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Plant Thermosensors

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Abstract

Plants are exposed to temperature conditions that fluctuate over different time scales, including those inherent to global warming. In the face of these variations, plants sense temperature to adjust their functions and minimize the negative consequences. Transcriptome responses underlie changes in growth, development, and biochemistry (thermomorphogenesis and acclimation to extreme temperatures). We are only beginning to understand temperature sensation by plants. Multiple thermosensors convey complementary temperature information to a given signaling network to control gene expression. Temperature-induced changes in protein or transcript structure and/or in the dynamics of biomolecular condensates are the core sensing mechanisms of known thermosensors, but temperature impinges on their activities via additional indirect pathways. The diversity of plant responses to temperature anticipates that many new thermosensors and eventually novel sensing mechanisms will be uncovered soon.

1. INTRODUCTION

1.1. Temperature Fluctuations Impose Challenges

Plants are exposed to temperatures that fluctuate over different time scales, including hours, days, seasons, and years, particularly with climate change (18). This situation imposes two challenges. First, plants can eventually become exposed to extreme temperatures, which cause membrane dysfunction, protein denaturation, and the generation of reactive oxygen species. Second, even without reaching those extremes, temperature affects plant resources. For instance, low temperatures increase water viscosity and slow down the release of minerals by the microbiome and the diffusion of ions, making these resources less accessible to the root. Warm temperatures increase transpiration, respiration, and photorespiration, reducing water and carbohydrate pools.

1.2. Plants Adjust to the Temperature Environment

Plants have two nonmutually exclusive strategies to deal with the temperature challenges: temperature acclimation and thermomorphogenesis. Temperature acclimation is a set of biochemical changes induced by cold or warmth that reduces the damage caused by the eventual exposure to more extreme temperatures. Thermomorphogenesis is the effect of temperature on plant morphogenesis, that is, on plant growth and development. Following this etymological definition, different organs and developmental transitions can vary in the temperature cues that they perceive. The rates of growth and development typically increase with average daily temperatures up to an optimum, beyond which they decrease with further temperature increments (92). However, there are processes that require cold to proceed, such as stratification to reduce seed dormancy facilitating germination and vernalization to accelerate flowering. Others require alternating temperatures, such as the germination of some seeds. The term thermomorphogenesis is often limited to the effects of warm temperatures compared to mild ones (101). To initiate acclimation or thermomorphogenesis, plants must sense temperature.

1.3. Temperature Sensation

Temperature sensation occurs when thermosensory receptors detect changes in the temperature stimulus. Sensation and perception are intrinsically linked but not the same. Perception involves a network that includes the thermosensors plus other signaling nodes that transform temperature sensations into information about the environment. For instance, sensing cold temperatures can inform that winter is about to either start or finish. The former requires short exposures to cold (a few days) and triggers changes that help the plant bear chilling (0°C–15°C) or even freezing (<0°C) conditions (cold acclimation). The latter requires prolonged exposures to cold (weeks to months), gradually increasing the number of cells in which a flowering repressor decreases its abundance until a threshold number of cells is reached, allowing flowering (vernalization) (7). The information provided by temperature cues is not always unequivocal. For instance, a reasonable hypothesis is that the sensation of warmth by young seedlings serves to perceive the danger of hot soil, accelerate the growth of their support organ (the hypocotyl), and move sensitive tissues away from this source of heat (37). However, an argument against this hypothesis is that heating the soil requires strong sunlight, a condition that prevents the hypocotyl growth response to warm temperatures (106).

1.4. Thermosensors

There are multiple ways in which temperature can affect cellular components, but not all these components are thermosensors. A thermosensor must directly decode the temperature stimulus

into cellular signaling by altering its own structure, activity, and/or interaction with other molecular components in order to trigger downstream responses (122). A thermosensor must be the *primum movens* that initiates the pathway of response to temperature (18). Four thermosensors have been identified so far in *Spermatophyta* (seed plants): phytochrome B (phyB) (54, 66), EARLY FLOWERING 3 (ELF3) (53), PHYTOCHROME-INTERACTING FACTOR 7 (PIF7) (21), and THERMO-WITH ABA-RESPONSE 1 (TWA1) (10). This review presents a deep-dive analysis into these thermosensors. For the reader interested in different aspects of the broad field of plant temperature responses, we refer to recent reviews with more emphasis on downstream signaling events (41, 56), thermomorphogenesis (18, 101), auxin-mediated thermomorphogenesis (8), temperature effects on wild and crop species (72), responses to cold temperatures (63), responses to heat (89, 108, 129, 133), responses to stress-inducing temperatures (28, 38), and epigenetic regulation of the responses to warmth and heat (96).

1.5. The Transcriptional Network Involving phyB, ELF3, and PIF7

Of the four known thermosensors, three were uncovered through their role in the promotion of hypocotyl growth by warm temperatures compared to mild ones. Before focusing on their specific sensing mechanisms, it is important to visualize their position within the network that controls this physiological output (**Figure 1*a***).

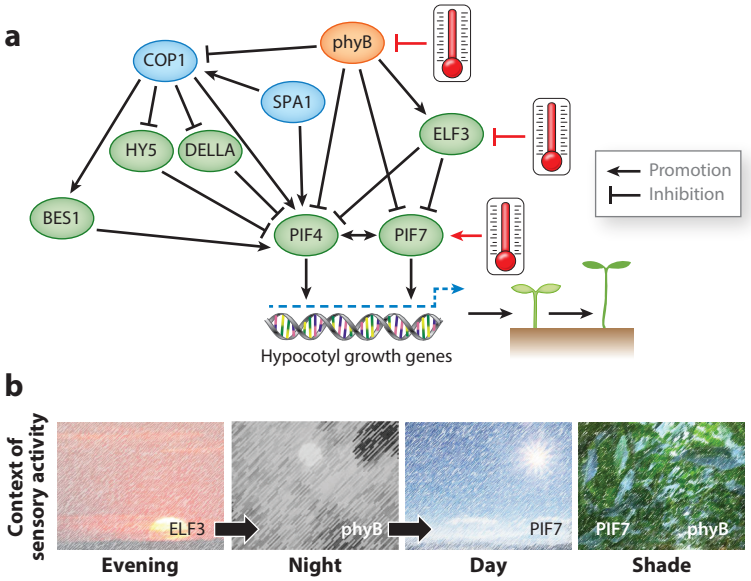


Figure 1

Multiple temperature inputs control the elongation of the hypocotyl in *Arabidopsis thaliana*. (*a*) Three thermosensors (phyB, ELF3, and PIF7) control gene expression to promote the elongation of the hypocotyl in response to warmth. Thermometers indicate the warm temperature inputs operating through the thermosensors. Green represents the transcriptional regulators, blue represents the components of E3 ligase complex, and orange represents the photoreceptor. (*b*) Environmental context in which each thermosensor conveys temperature information to control hypocotyl growth. The thick arrow indicates the carrying-over of information from one context to the other. Abbreviations: BES1, BRI1-EMS-SUPPRESSOR 1; COP1, CONSTITUTIVELY PHOTOMORPHOGENIC 1; ELF3, EARLY FLOWERING 3; HY5, ELONGATED HYPOCOTYL 5; phyB, phytochrome B; PIF, PHYTOCHROME-INTERACTING FACTOR; SPA1, SUPPRESSOR OF PHYA-105 1.

PIF4 (33, 62, 113) and PIF7 (21, 32) are two basic helix-loop-helix transcription factors that, in response to warm temperatures, bind to the G-box motif of the promoter of numerous genes, including auxin synthesis and signaling genes that are important to enhance hypocotyl growth (**Figure 1*a***). PIF4 physically interacts with components of the INO80 chromatin remodeling complex, favoring the removal of H2A.Z and the expression of target genes in response to warm temperature (127) [a similar task could be accomplished by PIF7 (123)]. Thus, the *pif4* and *pif7* mutants show poor hypocotyl elongation in response to warmth (21, 32, 33, 62, 106, 113). PIF7 and PIF4 do not mutually regulate their accumulation, but they interact and form heterodimers, which are important for the expression of growth genes (21, 32, 57). PIF5 and PIF3 (not included in **Figure 1*a***) also play a role in the promotion of hypocotyl growth by warm temperature, though less important than that of PIF4 and PIF7 (106). PIF7 also binds to target gene promoters as a heterodimer with PIF3 (67).

phyB is a photoreceptor and a negative regulator of hypocotyl growth (54, 66); therefore, the *phyB* mutant shows elongated hypocotyl at mild temperatures, and its response to warmth can be stronger than that of the wild type. phyB physically interacts with PIFs, negatively regulating their activity by favoring their phosphorylation and degradation in the proteasome, reducing their intrinsic ability to bind target genes and sequestering them to nuclear condensates (19, 93, 97, 126).

ELF3 is a transcriptional regulator that represses hypocotyl growth. Therefore, the *elf3* mutant shows elongated hypocotyls at mild temperatures (13, 102). ELF3 forms a transcriptional complex that directly represses the expression of the *PIF4* (and *PIF5*) gene (87, 111). In addition, ELF3 also sequesters PIF4 (85) and PIF7 (50) by direct interaction, impeding their binding to target promoters. phyB increases the stability of ELF3 (85), and both are recruited to many common promoter binding sites (30). Warm temperatures have three convergent inputs on this network, negatively regulating phyB and ELF3 and positively regulating PIF7 (see Sections 2–4).

CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1) and SUPPRESSOR OF PHYA-105 1 (SPA1) to SPA4 homologs form the substrate recognition module of a multimeric E3 ligase (99). COP1 increases its nuclear abundance in response to warmth, targeting the transcription factor ELONGATED HYPOCOTYL 5 (HY5) and DELLAs (9, 95) for degradation in the 26S proteasome. HY5 represses *PIF4* gene expression (26) and competes with PIF4 for binding to its targets (36, 117) while DELLAs sequester PIF4, preventing its binding to the target gene promoters (25) (**Figure 1*a***). COP1 also enhances PIF4 protein abundance by posttranscriptional mechanisms (36). In hypocotyl cells, COP1 protects the BRI1-EMS-SUPPRESSOR 1 (BES1) transcription factor from degradation (24); stable BES1 can promote gene expression synergistically with PIF4 (75). phyB represses COP1 activity (99), but, given the quantitatively important role of COP1 in the hypocotyl growth response to warm temperature (26, 27, 36, 84, 95), a more direct temperature input cannot be ruled out. In addition to its role in the COP1/SPA complex, the kinase activity of SPA1 helps stabilize PIF4 to enhance thermomorphogenesis (36, 65). HEAT SHOCK PROTEIN 90 (HSP90; not represented in **Figure 1*a***) enhances the interaction between COP1 and ELF3, releasing PIF4 activity (130).

Another layer of control, with reduced overlap with the control of transcription, is provided by temperature-induced changes in splicing, which in many cases depends on PIF4 providing a link to the thermosensory module (51, 71).

Modeling the vernalization response in *Arabidopsis* requires the inclusion of multiple thermosensory inputs informing about different aspects of the environment (3). This has been interpreted in terms of temperature sensation taking place diffusely throughout the components of the regulatory network, without the involvement of dedicated thermosensors (3). However, the case of hypocotyl growth demonstrates that multiple thermosensors can coexist within a given network (**Figure 1*a***). The information provided by these thermosensors is not fully redundant

(**Figure 1*b***). Due to its gene expression pattern, ELF3 senses temperature mainly during the evening and conveys temperature information into the night because of persistent effects on PIF4 (81). phyB senses temperature under shade and during the night because strong light reduces its temperature-sensing capacity (110) (see Section 2.2.2), and its persistent status conveys night temperature information into the following day (80). PIF7 senses temperature during the photoperiod (21) because its peak of expression occurs during the morning (35, 50, 57, 64) and under shade, which favors its protein stability (16, 69).

phyB, ELF3, and PIF7 affect the expression of genes associated with diverse processes (see Section 5). However, wiring among these network components can change significantly among plant organs (24). Furthermore, phyB, ELF3, and PIF7 do not have an obvious thermosensory function in the roots (11). Thus, the action of the network should not be extrapolated to other organs or processes without specific analysis.

1.6. Overview of the Thermosensory Mechanisms

Each thermosensor has a primary mechanism to decode the temperature stimulus. Of the diverse array of mechanisms described in living organisms, this section focuses on those relevant to established or candidate plant thermosensors.

1.6.1. Temperature can affect the balance between alternative molecular structures. Depending on temperature, proteins can assume structures with different activity. For instance, temperatures within the physiological range do not directly influence the activation of photosensory receptors by photochemical reactions, but they can affect their relaxation or reversion from the active to the inactive conformation, shifting the balance between these variants. Similarly, RNAs can form hairpins that affect their accessibility to the translation machinery; if the relaxation of this structure directly depends on temperature, temperature will affect the synthesis of the encoded protein.

1.6.2. Temperature affects the formation of biomolecular condensates. Many cellular functions are confined within compartments that are not surrounded by membranes. Liquid–liquid phase separation or condensation can drive the formation of these compartments (1). In this process, nucleic acids or proteins, with multiple regions capable of weak interactions, condense into a dense phase that coexists with a dilute phase. This occurs when local concentrations are above a critical value, which directly depends on temperature (1). Within the condensates, molecular components can increase their dwelling time in the vicinity of partners or, alternatively, become sequestered and unable to interact with partners outside of the compartment.

1.6.3. Temperature affects membrane fluidity. Temperature has direct effects on membranes because cooling causes lipids to lose entropy and pack closely, reducing membrane fluidity (76). Therefore, membranes could act as temperature sensors by affecting the activity of proteins able to sense membrane fluidity. Cold temperatures increase the accumulation of desaturated fatty acids, partially counteracting the direct effect on membrane viscosity (76).

2. phyB

2.1. phyB Is a Photosensory Receptor

phyB is a photosensory receptor, and this function affects its thermosensory capacity.

2.1.1. Photochemical reactions. phyB monomers attach a phytochromobilin chromophore to themselves. In tissues that have never received light, phyB is in its inactive red light–absorbing form (Pr) as a Pr-Pr homodimer. Red light transforms Pr into the metastable far-red

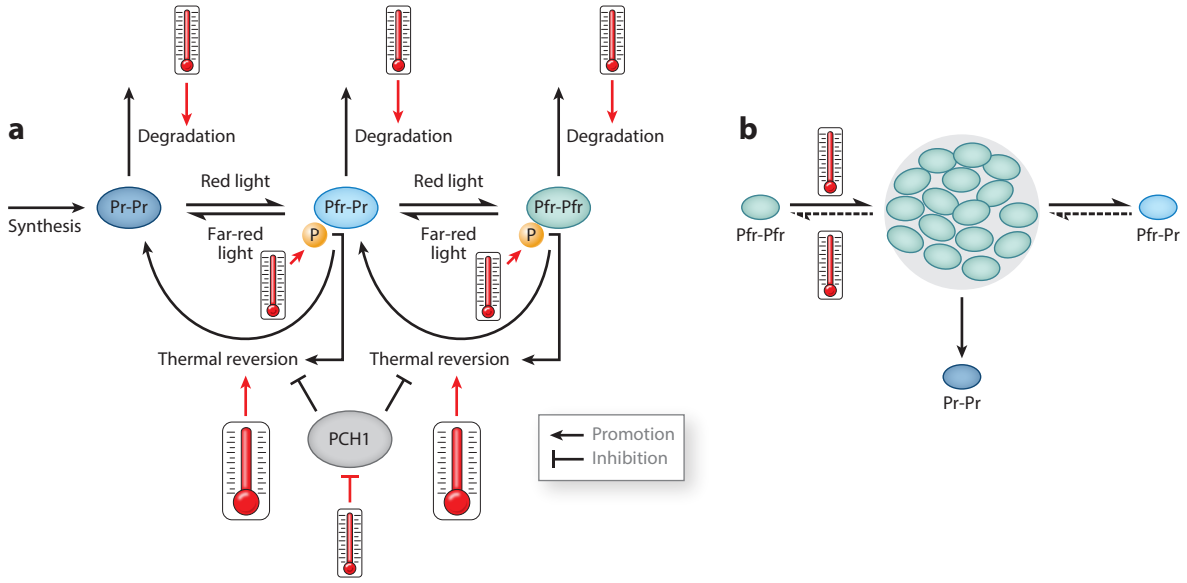


Figure 2

Primary and secondary temperature inputs into phyB dynamics. (*a*) The effect on thermal reversion is the primary temperature sensory mechanism of phyB. Temperature also affects the degree of phyB phosphorylation at S86 and the abundance of PCH1, both of which in turn regulate the rate of dark reversion. Finally, temperature affects phyB degradation while the effects on synthesis could be minor. (*b*) Temperature affects the balance between the rates of phyB assembly to nuclear bodies and the rate of departitioning out of the condensates. In part, this reflects the effects of temperature on Pfr-Pfr (see panel *a*), but the reduction in the size of the nuclear bodies under cold conditions cannot in principle be accounted for in the same way. Abbreviations: PCH1, PHOTOPERIODIC CONTROL OF HYPOCOTYL 1; Pfr, far-red light–absorbing form of phyB; phyB, phytochrome B; Pr, red light-absorbing from of phyB.

light–absorbing form (Pfr), and far-red light transforms Pfr back to the Pr form (15) (**Figure 2*a***). Therefore, the tissues exposed to light bear a mixture of Pr-Pr, Pr-Pfr, and Pfr-Pfr dimers, and only the Pfr-Pfr homodimer is considered biologically active (61). Photosynthetic tissues absorb red light and reflect and transmit far-red light, reducing the red/far-red ratio. Close, large, and green neighbors produce a strong relative enrichment of far-red light, and this environment reduces the abundance of the active Pfr-Pfr homodimer, providing information about the threat imposed by neighbor competitors (17).

2.1.2. phyB undergoes light-dependent liquid–liquid phase separation to form nuclear bodies. phyB is synthesized as Pr in the cytoplasm, and, upon phototransformation, Pfr migrates to the nucleus to exert its biological activity (47, 128). Pfr does not leave the nucleus if it is transformed back to Pr (61). For Pfr-Pfr, assembly to nuclear bodies is faster than disassembly, but assembly is slower than disassembly for Pfr-Pr and negligible for Pr-Pr (61) (**Figure 2*b***). Thus, by establishing Pr-Pr, far-red light causes rapid departition of phyB from the condensates (103). The nuclear bodies include ∼1,500 phyB dimers, proteins that directly interact with phyB (i.e., primary clients), and proteins that interact with phyB direct partners but not with phyB itself (i.e., secondary clients) (6, 19, 58). phyB nuclear bodies are spherical in shape, can rapidly coalesce into larger nuclear bodies, and recover within a few minutes after photobleaching, both in plant and mammalian cells, which are features that support their formation by liquid–liquid phase separation (19). Experiments with phyB synthesized in vitro would be necessary to confirm that the formation of condensates is an intrinsic property of the phyB (19). The C-terminal domain

of phyB provides the driving force for phase separation, modulated by the intrinsically disordered region present at the N-terminal extension of phyB (19).

Given their dependence on Pfr-Pfr, the formation of phyB nuclear bodies correlates with phyB activity (61, 120). Numerous phyB-interacting partners are present in these nuclear bodies (20, 46, 55, 58, 100), and condensation could be a mechanism to increase the residence time (or dwell time), elevating the likelihood of signaling events. However, the specific function of these nuclear bodies remains elusive. The N-terminal half of phyB fused to green fluorescent protein to favor dimerization is able to perform some phyB activities without forming nuclear bodies (77, 91, 94).

2.2. Warm Temperatures Reduce phyB Activity

This section describes the primary mechanism of phyB-mediated temperature sensation, its dependence on light conditions and internal regulations, and its consequences for the dynamics of nuclear bodies.

2.2.1. Temperature-dependent thermal reversion. Each Pfr monomer can spontaneously revert to Pr in a reaction called thermal reversion (60) (**Figure 2*a***). Thus, phyB is able to sense not only the red/far-red ratio but also the fluence rate of red light (61, 118) because thermal reversion competes with photochemical reactions and therefore enhances light dependency. phyB is a temperature sensor because it combines rapid thermal reversion with a rate of thermal reversion that is strongly dependent on temperatures between 10°C and 30°C (54, 66). Therefore, warm temperatures reduce the proportion of the active Pfr-Pfr dimer (**Figure 2*a***). Several residues at the core of the photosensory module are important determinants of phyB thermal reversion because the chromophore is highly sensitive to local changes, and the key biophysical parameters of phyB are conserved in monocots and eudicots (14). The rate of thermal reversion is faster from the Pfr-Pr to the Pr-Pr dimers than from the Pfr-Pfr to the Pfr-Pr dimers (61).

2.2.2. The ability of phyB to sense temperature depends on light conditions. If a tissue is in full darkness and there is no Pfr, thermal reversion cannot operate and phyB cannot sense temperature. However, at the other extreme, high irradiance renders phyB thermosensory capacity negligible because under these conditions photochemical reactions become very fast and thermal reversion is unable to compete (**Figure 2**). During a clear summer midday, full sunlight reaching plant tissues can be >2,000 μmol m^{-2} s^{-1} (400–700 nm). For full-length phyB in vitro, achieving near-saturating Pfr levels requires 1,250 μmol m^{-2} s^{-1} of monochromatic red light or 4,400 μmol m^{-2} s^{-1} of white light (14). In vivo, thermal reversion is slower (see Section 2.2.3) and the temperature dependency of phyB activity is estimated to be significant below approximately 300 μmol m^{-2} s^{-1} sunlight, that is, under canopy shade, on cloudy days, at the extremes of the natural photoperiod (110), and during the night (**Figure 1*b***). Therefore, warm temperatures reinforce the effect of shade (105) because shade reduces red light, increasing the impact of thermal reversion (**Figure 2**). During the night, thermal reversion dominates phyB dynamics because phototransformations are absent, but phyB also senses temperature during the day (66, 100). Maximum absolute rates of thermal reversion occur during the day because at night thermal reversion is limited by its slowest step (Pfr-Pfr to Pfr-Pr), as the fast step (Pfr-Pr to Pr-Pr) rapidly depletes the Pfr-Pr pool (**Figure 2*a***). During the day, the Pfr-Pr pool is fed by not only thermal reversion from Pfr-Pfr but also the phototransformation of Pr-Pr, maintaining higher levels to initiate the fast step of thermal reversion.

2.2.3. Thermal reversion is a regulated process. The rate of thermal reversion depends on the developmental context and is reduced by prolonged exposure to light (103). PHOTOPERIODIC CONTROL OF HYPOCOTYL 1 (PCH1) and PCH1-LIKE (PCHL) are proteins that interact physically with phyB and reduce the rate of thermal reversion (29, 44)

(**Figure 2*a***). Furthermore, phyB has a distinctive N-terminal extension that reduces the rate of thermal reversion but does not affect its temperature dependence (14). Several amino acids of this N-terminal extension are phosphorylated (78, 86, 121), and phosphorylation of S86 is particularly relevant because it causes a 50-fold enhancement of the in vivo thermal reversion rate of the Pfr-Pr heterodimers and a twofold increase of that of the Pfr-Pfr homodimers (121) (**Figure 2*a***). The expression of *PCH1* and *PCHL* increases in response to light (29), while phosphorylation of S86 decreases (121), and these changes at least partially account for the light enhancement of Pfr stability. PCH1 and PCHL are components of phyB nuclear bodies (44, 58), which might explain the correlation between nuclear bodies and phyB Pfr stability, particularly during the night (120).

2.2.4. phyB nuclear bodies are temperature dependent. The size of phyB nuclear bodies depends on not only light but also temperature conditions (66) (**Figure 2*b***). Warm temperatures, in contrast to mild temperatures, accelerate thermal reversion, reducing Pfr-Pfr, and, since nuclear body formation depends on Pfr-Pfr, the size of phyB nuclear bodies decays with warm temperatures (66). Phosphorylation of S86 accelerates thermal reversion and reduces nuclear body formation (78). Conversely, PCH1 and PCHL reduce phyB thermal reversion and enhance the formation of phyB nuclear bodies (29, 44, 45). phyB molecules mutated close to the site of attachment of the chromophore show reduced thermal reversion and large nuclear bodies at warm temperatures (66). Therefore, there is a clear correlation between these two processes. Soon after temperature transitions, warmer conditions reduce the size of phyB nuclear bodies, but the opposite occurs under prolonged temperature treatments likely because under these conditions warmer temperatures increase phyB protein abundance (40). The dynamics induced by temperature shows differences with that induced by shade (40, 118). For instance, a set of nuclear bodies associated with the nucleolus is stable under warm temperatures but disassembles under shade (40). Temperature stability could be caused by a higher proportion of Pfr-Pfr in certain nuclear bodies because thermal reversion proceeds more slowly from this homodimer (40). Temperature dependency of phyB nuclear body formation requires the N-terminal photosensory domain (40) and, more specifically, the intrinsically disordered region of the phyB N-terminal extension (19).

2.3. Multiple Temperature Inputs Modify phyB Activity

Temperature sensation by phyB occurs through its direct effects on the rate of Pfr to Pr thermal reversion, which can be demonstrated with phyB synthesized in vitro (14, 66). However, in vivo, temperature has additional effects on phyB activity.

2.3.1. Temperature has indirect effects on thermal reversion. Warm temperatures decrease the abundance of PCH1 by transcriptional and posttranscriptional mechanisms (80) and increase the phosphorylation of S86 (121). Thus, warmth reduces the activity of phyB by accelerating thermal reversion directly as well as indirectly by enhancing the phosphorylation of S86 and lowering the abundance of PCH1 (**Figure 2*b***).

2.3.2. Temperature modifies phyB nuclear bodies beyond its effects on thermal reversion. Despite slowing down thermal reversion and hence increasing Pfr-Pfr, low temperatures can reduce the size of phyB nuclear bodies (66). The maximum size of these condensates can be observed at intermediate temperatures, but this optimum depends on light conditions and the duration of the exposure to the different temperatures (40, 66, 80). Thus, in addition to affecting the rate of thermal reversion and thereby the abundance of Pfr-Pfr homodimers, temperature affects the balance between Pfr-Pfr assembly to and disassembly from the nuclear bodies. If nuclear body formation becomes limiting for phyB activity at low temperatures, phyB would sense

temperature primarily by modulating thermal reversion and secondarily by modulating nuclear body formation. However, this remains to be tested. Chen et al. (19) have argued that since phyB Pfr is more stable in the nuclear bodies, warm temperatures would directly disassemble the nuclear bodies, rendering phyB Pfr more susceptible to thermal reversion. According to this view, the primary temperature-sensing mechanism would relate to nuclear body dynamics and not to thermal reversion. The idea is based on the observation that a constitutively active phyB (YHB) unable to phototransform or thermally revert (112) forms condensates that decrease their size in response to warm temperatures when expressed in mammalian cells or in planta (19). However, the latter appears to contradict detailed observations by Hahm et al. (40), showing that YHB-YFP forms thermostable nuclear bodies in *Arabidopsis*. Furthermore, since YHB does not form Pfr, the dynamics of YHB nuclear bodies does not necessarily reflect that of native Pfr condensates (40, 112).

2.3.3. phyB protein turnover stores temperature information. Temperature has no large, consistent effects on *PHYB* transcript levels, but higher temperatures can either increase (40, 80) or decrease (49, 65) phyB protein abundance by posttranscriptional mechanisms. The balance between such opposite effects depends on the context, but systematic analysis of this dependency is still lacking. phyB undergoes degradation in the 26S proteasome, preferentially in the Pfr form (83). Increased phyB abundance under warm temperatures could be accounted for by its enhancement of thermal reversion to Pr, which is more stable (40, 80). Consistently with this interpretation, warm temperatures, in contrast to low temperatures, increase phyB nuclear abundance and total protein abundance during the night if the seedlings initiate the dark period with high Pfr levels (i.e., when thermal reversion is possible) but not if they receive a pulse of far-red light at the end of the day (leaving negligible amounts of Pfr for thermal reversion) (80). Furthermore, the *pch1* mutant background, which destabilises phyB Pfr, also eliminates this effect of temperature on phyB abundance (80). When the seedlings receive the far-red light pulse at the end of the day, warm night temperatures reduce phyB nuclear and total abundance, likely by increasing the rate of Pr degradation. These changes in phyB abundance carry night temperature information to the mechanisms controlling growth during the following day (**Figure 1*b***). Reduced phyB abundance with increasing temperatures could result from negative regulation of PIF-induced degradation (49, 65). For instance, in response to cold temperatures, C-REPEAT BINDING FACTORs (CBFs) interact with PIF3, attenuating PIF3-induced degradation of phyB (49).

3. ELF3

3.1. Warm Temperatures Reduce the Binding of the Evening Complex to Its Target Genes

ELF3 together with ELF4 and the MYB domain transcription factor LUX ARRYTHMO (LUX) constitute the Evening Complex. The Evening Complex is a component of the circadian clock involved in the repression of numerous genes, including *PIF4* and *PIF5* early in the evening, playing a fundamental role in rhythmic hypocotyl growth (30, 87). Warm temperatures increase *PIF4* gene expression and hypocotyl growth by reducing the inhibitory effect of ELF3 on both of these variables (13, 79). Warmth reduces the binding of the Evening Complex to its target promoters in vivo (13, 30) and in electrophoretic mobility shift assays (111), demonstrating that temperature sensing involves no other protein beyond the Evening Complex components themselves. Of the three proteins, LUX is the only one able to bind target DNA sequences through its MYB domain. The interaction with ELF3 impairs LUX binding capacity, and interaction of ELF4 with ELF3 restores binding (111). However, LUX binding is not temperature sensitive (111), and in vivo control of gene expression requires the three components of the complex (30). As noted above,

ELF3 physically interacts with the PIF4 and PIF7 proteins, preventing their binding to target gene promoters, in an action that is independent of the Evening Complex (50, 85). However, it is not clear whether temperature affects this interaction.

3.2. The Thermal Responsiveness of ELF3 Depends on Its Prion-Like Domain, Which Undergoes Liquid–Liquid Phase Separation In Vitro

A polyglutamine (polyQ) repeat is present at the C terminus of *Arabidopsis* ELF3, which shows substantial variation among accessions (115). Complementing the *elf3* mutant with ELF3 proteins containing longer polyQ repeats moderately increases the hypocotyl growth response to temperature, while replacing the predicted prion-like domain where the polyQs are embedded impairs the thermal responsivity of hypocotyl growth, target gene expression, and ELF3 association to target promoters (53). Intriguingly, ELF3 with a given polyQ length behaves differently in different accessions (119).

Expression of the ELF3 prion-like domain in vitro gives a dilute phase of uniform polymers, which forms liquid droplets in response to higher temperatures (48, 53). These droplets are highly mobile in solution and can fuse, indicating the intrinsic capacity of the ELF3 prion-like domain to undergo liquid–liquid phase separation. A prion-like domain where the polyQ repeat has been deleted is still able to phase separate in vitro, but increasing the number of polyQs enhances the sensitivity of phase separation to temperature (48). The liquid condensed state ages into a hydrogel, which is likely to form in vivo, because the condensates do not exhibit full fluorescence recovery after photobleaching (48).

3.3. ELF3 Forms Temperature-Dependent Biomolecular Condensates

ELF3 forms nuclear condensates in *Arabidopsis* (42). In yeast, the formation of condensates increases with temperature (53). However, in hypocotyl cells of *Arabidopsis*, warm temperatures can either enhance or reduce the formation of nuclear bodies containing ELF3 (81, 107). The pattern of nuclear body formation is dynamic in hypocotyl cells, and warm temperatures have a small inhibitory effect during the morning, compared to cold temperatures, but strongly promote the formation of nuclear bodies in the afternoon (81). In root cells, where ELF3 does not appear to work as a thermosensor, warm temperatures reduce ELF3 nuclear bodies (11, 107). Bear in mind that ELF3 is predicted to interact with many proteins (43), and, in different contexts (tissues, times of day, temperature histories, etc.), ELF3-containing condensates could have different compositions, affecting their response to temperature. Therefore, scaling up from the temperature-dependent in vitro phase separation of the ELF3 prion-like domain to the dynamics of ELF3 nuclear bodies in plant cells is not simple.

3.4. Biomolecular Condensates and ELF3 Activity

Since both the reduction of ELF3 activity by warm temperatures in *Arabidopsis* and the formation of ELF3 condensates in root cells and yeast cells require the prion-like domain (53), a linear extrapolation would predict that ELF3 nuclear bodies in planta represent the inactive state of ELF3 and increase in size and/or number with warmer temperatures. Perfectly in line with the latter expectation, in hypocotyl cells during the afternoon, nuclear body formation strongly correlates with reduced ELF3 repression of *PIF4* expression (81). However, ELF3 from the accession Shahdara shows impaired formation of nuclear bodies and enhanced expression of genes repressed by ELF3, suggesting a positive correlation between nuclear body formation and ELF3 activity (4).

3.5. ELF3 Condensates Store Information About Warm Conditions

The proteins that oligomerize to form condensates and establish long-lasting signaling changes, encoding a memory of previous conditions, are called mnemons (104). ELF3 provides a memory of warm daytime temperatures into the night (81) (**Figure 1*b***) because the dynamics of the nuclear bodies shows hysteresis; that is, the formation of nuclear bodies is more sensitive to a rise in temperature than their disassembly is to a reduction in temperature. Therefore, when temperature drops at night, ELF3 remains in the inactive nuclear bodies (81).

4. PIF7

4.1. The *PIF7* Transcript Senses Temperature and Modulates Its Rate of Translation

Warm temperatures rapidly increase PIF7 protein levels (<90 min after the transfer to 28°C) (32). The 5′-untranslated region of the *PIF7* messenger RNA contains a hairpin located approximately 30 nucleotides upstream from the start AUG codon (21). Temperatures above 22°C apparently cause partial unfolding of the hairpin and a more relaxed conformation (**Figure 3**). This alternative conformation is stable up to 32°C and selectively enhances the translation of the *PIF7* transcript, leading to increased protein synthesis (21). In addition to its action through this primary sensing mechanism controlling translation, temperature affects *PIF7* gene expression. Furthermore, temperature effects on PIF7 interaction partners phyB and ELF3 likely control PIF7 protein stability and the capacity to bind target gene promoters (**Figure 3**).

4.2. Temperature Also Affects PIF7 via Its Interaction with phyB

Although temperature sensing occurs though the *PIF7* transcript, temperature has additional effects on the PIF7 protein, some of which are mediated by phyB.

4.2.1. PIF binds phyB. PIF7 binds through its N terminus specifically to the active conformer of phyB but not to any of the other phytochromes (68). phyB reduces the activity of PIF7, and low red/far-red ratios release PIF7 from this inhibition, allowing PIF7 to contribute to the growth response to cues of neighbors (69). Since warm temperatures also reduce phyB activity (see

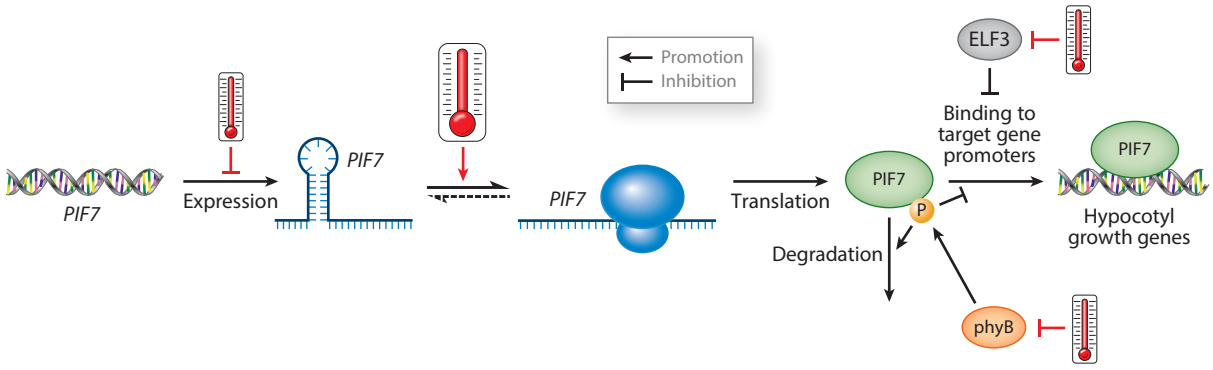


Figure 3

Primary and secondary temperature inputs into PIF7 dynamics. The effect on *PIF7* transcript structure is the primary temperature sensory mechanism of *PIF7*. Temperature also affects the expression of the *PIF7* gene. phyB affects PIF7 degradation, and both phyB and ELF3 affect the binding of PIF7 to its target gene promoters. Since phyB and ELF3 are thermosensors, they could provide another temperature input to the control of PIF7. Abbreviations: ELF3, EARLY FLOWERING 3; P, phosphate; phyB, phytochrome B; PIF7, PHYTOCHROME-INTERACTING FACTOR 7.

Section 2), the PIF7 responses to red/far-red ratios are hypothesized to take place under warm temperatures (**Figure 3**). In the sections that follow, we will consider the effects of red/far-red ratios. In most cases, the significance of phyB regulation of PIF7 activity in response to warm conditions remains to be tested.

4.2.2. phyB induces the phosphorylation of PIF7. PIF7 is phosphorylated throughout a long-day cycle or under continuous white light but becomes gradually dephosphorylated during the night of short days (50, 67, 69, 123). A transfer to a low red/far-red ratio rapidly (10 min) and persistently decreases the amount of phosphorylated PIF7 while increasing the accumulation of dephosphorylated PIF7—a shift that is reversed rapidly (<15 min) when seedlings were returned to a high red/far-red ratio (50, 69, 123). In addition to increasing PIF7 protein levels, warm temperatures increase the relative abundance of the faster-migrating isoform, presumably dephosphorylated PIF7 (32). Following dephosphorylation, PIF7 binds to its target sites (123).

4.2.3. PIF7 is present in nuclear bodies containing phyB. PIF7 is a constitutively nuclear protein with a diffuse distribution in dark-grown seedlings, which rapidly (<2 min) and persistently forms nuclear bodies upon exposure to red or white light (57, 68). PIF7 and phyB colocalize in the nuclear bodies (57, 68, 123, 126). Transfer to light does not induce PIF7 nuclear bodies in the *phyB* mutant, and, in the presence of phyB, the shift from a high to low red/far-red ratio rapidly (<30 min) disperses PIF7 to the nucleoplasm (123). The presence of PIF7 in nuclear bodies containing phyB would reduce its capacity to bind to the target gene promoters (126). Early studies have not revealed changes in PIF7 subnuclear distribution in response to cold temperatures (57), but a more detailed analysis involving a wider temperature range is warranted.

4.2.4. PIF7 is present in different nuclear bodies upon reduction of active phyB. PIF7 bears a nuclear localization signal and a Q-rich motif at its extreme C terminus, which is absent in PIF3 and PIF4 (68). Upon release from phyB nuclear bodies under a low red/far-red ratio, PIF7 forms smaller nuclear bodies thanks to its own capacity to undergo liquid phase separation, likely related to the intrinsically disordered region present at the C terminus (126). The functional significance of these PIF7 nuclear bodies remains untested.

4.2.5. phyB affects PIF7 stability. The levels of PIF7 protein oscillate during the day. Under long days, PIF7 peaks in the morning, leading to relatively low levels during the night, that is, broadly matching the changes in messenger RNA (mRNA) levels (35, 123). However, under short days, PIF7 abundance peaks at night (50). Although it is more stable in the presence of active phyB than other PIFs, PIF7 is still targeted to degradation in the 26S proteasome. Dephosphorylation of PIF7, induced by low red/far-red treatments that reduce phyB activity, enhances PIF7 stability (35, 69). Two ubiquitin-specific proteases, UBP12 and UBP13, directly deubiquitinate PIF7 and mediate this response, (132) and treatment with a proteasome inhibitor moderately increases PIF7 protein levels (21).

4.3. Temperature Affects *PIF7* Transcript Levels

The expression of *PIF7* increases under long days compared to short days (64), but the peak occurs in the morning in both cases (35, 50, 57, 64). In contrast to that of *PIF4*, the expression of *PIF7* is not significantly affected by ELF3 (50). Warm temperatures can decrease *PIF7* transcript abundance, but this depends on growth conditions (16, 32). The temperature sensor behind this response has not been identified. Despite this uncertainty, temperature effects on *PIF7* transcript abundance condition the impact of temperature effects on *PIF7* translation. After plant exposure to several days of warmer temperatures during the central portion of the photoperiod and cooler temperatures during the rest of the day (which is quite common in natural settings), the effects

of temperature on the steady-state levels of the *PIF7* transcript become reduced and the resulting increase in protein levels is more obvious (21). Conversely, when seedlings grown at a constant temperature (e.g., 17°C) are shifted to warmer conditions (27°C), reduced *PIF7* transcript levels partially counteract enhanced translation (21).

5. TWA1

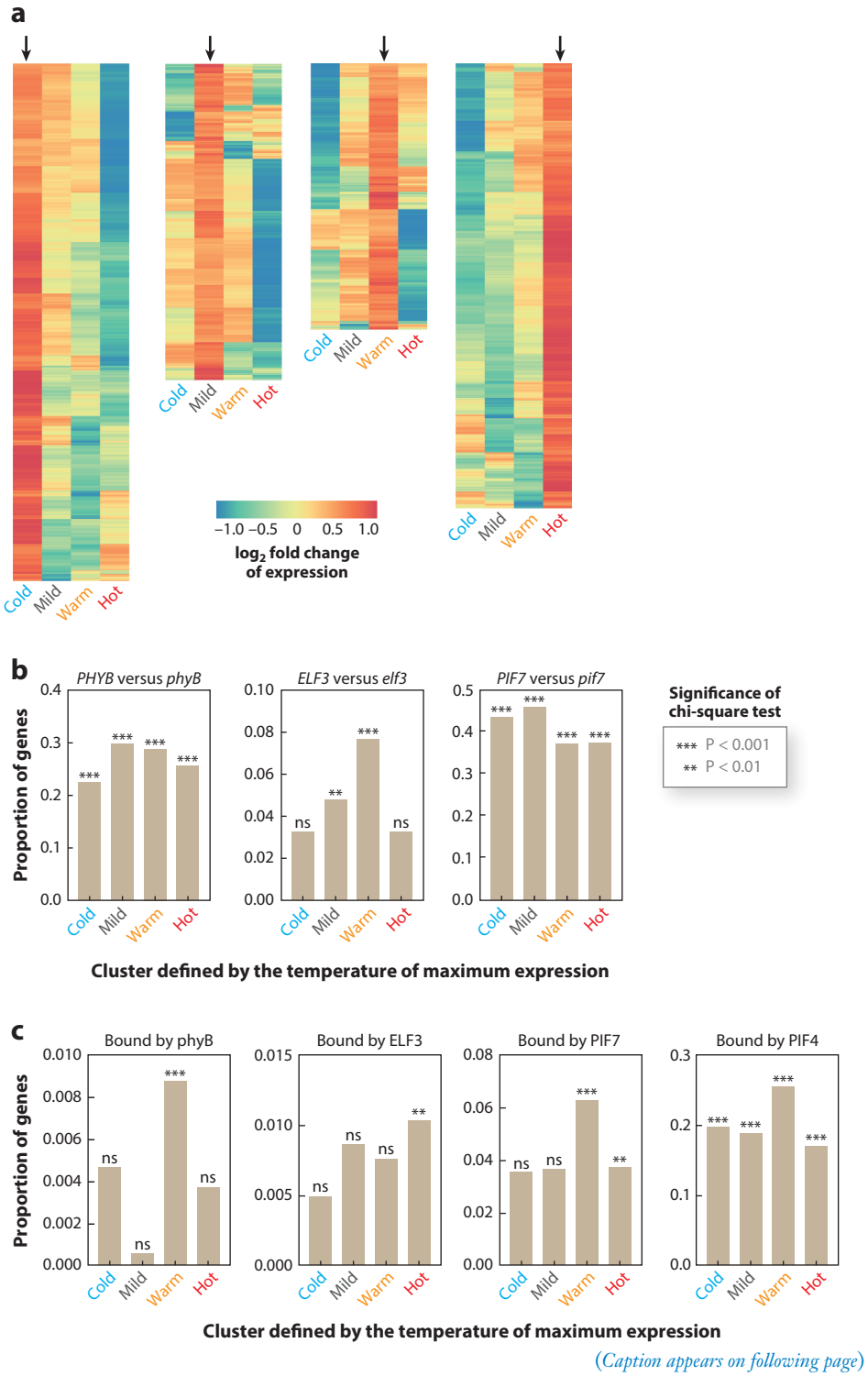
Plant species from cold or warm environments tend to have higher tolerance to extreme low or high temperatures, respectively (72). Basal tolerance is important when plants experience a sudden exposure to stressful temperatures. Acquired tolerance comes into play when plants experience a gradual transition from mild to extreme temperatures, which allows acclimation (38). Proper synthesis, perception, and signaling of the stress hormone abscisic acid (ABA) are crucial for basal and acquired heat tolerance (10). TWA1 was recently identified in a screening of mutants hypersensitive to ABA and impaired basal and acquired heat tolerance (10).

Increasing the temperature between 20°C and 30°C moves apart the terminal domains of TWA1 without significantly affecting its abundance (10). A stretch of 20 amino acids within the TWA1 N-terminal region is necessary for thermosensing and shows high sequence variability between the *Arabidopsis thaliana* and *Arabidopsis lyrata* orthologs, which explains the differences in sensitivity between these two species (10). As a result of the warmth-induced intramolecular rearrangement, the C-terminal region of TWA1 binds to the JASMONATE-ASSOCIATED MYC2–LIKE 2 (JAM2) transcription factor, and the N-terminal region interacts with TOPLESS (TPL) transcriptional repressors through two TWA1 ethylene-responsive element binding factor–associated amphiphilic repression (EAR) motifs (10). TPL-TWA1-JAM2 complex formation occurs in subnuclear domains, repressing ABA-responsive gene expression to allow optimal regulation of the heat stress response (10) through the master pathway initiated by HEAT SHOCK FACTOR 1 (HSF1) transcription factors (89).

6. THERMOSENSORS AND TRANSCRIPTOME RESPONSES TO TEMPERATURE

6.1. Overview

Figure 4*a* offers an overview of the impact of temperature on the transcriptome, based on the meta-analysis of data published for *Arabidopsis* seedlings grown at mild temperatures and transferred to cold, warm, or hot temperatures (8). Almost 11,000 genes showed robust responses to temperature, emerging above the variability introduced by the different experimental conditions incorporated into the analysis. The clusters with more genes are those showing maximum expression at extreme temperatures. In these cases, the minimum is not necessarily at the opposite end of the temperature scale, demonstrating that some genes tend to increase their expression at both extreme temperatures (although not symmetrically). Among the gene ontology (GO) terms, "ribosome biogenesis" and "cold acclimation" are overrepresented among the genes with the highest expression at cold temperatures, "photosynthesis" and "cell cycle" at mild temperatures, "vesicle-mediated transport" and "DNA modification" at warm temperatures, and "protein maturation" and "mRNA metabolic process" at hot temperatures (see **Supplemental Table 1**). Temperature effects on plants result from not only temperature sensation but also other consequences of the temperature environment. For instance, the promotion of root hair growth by low temperatures is considered the consequence of reduced nitrate diffusion and the resulting perception of low nitrate by the root (90). Furthermore, cells can respond to the damage caused by heat (82), and growth reduction by cold can increase the concentration of signaling molecules (131). It must be noted



(*Caption appears on following page*)

Figure 4 (*Figure appears on preceding page*)

Temperature profoundly affects the transcriptome of *Arabidopsis thaliana*. (*a*) Meta-analysis of the transcriptome in seedlings grown at mild temperatures and transferred to cold, warm, or hot temperatures. Differentially expressed genes are grouped according to the temperature of maximum expression (marked by the arrows). For further details of the analysis and overrepresented GO terms, see **Supplemental Table 1**. (*b*) Proportion of genes of each cluster with expression affected by the *phyB* (124) or *elf3* (30) mutations at mild temperatures or by the *pif7* (69) mutation at warm temperatures compared to the wild type [counts were downloaded and aligned using Galaxy Europe (**https://usegalaxy.eu/**) and analyzed with DESeq2 in R software (73)]. (*c*) Proportion of genes of each cluster bound by phyB (54), ELF3 (13), PIF7 (21), and PIF4 (88) in chromatin immunoprecipitation sequencing experiments. Asterisks indicate the significance of the chi-square test (Yates correction) comparing each cluster to the genome. Abbreviations: ELF3, EARLY FLOWERING 3; GO, gene ontology; ns, not significant; phyB, phytochrome B; PIF, PHYTOCHROME-INTERACTING FACTOR.

that the relatively short duration of the experiments under the controlled conditions included in the meta-analysis minimizes but does not exclude these collateral effects of temperature.

6.2. Temperature Effects Involving phyB, ELF3, and/or PIF7

The contribution of known thermosensors to transcriptome responses to a wide range of temperatures has not been investigated systematically. To tentatively explore this issue, we calculated the proportion of genes of each cluster with expression affected by the *phyB* or *elf3* mutations at mild temperatures or by the *pif7* mutation at warm temperatures (**Figure 4*b***) and the proportion of genes of each cluster bound by phyB, ELF3, PIF7, or PIF4 in chromatin immunoprecipitation sequencing experiments (**Figure 4*c***). Similar information is not available for TWA1 yet. The results suggest potential links between a large proportion of the temperature-responsive genes and phyB, ELF3, and/or PIF7, but they are not necessarily acting as thermosensors (11).

6.3. phyB and PIF7 Beyond Warmth

Since phyB and PIF7 were identified as thermosensors by using the hypocotyl growth response, a process that reaches a maximum at warm temperatures, it was unexpected that the genes with expression controlled by these thermosensors spread evenly through the four clusters (**Figure 4*b***). However, phyB and PIF7 can affect plant responses to cold or heat. Depending on growth conditions, phyB can enhance (49, 59) or decrease (64) freezing tolerance. PIF4 and PIF7 negatively regulate the expression of *CBF* genes under mild temperatures and repress freezing tolerance (49, 57, 64). Of note, the GO phrase "response to red or far-red light" is overrepresented among the genes with maximum expression in response to cold (see **Supplemental Table 1**). The *phyB* mutant is constitutively tolerant to heat stress due to its reduced expression of several *FATTY ACID DESATURASE* (*FAD*) genes and a lower proportion of fully unsaturated fatty acids (11). The possibility that phyB senses warm temperatures causing acclimation to heat cannot be ruled out because the phenotype of the *phyB* mutant and the effect of warm temperatures, which reduce phyB activity, both lead to this outcome (5). COP1 facilitates nuclear accumulation of HEAT SHOCK TRANSCRIPTION FACTOR A1D (HSFA1d) (116), and BES1 interacts with HSFA1s to promote heat tolerance (2), providing additional links of the thermosensory module (**Figure 1*a***) with heat responses.

7. SEARCHING FOR ADDITIONAL THERMOSENSORS

7.1. Other Plant Thermosensors Are Likely to Exist

There are two reasons to suspect the existence of additional thermosensors. First, there are sensory responses to temperature, such as primary root growth or root branching, in which none of

the known thermosensors have an obvious function as sensors (11). Second, there are other photoreceptors, proteins that form biomolecular condensates, RNAs that form hairpins, and proteins that interact with membranes that participate in plant responses to temperature.

7.2. Other Photosensory Receptors

In addition to phyB, other photoreceptors could serve as temperature sensors.

7.2.1. Phytochromes. There are five *PHY* genes (*PHYA* to *PHYE*) in *Arabidopsis* that show strong sequence conservation (22). In the *phyB* mutant, the hypocotyl growth response to temperature is steeper than in the wild type, which is consistent with phyB being a repressor of the effect of warm temperatures (54, 66). The quintuple *phy* mutant displays a constitutive warm temperature response (54). The lack of temperature responses despite the presence of ELF3 and PIF7 could indicate collateral effects of poor light signaling rather than a thermosensory role of other phytochromes. In fact, the high rates of thermal reversion that make phyB a suitable thermosensor are not present in other phytochromes (14). While the photosensory module of phyB shows 93% reversion to Pr in vitro in 3 h, those of phyA and phyC show 7% and 17% reversion after 8 h, respectively, and the photosensory module of phyE remains stable over multiple days at 25°C (14). phyE also shows negligible thermal reversion in vivo, and its physiological activity shows poor fluence rate dependency, consistent with the lack of thermal reversion that would enhance the need for light to compensate for the decay of Pfr (121). Therefore, although phyA, phyC, and phyE photosensory modules display strong temperature dependence, their basic rates of thermal reversion appear to be too slow to confer thermosensor capabilities comparable to those of phyB (14). However, phyA has a physiological role under very low fluences of light (12), where slow thermal reversion could eventually have implications, and in some species phyA temperature-dependent reversion would not be that slow (109). Furthermore, for technical reasons, it has not been possible to measure the rate of phyD thermal reversion in vitro or in vivo (14, 121), but the physiological activity of phyD shows strong fluence rate dependency and decreases with warm temperatures and with phosphorylation at residues placed at the N-terminal extension (121), which are key features of phyB as a thermosensor. Finally, it would also be interesting to investigate if the formation of nuclear bodies responds to temperature in other phytochromes and eventually confers thermosensory capacity to them.

7.2.2. The UV-B photoreceptor. UV RESISTANCE LOCUS 8 (UVR8) is a dimeric photoreceptor that upon ultraviolet B (UV-B) excitation shifts to the monomer conformation, interacts with downstream signaling partners, and slowly redimerizes (98). Under natural radiation, warm temperatures decrease the proportion of UVR8 present as a monomer due to faster reversion to the dimer state (31), suggesting that UVR8 could contribute to temperature sensation.

7.2.3. Phototropins. The blue light photoreceptor phototropin that controls chloroplast intracellular position is also a temperature sensor in the liverwort *Marchantia polymorpha* (34). Low temperatures extend the lifetime of light-activated phototropin, favoring chloroplast location to the periphery of the cell to avoid damage when the biochemical reactions of photosynthesis are too slow to deal with the products of light excitation (34). Phototropins could be temperature sensors in seed plants.

7.3. Biomolecular Condensates

The *Arabidopsis* scaffold protein FRIGIDA (FRI) integrates a complex that deposits histone H3 methylation marks that promote the expression of the flowering repressor *FLOWERING LOCUS C* (*FLC*) (70). Strong, functional *FRI* and *FLC* alleles prevent *Arabidopsis* accessions from northern

latitudes from flowering during severe winters (52). To allow flowering in spring, the repression is dismantled by vernalization during winter (63). FRI forms nuclear condensates that increase their size and number during cold exposure and never colocalize with nascent *FLC* transcripts, suggesting that their function is to prevent FRI from promoting the expression of *FLC* during winter (134). In addition to the C-terminal disordered domain and two coiled-coil domains of FRI, the formation of these condensates requires other proteins and long noncoding RNA, which could increase FRI stability (134). At the other end of the temperature scale, RNA-BINDING GLYCINE-RICH PROTEIN D2 (RBGD2) and RBGD4 undergo liquid–liquid phase separation in vitro and condense into stress granules in response to heat, improving the tolerance to this stress (135).

7.4. Hairpins

Hairpin sequences similar to that observed in the *PIF7* transcript are present in other transcripts important for temperature responses such as *HEAT SHOCK TRANSCRIPTION FACTOR A2* (*HSFA2*) and *WRKY22* (21).

7.5. Cellular Membranes

Rice (*Oryza sativa*) plants are sensitive to cold, and several membrane-associated proteins confer enhanced cold tolerance. For instance, *COLD1* and *CHILLING TOLERANCE IN GENGDAO/JAPONICA RICE 1* (*COG1*) were identified as major genes of quantitative trait loci (QTLs) that positively modulate chilling tolerance in japonica rice (74, 125). COLD1 localises on plasma membrane and endoplasmic reticulum and interacts with the G-protein α subunit, accelerating its GTPase activity and stimulating the influx of Ca^{2+} in response to cold temperature (74). Low temperatures increase the interaction between the membrane-localized COG1 leucine-rich repeat receptor-like protein and its interacting partner, SOMATIC EMBRYOGENESIS RECEPTOR KINASE-LIKE 2 (SERL2) (125). An additional example is provided by CALRETICULIN 3 (CRT3), a Ca^{2+}-binding protein localized to the endoplasmic reticulum, required for cytoplasmic elevation of Ca^{2+} concentration in response to cold and chilling tolerance, which interacts with CALRETICULIN B–LIKE PROTEIN KINASE 7 (CIPK7), enhancing its kinase activity (39). These could be cases of responses triggered by changes in membrane fluidity. However, in response to cold treatments in vitro, CRT3 undergoes changes in secondary structure and increases the interaction with CIPK7 (39), suggesting that CRT3 could by itself be involved in cold sensing. Similarly, MITOGEN-ACTIVATED PROTEIN KINASE KINASE KINASE KINASE 4 (MAP4K4)/TARGET OF TEMPERATURE 3 (TOT3) is a plasma membrane–localized protein required for warm temperature–induced hypocotyl elongation in *Arabidopsis* and leaf sheath elongation in wheat (119).

In *Arabidopsis*, under long days, FLOWERING LOCUS T (FT) is synthesized in phloem companion cells and moves through the plasmodesmata to the sieve elements to be transported toward the apex and trigger flowering (23). At 16°C, FT interacts with negatively charged phospholipids, preferentially phosphatidylglycerol, of the membranes of companion cells and becomes sequestered there (114). Retention in companion cells is reduced at 23°C, plausibly due to changes in phosphatidylglycerol saturation, allowing FT to reach the sieve tubes, move to the apex, and induce flowering (114).

8. CONCLUSIONS

phyB, ELF3, and PIF3 are thermosensory components of a signaling network controlling gene expression by transcriptional and posttranscriptional mechanisms. Despite some redundancy, they

input complementary information to the network. This network participates in transcriptomic responses to temperature that vary in their patterns and functions, but apparently not always in a thermosensory capacity. TWA1 has been identified as a temperature sensor and transcriptional regulator for thermotolerance, but a role in thermomorphogenesis cannot be excluded with current information.

Specific temperature-sensing mechanisms include changes in protein or transcript conformation and in the dynamics of biomolecular condensates. In addition to impacting the core sensing feature, temperature affects other aspects of the sensor dynamics as a consequence of (*a*) the modifications driven by the primary temperature-sensing mechanism (e.g., phyB Pfr thermally reverts to Pr, which is less susceptible to proteolytic degradation), (*b*) the action of another known or unidentified thermosensor (e.g., phyB phosphorylates PIF7), or (*c*) a second, apparently direct action of temperature (e.g., temperature affects the levels of Pfr-Pfr but also the rates of Pfr-Pfr assembly/disassembly to/from phyB nuclear condensates).

Given the profound and diverse effects of temperature on plants, many additional thermosensors are likely awaiting identification. Critical areas for future investigation include (*a*) the search for further thermosensors and potentially novel sensing mechanisms and (*b*) a more precise understanding of the links between in vitro phase separation, in vivo condensate dynamics, and thermosensor activity.

DISCLOSURE STATEMENT

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