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SHORT COMMUNICATION

Synchronization of photoperiod and temperature signals during plant thermomorphogenesis

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ABSTRACT
It is well-known that even small changes in ambient temperatures by a few degrees profoundly affect plant growth and morphology. This architectural property is intimately associated with global warming. In particular, under warm temperature conditions, plants exhibit distinct morphological changes, such as elongation of hypocotyls and leaf petioles, formation of small, thin leaves, and leaf hyponasty that describes an upward bending of leaf petioles. These thermoresponsive morphological adjustments are termed thermomorphogenesis. Under warm temperature conditions, the PHYTOCHROME INTERACTING FACTOR 4 (PIF4) transcription factor is thermoactivated and stimulates the transcription of the *YUCCA8* gene encoding an auxin biosynthetic enzyme, promoting hypocotyl elongation. Notably, these thermomorphogenic growth is influenced by daylength or photoperiod, displaying relatively high and low thermomorphogenic hypocotyl growth during the nighttime under short days and long days, respectively. We have recently reported that the photoperiod signaling regulator GIGANTEA (GI) thermostabilizes the REPRESSOR OF *ga1-3* transcription factor, which is known to attenuate the PIF4-mediated thermomorphogenesis. We also found that the N-terminal domain of GI interacts with PIF4, possibly destabilizing the PIF4 proteins. We propose that the GI-mediated shaping of photoperiodic rhythms of hypocotyl thermomorphogenesis helps plant adapt to fluctuations in daylength and temperature environments occurring during seasonal transitions.

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Text

The effects of environmental temperatures on plant growth and adaptive behaviors are quite complicated. It is extensively studied that plants respond to stressful high and low temperatures by altering plant physiology and morphology.[1,2] It is interesting that small temperature changes by a few degrees markedly affect plant growth and developmental patterns. These plant responses would underlie the frequently reported alterations in plant morphology and physiology that occur in response to global warming, an ecological worldwide issue in recent decades.[3,4] Under warm temperature environments, plants exhibit diverse morphological and architectural changes, which are readily recognized by hypocotyl elongation, leaf hyponasty, and small, thin leaves.[5–7] It is also known that flowering is accelerated at warm temperatures.[8,9] These thermoinduced morphological alterations are termed thermomorphogenesis.[6,7,10] It is widely perceived that the thermomorphogenic traits facilitate dissipation of plant body heat and evaporative leaf cooling, contributing plant adaptation to warm temperatures.[7,10]

A large set of molecular genetic and physiological approaches have functionally identified thermoresponsive genes and established associated molecular mechanisms governing plant thermomorphogenic hypocotyl growth.[6,11–13] The PIF4 transcription factor plays a central role in thermomorphogenic hypocotyl growth. It is activated under warm temperatures and transcriptionally activates the expression of the *YUC8* gene encoding an auxin biosynthetic enzyme, leading to hypocotyl elongation.[6]

Interestingly, accumulating evidence indicate that thermomorphogenic growth is mediated by daylength information, reaching its peak around midday under long days (LDs) but at the end of the night under short days (SDs).[12,13] It is known that the E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1), which functions in light signaling,[14] conveys temperature information to hypocotyl thermomorphogenesis.[11,13] It is therefore anticipated that plants would operate distinct molecular mechanisms that monitor daylength information to shape the diel rhythms of thermomorphogenic responses under fluctuating photoperiod and temperature environments, which frequently occur during seasonal transitions in nature.[15,16]

Plant growth hormone gibberellic acid (GA) is known to promote hypocotyl growth. Five DELLA proteins, such as REPRESSOR OF *ga1-3* (RGA), GIBBERELLIC ACID INSENSITIVE (GAI), RGA-LIKE 1 (RGL1), RGL2, and RGL3, are major repressors of GA signaling in *Arabidopsis*.[17] Previous studies have shown that the DELLA proteins suppress hypocotyl elongation by reducing the protein stability and DNA-binding affinity of PIFs.[18,19] Meanwhile, it is known that DELLA proteins play crucial roles in photoperiod-dependent plant defense responses.[20] On the basis of the physiological functions of DELLA proteins, it is suggested that DELLA proteins are also involved in the photoperiodic rhythms of PIF4-dependent thermomorphogenic growth.

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We have recently reported that the light signaling mediator GIGANTEA (GI), which accumulates to a high level under LDs,[21] acts as a distinct molecular chaperone in stabilizing the DELLA proteins at warm temperatures,[22] which are otherwise rapidly degraded through the gibberellic acid (GA)-mediated ubiquitination-proteasome pathway. Notably, the N-terminal domain of GI, which harbors a chaperonic activity,[23] interacts with RGA. It is likely that the chaperonic activity of GI either protects DELLA proteins from the as-yet unidentified ubiquitin-proteasome pathways or facilitates the formation of active GI-DELLA complexes. DELLA proteins, such as RGA, act as a suppressor of PIF4 function.[18,19] Therefore, the GI-mediated stabilization of DELLA proteins causes the suppression of PIF4 function during hypocotyl thermomorphogenesis, leading to a relatively low thermomorphogenic hypocotyl growth in the nighttime under LDs. In contrast, under SDs, GI accumulation is reduced,[21] leading to the reduction of DELLA proteins and the resultant elevation of PIF4 function.

Notably, recent studies have shown that GI regulates the PIF activity at multiple levels.[24,25] Consistent with this, we found that the N-terminal domain of GI directly interacts with PIF4 (Figure 1). On the basis of the enhanced stability PIF4 proteins in GI-defective mutants at warm temperatures,[22] it is envisioned that the N-terminal domain of GI triggers the degradation of PIF4 by recruiting E3 ubiquitin ligases, which is in contrast to its role in stabilizing DELLA proteins. Notably, the BLADE-ON-PETIOLE proteins (BOPs) form an E3 ubiquitin ligase complex to induce PIF4 protein turnover.[26] Thus, it will be worthy of investigating the potential relationship between GI and BOPs during thermomorphogenesis.

What is the physiological relevance of the GI-mediated photoperiodic rhythmicity of hypocotyl thermomorphogenesis? It might be functionally linked with the GI-mediated flowering promotion in Arabidopsis. While Arabidopsis flowering is promoted under LDs, it is delayed under SDs.[27] The two distinct GI-mediated processes are likely to be metabolically associated with each other, and, as a result, saving metabolic resources by attenuating thermomorphogenic hypocotyl growth would facilitate flowering acceleration under LDs.

It is also notable that the GI-mediated regulation of PIF4 function provides a molecular clue as to an external coincidence model underscoring hypocotyl thermomorphogenesis.[28] It has been suggested that hypocotyl thermomorphogenesis actively occurs when plants simultaneously perceive warm temperature and timing signals.[13,28] In this signaling scheme, the phytochromes are inactivated at warm temperatures, leading to thermal activation of PIF4,[29] while GI conveys photoperiodic information to attenuate PIF4 activity (Figure 2). The photoperiod-temperature signaling crosstalk would be necessary during seasonal transitions, when the synchronization of daylength information and temperature cues is often interrupted.[30]

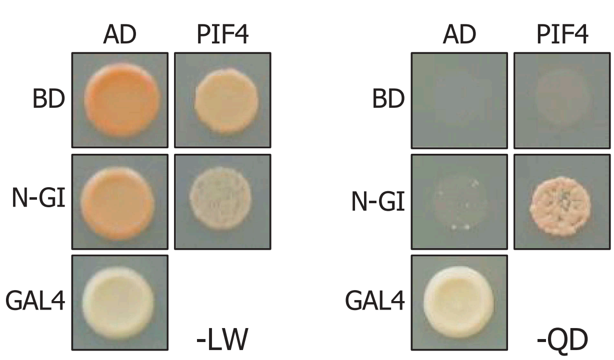

Figure 1. The N-terminal domain of GI interacts with PIF4. The protein-protein interactions between the N-terminal domain of GI (N-GI) covering residues 1–507 and PIF4 were analyzed by yeast two-hybrid assay. -LW indicates Leu and Trp dropout plates. -QD indicates Leu, Trp, His, and Ade dropout plates.

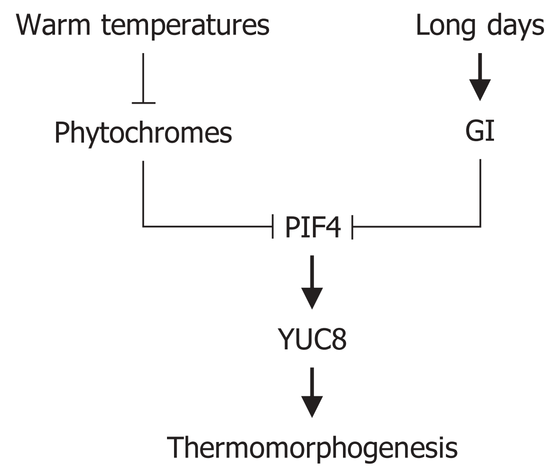

Figure 2. An external coincidence model for hypocotyl thermomorphogenesis. In this model, warm temperatures activate PIF4 perhaps by accelerating the thermal conversion of the phytochromes. Meanwhile, GI conveys photoperiod information to attenuates PIF4 function. Therefore, the rhythmic coincidence of the two external signals shapes the photoperiodic rhythms of thermomorphogenic hypocotyl growth.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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