting stimuli[27,43,86].

Initial groundwork in this area was laid by Rausenberger and colleagues[87], who constructed the first kinetic model for light signalling. This model captured key aspects of phyB photochemistry including photoreceptor protein dynamics to hypocotyl length[87,88]. The model also highlighted the combined network features that were required to deliver fluence-rate dependency of phyB. A more recent study extended the Rausenberger[87] model to incorporate PIF control of hypocotyl elongation[43]. This revised model provided a framework to understand how changes in the light and temperature environment alter signalling through the phyB–PIF circuit. The study revealed that temperature has a strong impact on how light regulates hypocotyl elongation by showing that fluence-rate-dependent hypocotyl elongation is attenuated at 22 °C compared with 17 °C. Furthermore, at 27 °C increasing fluence rates do not inhibit but instead promote elongation above a low irradiance threshold. This implies that temperature can completely switch the mode of light action, possibly by increased photoconversion between active Pfr and inactive Pr forms at higher fluence rates, resulting in less efficient phyB signalling. This scenario predicts that phyB would be less effective in degrading PIF proteins at increased fluence rates at 27 °C. However, this is not the case, as a strong fluence-rate-dependent depletion of PIF4 (and PIF3) protein levels was observed at both 22 °C and 27 °C (ref. 43). Model analysis provided an alternative hypothesis: that fluence-rate-dependent factors are required to modulate PIF activity. At moderate temperatures these factors suppress PIF action, but at higher temperatures they activate PIFs. This hypothesis was partially validated, as HY5 was shown to be a strong PIF suppressor at cooler temperatures, particularly as fluence rates increase[43,69]. Nevertheless, the molecular or biochemical entity that mediates light activation of PIFs at higher temperatures has yet to be determined.

Although such steady-state hypocotyl models provide useful formats to conduct network structure–function analyses, rhythmicity of PIF-mediated hypocotyl elongation requires integration of the circadian clock and natural photoperiods[59–62]. A study by Seaton and colleagues[27] constructed the first external coincidence model for hypocotyl growth. This was accomplished by integrating the evening complex (EC) and light regulation of PIF4, PIF5 and their direct

Figure 3 | Thermomorphogenesis in crop species. Compared with the situation in the model plant *Arabidopsis* (see Fig. 1a), temperature-induced hypocotyl elongation seems widely conserved among crop species. Shown here are cabbage (*Brassica oleracea*) and tomato (*Solanum lycopersicum*). Both have been grown for 7 days at 20 °C versus 7 days at 28 °C under long-day conditions with 90 μmol m^{-2} s^{-1} white light.

targets, *ARABIDOPSIS THALIANA HOMEOBOX 2* (*ATHB2*) and *IAA29*[59,61,76]. This model configuration matched observed photoperiod responses of *ATHB2* and *IAA29* in wild-type and simulated clock mutants. As temperature modulates *PIF4* expression through the EC[25,26], the authors[27] tested whether this response could be captured by the model. By introducing temperature modulation of EC affinity for the *PIF4* promoter, the model was able to match the temperature-induced early rise of *PIF4* expression, and the associated changes in *ATHB2* and *IAA29*, substantiating the proposed mode of thermal *PIF4* regulation through the EC.

Based on the described examples, combining modelling and experimental approaches has proved to be important in deciphering biological complexity. The highlighted studies[27,43,68] provide conceptual frameworks to understand how the mode of PIF4 control by light is switched by temperature, and to understand the temperature-dependent nocturnal rise in PIF4 transcription in a diurnal cycle. The study by Seaton and colleagues[27] also provides a systems-level understanding of how temperature and photoperiodic signals integrate to control growth.

Chromatin-level regulation of thermomorphogenesis

Temperature influences virtually every biological process, and a key feature of investigating the impact of temperature on any given organism is that passive, thermodynamic effects of temperature on biomolecules need to be separated from active thermal perception and signalling[89]. Among the processes that are tightly controlled by temperature are gene transcription and mRNA degradation. Sidaway-Lee and colleagues[90] noted that both transcription and mRNA decay rates passively increased in response to higher ambient temperatures in *Arabidopsis*. In an effort to dissect active and passive thermal regulation, they found that active temperature-directed changes in mRNA abundances could be assigned to temperature-mediated regulation of transcription, rather than to mRNA decay[90]. The authors next determined which epigenetic modifications were related to temperature-mediated transcriptional regulation and found that H3K27me3 was associated with genes exhibiting both high and low levels of temperature-dependent transcriptional regulation. This epigenetic mark was depleted from genes showing passive temperature-mediated regulation only[90]. Global changes in several other epigenetic marks, including H3K4me3, H3K9Ac and DNA methylation, were not implicated in active thermoregulation of gene expression[90], but contribution of these marks to the expression of specific thermomorphogenesis-regulating genes cannot be excluded. The prominent role for epigenetic modifications in thermomorphogenesis control was recently supported by the example of FCA-mediated H3K4me2 removal from the *YUC8* promoter, described above, which probably restricts PIF4 binding and thereby attenuates thermomorphogenesis[36].

In addition to epigenetic modifications, chromatin remodelling has a prominent role in thermomorphogenesis. ACTIN RELATED PROTEIN 6 (ARP6) controls H2A.Z-nucleosome incorporation into chromatin[91], and plants carrying mutations in *ARP6* display several aspects consistent with a constitutive thermomorphogenic response, such as longer hypocotyls and petioles and a transcriptome profile typical for high ambient temperatures, even at lower growth temperatures[35] (Fig. 2c). This implies a role for H2A.Z-containing nucleosomes in thermal regulation of transcription. H2A.Z-nucleosomes are highly enriched at the beginning of genes at the +1 position, adjacent to the transcription start site. For some genes, such as *HEAT SHOCK PROTEIN 70* (*HSP70*), it has been shown that the occupancy of the +1 H2A.Z-nucleosome is rate-limiting for expression. Consequently, *HSP70* was more highly expressed in the *arp6* background compared with wild type at low ambient temperatures. Based on these observations, it was hypothesized that the observed high-temperature-induced H2A.Z eviction may provide thermal information to the cell by allowing better accessibility for transcriptional regulators that ultimately orchestrate thermomorphogenesis[35]. H2A.Z eviction therefore seems to enable temperature-dependent expression for at least some — and possibly many — genes. A key question is whether H2A.Z-nucleosome eviction is a direct response to temperature (suggesting that it is thermosensory) or whether it is mediated indirectly, for example by means of a temperature-responsive chromatin remodelling factor. Notably, however, *arp6* mutants still show an increase in hypocotyl elongation at warmer temperatures, suggesting that H2A.Z-nucleosomes themselves do not transmit all temperature information.

Future challenges, knowledge transfer and conclusions

Numerous open questions about temperature signalling and response networks remain to be resolved before comprehensive understanding of thermomorphogenesis regulation is reached. It is likely that many relevant thermomorphogenesis regulators remain to be identified and their signalling hierarchies need to be investigated to understand how multiple conflicting signals are integrated in coordinated plant growth and development. Importantly, the thermomorphogenesis mechanisms described here are probably operating across a broad range of non-damaging temperatures, beyond the somewhat rigid temperature range of ~20 to ~29 °C normally used in thermomorphogenesis research in *Arabidopsis* (Table 1). To fully understand plant acclimation to warmer temperatures, a broader temperature range needs to be taken into account. Above all, however, the exact mechanisms by which small changes in ambient temperature are sensed remain enigmatic. H2A.Z eviction and subsequent changes in chromatin suggest a possible temperature-sensing mechanism, but this needs to be confirmed. The data are consistent with a model whereby H2A.Z-nucleosomes at the transcriptional start site[35] and/or the gene body[90] may be rate-limiting for the expression of other key genes in the thermomorphogenesis pathway, such as *PIF4*, or PIF4 targets. Alternatively, the enhanced elongation phenotype of *arp6* may arise from a parallel pathway.

Our currently rather limited understanding of ambient temperature perception is in contrast to many other signal transduction pathways. This may be due in part to the involvement of numerous processes, prohibiting the elucidation of a 'temperature receptor'. Among these, temperature effects on transcriptional rates, protein–protein interaction, protein turn-over, changes in subcellular localization and changes in rates of metabolism might intricately contribute to altered physiological read-outs of known and unknown signalling processes. The recent identification of natural *CRYPTOCHROME 2* alleles and their role in thermomorphogenesis[50] emphasizes that

the identification of additional, as yet unknown rate-limiting and crucial signalling hubs within this network of sensors and response elements constitutes a considerable challenge, as does experimental design and interpretation. In this respect, the role of metabolism in thermomorphogenesis deserves more attention. Carbon starvation occurs in plants shifted to high ambient temperatures, and this correlates with thermomorphogenesis phenotypes[54]. Moreover, PIFs including PIF4 are required for sucrose-induced hypocotyl elongation, and PIF5 has been shown to be stabilized by sucrose[92,93]. Sugars induce auxin biosynthesis by stimulating auxin biosynthesis genes[94], an effect that might potentially be counteracted or enhanced by PIFs depending on specific growth conditions. Such data underscore that temperature, light, sugars, PIFs and auxin are part of a complex and not yet well-understood circuitry integrating environmental and metabolic cues into a coordinated growth response. Genetic analysis can be used to provide insight into the complex molecular networks underlying thermomorphogenesis, but major advances will require the combination of wet-lab genetic, physiological and biochemical approaches together with *in silico* modelling of dynamic structural plant phenotypes and the underlying genetic circuitries.

One aspect that needs particular consideration is the interaction of thermomorphogenesis with other environmental stresses. The relationship with drought deserves more attention, as thermomorphogenesis assists cooling by enhancing transpiration, which is only favourable under well-irrigated conditions[17]. Water is already growth-limiting in many parts of the world[95], and high temperatures and drought often occur simultaneously, suggesting that thermomorphogenic acclimation is not beneficial and can be even detrimental in these conditions. Accordingly, when combined, high temperatures and drought result in more severe inhibition of growth in plants than observed for only one individual stress[53]. Both stresses have impacts on growth through partly separate and partly parallel mechanisms that become additive when experienced together. Therefore, it is important to assess the contribution of thermomorphogenesis-regulatory networks on plant acclimation to other stresses and their combinations.

Climate change has already caused large-scale changes in distribution and behaviour of wild species, and unseasonably hot weather led to global disruptions in crop productivity, for example in 2003 and 2012. Further temperature increases during this century are forecast to exacerbate these problems[3–9,13–15]. Crop-level adaptations have the potential to reverse projected detrimental effects of climate change on agricultural yield[13–15]. Such adaptations could include the use of alternative varieties or even species, planting times, irrigation and fertilization regimes. Of all possibilities, cultivar adaptations are predicted to have the greatest positive impact on yields under the projected climate change[13]. If understood, one promising and socially accepted way to improve thermomorphogenic acclimation would be allele-mining combined with marker-assisted breeding approaches. In this respect, the general conservation of thermomorphogenesis responses in crop species is certainly promising (Fig. 3). However, in a study on genetic variability in developmental rates in 18 species, including the 14 most cultivated crops worldwide, it was found that temperature dose–response curves of developmental processes are strikingly similar between cultivars/lines even if these originated from very different climates[96]. It is therefore likely that current crop-breeding approaches will need to be complemented with more directed genetic engineering approaches that enable genes from a wider range of backgrounds, as well as potentially synthetically designed genes with optimized temperature-response properties, to be introduced into key crops. A considerable advance in making this approach feasible is the advent of CRISPR/Cas9 technology enabling genome-wide targeting of genetic alterations. Additionally, it may be necessary to combine multiple genes or entire pathways to obtain desired crop protection, something that may not be feasible with conventional breeding approaches alone.

Potential targets for mining of favourable natural alleles could include the receptor-like kinase *ERECTA*, which was recently shown to play a critical role in high-temperature stress tolerance[97]. ERECTA probably acts by protecting against temperature-induced cellular damage, since overexpression of *ERECTA* conferred high temperature tolerance on *Arabidopsis*, tomato and rice in greenhouse and field conditions, without compromising growth and yield. Key thermomorphogenesis regulators such as PIF4 and elements of the EC are also good candidates. Allelic variation in *ELF3*, *ELF4*, *LUX* and other clock components, for example, has contributed to the domestication of several crop species in terms of flowering time adaptation[98]. Based on the experimental work in *Arabidopsis*, allelic variation of EC components can significantly affect thermomorphogenesis under controlled environmental conditions[26,28]. It remains to be investigated whether these alleles also cause differential temperature responses under natural environmental conditions, and whether similar differences can be observed in different crop species. On the bright side, the observation that H2A.Z-nucleosome-mediated temperature responses in the monocot model species *Brachypodium distachyon*[99] are similar to those observed in the dicot *Arabidopsis* suggests that at least some of the main molecular circuitries underlying thermomorphogenesis are functionally conserved.

Meeting future challenges to plant productivity imposed by globally increasing temperatures will require basic research in model plant species as well as applied approaches in crops. Integration of these ends of the spectrum will require directed efforts from the academic plant research community and private companies. Further development of thermomorphogenesis as a research area could ultimately provide efficient and timely leads for the initiation of appropriate breeding efforts to generate much-needed thermotolerant crops.

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Competing interests

The authors declare no competing financial interests.