CellPress

Spotlight

Thermosensing Enlightened

Carolin Delker,[1] Martijn van Zanten,[2] and Marcel Quint[1,*]

Understanding the molecular networks driving plant responses to high ambient temperatures is crucial for developing crop cultivars resistant to global warming. Although several factors involved in temperature signalling are known, a thermosensing mechanism had remained elusive. However, two recent publications demonstrate that the photoreceptor phytochrome B (phyB) also acts as a thermosensor.

Thermomorphogenesis

Increasing temperature typically induces the elongation growth of organs, including hypocotyls, stems, and petioles. As a result, plants display an open shoot architecture, which effectively enhances leaf-cooling capacity [1,2]. These thermomorphogenic adaptations phenocopy the shade avoidance response, an elongation growth-dependent escape mechanism exhibited by plants to outcompete neighbours in canopies (Figure 1A). Shade avoidance is controlled by a sophisticated array of photoreceptors to accurately sense, and respond to, changes in the light spectrum [3]. When plants are shaded by foliage, the blue and red light intensities decrease, because these wavelengths are absorbed by photosynthetic pigments, while the far-red component remains unaltered. Members of the chromophore-containing phytochrome photoreceptor family (phyA–E) are sensitive to red and far-red light. They display partial functional specificities with regard to the regulation of physiological or developmental processes in response to red and far-red light and different light intensities.

Phytochromes are synthesized in an inactive (Pr) form that can be photoconverted by red light into the far-red light-sensitive Pfr form. The bioactive Pfr dimer is able to enter the nucleus, where it prevents elongation growth by inhibiting the growth-promoting PHYTOCHROME INTERACTING FACTOR (PIF) transcription factor family [4]. Members of the same family of PIF proteins are also pivotal for thermomorphogenesis, with PIF4 having a predominant role [2,5] (Figure 1A). *PIF4* transcript levels are induced in response to increased temperatures, resulting in enhanced expression of growth-promoting genes, including hormone biosynthesis and signalling genes that ultimately drive thermomorphogenesis [2,5,6].

Light and temperature signalling are not only connected through their effects on PIF abundance. Pfr reverts to inactive Pr in a process called dark or thermal reversion. Temperature sensitivity of dark reversion was described early on in phytochrome research [7]. However, compared with light-mediated reactions, thermal reversion was considered to be too slow to effectively alter the bioactive Pfr pool even under moderate (red) light levels. In two back-to-back papers, the groups of Phil Wigge [8] and Jorge Casal [9] recently demonstrated that thermal reversion of phyB-Pfr increases exponentially as a function of temperature over one to two orders of magnitude in both low light and dark conditions. This is much faster than was previously assumed. As a consequence, both studies show, in a complementary manner, that increasing temperature affects the physiological output of phyB (hypocotyl elongation) by reducing the light-activated Pfr pool.

The Light Side of Thermal Reversion

The work of the Casal group [9] focussed on the biochemical characterisation of thermal reversion of phyB-Pfr under continuous light conditions. They observed that warm temperatures reduced the active Pfr:Pfr homodimer pool inversely proportional to the red and white light intensity *in vitro* and *in vivo*. The reversion of active phytochrome appeared to be especially sensitive to high temperature at low levels of irradiance [9]. The conversion involved a two-step mechanism via an intermediate heterodimer of Pr:Pfr and, interestingly, temperature appeared to specifically speed up the second step from the heterodimer to the Pr:Pr inactive homodimer. Furthermore, the size of typical nuclear bodies that accumulate the bioactive Pfr:Pfr pool was reduced by increasing temperatures above ~20°C in a nonlinear manner. To verify whether the temperature effect on nuclear bodies was a consequence of phyB-Pfr thermal reversion, wild type and mutated *phyB* alleles incapable of thermal reversion were introduced into a *phyB* null mutant background. The size of phyB nuclear bodies was determined at different temperatures and light conditions. Following a multivariate modelling effort, the authors [9] confirmed that nuclear body size behaviour is, in part, a function of the thermal reversion of phyB-Pfr.

The Dark Side of Thermal Reversion

The Wigge group focussed on the dark side of thermal reversion and established that phytochromes are required for temperature-mediated hypocotyl elongation [8]. Their approach was largely based on highly resolved temporal transcriptome analysis across a short-day diurnal cycle. It revealed that a large part of the genes that are misregulated in phytochrome quintuple knockout mutants (*phyabcde*) overlapped with changes in wild type gene expression induced by warm temperatures during the night. Less overlap was observed with the daytime warm temperature transcriptome [8]. This led to the hypothesis that thermal reversion may relieve the repression of the warm temperature transcriptome during

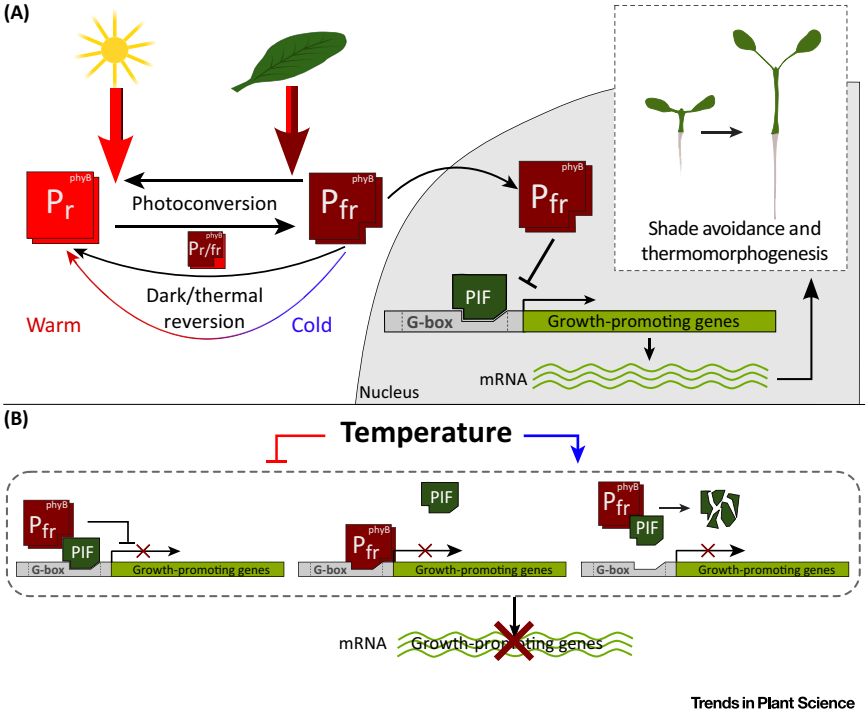

Figure 1. Model of Phytochrome B (phyB)-Mediated Temperature Sensing and Its Physiological Output. (A) Warm temperatures stimulate the reversion of photoactivated phyB-Pfr dimers, thereby derepressing PHYTOCHROME INTERACTING FACTOR (PIF)-mediated elongation growth (thermomorphogenesis) similar to far-red-enriched light conditions typical of canopies (shade avoidance). (B) Possible mechanisms of phyB-Pfr effects on PIF4-mediated elongation growth. PhyB-Pfr could act as co-repressor by binding to PIFs at G-boxes of growth promoting genes (left) or by competing for promoter binding sites (middle). Additionally, bioactive phyB could suppress PIF action by triggering its proteasomal degradation (right) [4].

the night. Indeed, a *phyB* mutant disabled in dark reversion exhibited constitutive repression of the warm temperature transcriptome during the dark period. Moreover, bioactive phyB was found to associate with PIF-binding sites (G-boxes) at promoters of dark period-expressed temperature-responsive genes in a manner that was reversely proportional to temperature cues. Based on these observations, the authors proposed that phyB has a dual role in controlling PIF4-mediated growth by both triggering its degradation and directly (co-)repressing expression of PIF4 target genes (Figure 1B). During warm nights, this repression of PIF-dependent elongation growth is quickly relieved by the fast thermal reversion of Pfr back to inactive Pr (Figure 1A). Conversely, growth suppression is sustained during cool nights. The emerging model also implies that phyB-mediated temperature sensing depends on prior light activation, because temperature-induced derepression of growth required previously activated repressive phyB Prf.

Open Questions

The breakthrough identification of a role for phyB in temperature sensing can directly be connected to previously identified crucial signalling components, such as PIF4, and raises several interesting questions. First, what is the exact nature of the PIF4 repression by phyB? Different scenarios could explain the interaction of phyB with G-box elements. One possibility would be indirect binding via the association with, and inhibition of, PIF4 (Figure 1B, left). Putative inhibition mechanisms entail conformational changes, blocking of the transcriptional machinery, or recruitment of transcriptional co-repressors. The co-repressor scenario in particular would provide a mechanism to greatly increase the dynamic range of the response. Alternatively, phyB and PIF4 might compete for promoter binding sites (Figure 1B, middle). Both possibilities (association/inhibition and competition for binding sites) could facilitate the inhibition of PIF4-mediated promotion of growth in addition to phyB-triggered proteasomal degradation of PIF4 (Figure 1B, right).

Second, do other phytochromes (phyA, C–E) or other photoreceptors (e.g., CRY2) that have partially distinct functions in light signalling also contribute to thermomorphogenesis in a similar manner? The analysis of temperature-induced hypocotyl elongation in *phyb-1* and *phyabcde* mutants indicated that phytochromes other than phyB appear to add repressive growth function, primarily at temperatures below 22°C. The hypothesis that other phytochromes may act as thermosensors under such cooler temperature conditions appears plausible, because the effect of phyA, phyD, and phyE on flowering time at 16°C is more prominent than that of phyB [10]. Given that the capacity of phyB temperature sensing appears to decline with increasing irradiance levels [9], the role of phyB or other light receptors in thermomorphogenesis under natural (i.e., high light) conditions needs to be examined in future work.

Third, how do natural variants of phyB contribute to alternate thermosensing in different arabidopsis (*Arabidopsis thaliana*) accessions or plant species? Natural variation in phyB activity has already been associated with differential light responses in arabidopsis [11]. Furthermore, phyB was identified as a potential thermomorphogenesis regulator in a quantitative genetic analysis [12], indicating that natural variation in temperature response may indeed be triggered via the

differential sensing capacities of phyB variants. Consequently, phyB may be an attractive candidate for allele-mining and targeted breeding approaches to reduce, or enhance, thermomorphogenic growth responses in crop species in efforts to breed climate warming-resilient cultivars. These and other matters will undoubtedly be addressed in future research endeavours following the milestone discovery of phyB acting as a thermosensor *in planta.*

Acknowledgements

This work was supported by a grant from the Deutsche Forschungsgemeinschaft to MQ (Qu 141/3-1).

[1]Institute of Agricultural and Nutritional Sciences, Martin Luther University Halle-Wittenberg, Betty-Heimann Str. 5, 06120 Halle (Saale), Germany
[2]Molecular Plant Physiology, Institute of Environmental Biology, Utrecht University, Padualaan 8, 3584 CH Utrecht, The Netherlands

*Correspondence:
marcel.quint@landw.uni-halle.de (M. Quint).

http://dx.doi.org/10.1016/j.tplants.2017.01.007

References

1. Crawford, A.J. *et al.* (2012) High temperature exposure increases plant cooling capacity. *Curr. Biol.* 22, R396–R397
2. Quint, M. *et al.* (2016) Molecular and genetic control of plant thermomorphogenesis. *Nat. Plants* 2, 15190
3. Fraser, D.P. *et al.* (2016) Photoreceptor crosstalk in shade avoidance. *Curr. Opin. Plant Biol.* 33, 1–7
4. Lorrain, S. *et al.* (2008) Phytochrome-mediated inhibition of shade avoidance involves degradation of growth-promoting bHLH transcription factors. *Plant J.* 53, 312–323
5. Koini, M.A. *et al.* (2009) High temperature-mediated adaptations in plant architecture require the bHLH transcription factor PIF4. *Curr. Biol.* 19, 408–413
6. Franklin, K.A. *et al.* (2011) Phytochrome-interacting factor 4 (PIF4) regulates auxin biosynthesis at high temperature. *Proc. Natl. Acad. Sci. U. S. A.* 108, 20231–20235
7. Schäfer, E. and Schmidt, W. (1974) Temperature dependence of phytochrome dark reactions. *Planta* 116, 257–266
8. Jung, J.-H. *et al.* (2016) Phytochromes function as thermosensors in *Arabidopsis*. *Science* 354, 886–889
9. Legris, M. *et al.* (2016) Phytochrome B integrates light and temperature signals in *Arabidopsis*. *Science* 354, 897–900
10. Halliday, K.J. and Whitelam, G.C. (2003) Changes in photoperiod or temperature alter the functional relationships between phytochromes and reveal roles for phyD and phyE. *Plant Physiol.* 131, 1913–1920
11. Filiault, D.L. *et al.* (2008) Amino acid polymorphisms in *Arabidopsis* phytochrome B cause differential responses to light. *Proc. Natl. Acad. Sci.* 105, 3157–3162
12. Box, M.S. *et al.* (2015) ELF3 controls thermoresponsive growth in *Arabidopsis*. *Curr. Biol.* 25, 194–199

Forum

Ethnobotany, Phylogeny, and 'Omics' for Human Health and Food Security

Teresa Garnatje,[1] Josep Peñuelas,[2,3] and Joan Vallès[4,*]

Here, we propose a new term, 'ethnobotanical convergence', to refer to the similar uses for plants included in the same node of a phylogeny. This phylogenetic approach, together with the 'omics' revolution, shows how combining modern technologies with traditional ethnobotanical knowledge could be used to identify potential new applications of plants.

Ethnobotany and the Search for New Drugs and Foods: The Classical Approach

Plants have always been a crucial resource for humans. Ethnobotany, located at the interface of natural and social sciences, is a discipline that addresses the relationships between humans and plants. Among the numerous applications of plants, those related to human health and well-being are the most diverse. Bioprospecting for new drugs with a botanical origin and for new food crops has classically been based on ethnobotanical information. Ethnobotanically directed bioprospecting has become more powerful than random assays for finding and identifying bioactive compounds from plants. Aspirin [from *Filipendula ulmaria* (L.) Maxim.], codeine and papaverine (from *Papaver somniferum* L.), colchicine (from *Colchicum autumnale* L.), digoxin and digitoxin (from *Digitalis purpurea* L.), tetrahydrocannabinol and cannabidiol (from *Cannabis sativa* L.), and vinblastine and vincristine [from *Catharanthus roseus* (L.) G.Don] are among the most famous classical drugs developed from ethnobotanical leads [1]. The first evidence for the anticancer properties of paclitaxel, from *Taxus* L. spp., came from its toxic effects on murine leukemia cells, in agreement with the well-known general toxicity of this genus of plants. The success of this anticancer product highlights the promising role of plant products in drug development. More recently, during the avian flu epidemic, oseltamivir was developed from *Illicium verum* Hook.f. based on ethnobotanical data from Chinese traditional medicine. Ethnobotanical records also led to the isolation and development of artemisinin (from *Artemisia annua* L.) as a powerful antimalarial drug [2], whose relevance was recognised with the 2015 Nobel Prize in Physiology or Medicine.

Linking Ethnobotanical Convergence, Phytochemistry, and Molecular Phylogenies to Predict Plant Uses

New perspectives have emerged with the development of new molecular tools, especially for DNA sequencing; these enable phylogenetic reconstruction with hot nodes clustering potentially useful plants, including species traditionally used for medicinal purposes (Figure 1). Promising predictions of medicinal plant uses have been developed based on the conjunction of ethnobotanical, phytochemical, and molecular phylogenetic data [3].

The use of the same (or closely related) species in the same ways in different cultures indicates that different and often non-interacting human groups have independently acquired this knowledge. This results from the fact that some plants have similar morphological characteristics because they have a close phylogenetic placement, a phenomenon termed 'evolutionary convergence'. Thus, we propose to use the term 'plant-use