

Not too short and not too long: SMAX1 optimizes hypocotyl length at warmer temperature

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In Brief

What happens to life on earth as the planet warms? In warm-blooded animals, behavioral changes (such as seeking shade and migrating) represent immediate strategies to maintain a constant body temperature in response to increasing air temperatures. Nonetheless, a recent review highlighted that several animal species acclimated to warmer environments have gradually increased the size of specific body parts (Ryding et al., 2021). These morphological changes—called shape-shifting—represent adaptive strategies to alleviate overheating by enlarging the surface area of appendages (e.g. beaks, ears, tails) that play key thermoregulatory roles. Plants cannot migrate but have developed several cooling capacities to cope with warmer climates throughout their life cycle. A set of adaptive strategies—collectively defined as *thermomorphogenesis*—includes stem elongation to move the delicate shoot apex away from the warm soil, and upward bending of leaves to increase air circulation near green organs.

In the past decade, molecular genetic studies in the model species *Arabidopsis thaliana* have deciphered the complex regulatory mechanisms governing thermomorphogenic responses, especially at early stages of plant development. During seedling establishment at elevated ambient temperatures, the elongation of the hypocotyl (i.e. embryonic stem) is largely controlled by a regulatory module constituted by the temperature-sensitive photosensor Phytochrome B (PhyB), the transcription factor PHYTOCHROME-INTERACTING FACTOR 4 (PIF4), and downstream genes responsive to the growth hormone auxin (reviewed in Legris et al., 2017). Additional regulators have been identified, but functional relationships between different components of this signal transduction pathway in response to fluctuating temperatures remain to be elucidated.

In this issue, **Young-Joon Park and colleagues** (Park et al., 2022) carried out thermomorphogenic assays to identify mutants with impaired hypocotyl elongation at warm temperature (28°C) versus normal temperature (23°C). Mutations in *SUPPRESSOR OF MAX2 1* (SMAX1), a gene involved in seed germination and photomorphogenesis downstream of the KARRIKIN INSENSITIVE2 (KAI2) receptor (Stanga et al., 2013), reduced thermal sensitivity of hypocotyl growth. Indeed, *smax1* mutants displayed shorter hypocotyls as compared to wild-type seedlings, not only at higher temperatures but also in different light conditions.

Transcriptomic analyses revealed that *smax1* mutants grown at warmer temperatures showed differential expression of auxin-related genes, and specifically downregulation of the auxin biosynthetic gene *YUCCA8* (*YUC8*), a downstream target of PIF4. These findings suggested a functional link between SMAX1 and the PhyB–PIF4 module in thermal induction of hypocotyl growth, further corroborated by genetic analyses of higher order mutants. Interestingly, the SMAX1 protein does not directly interact with PIF4 but rather affects its transactivation ability on *YUC8*, likely through physical association with PhyB and consequent release of its inhibitory effect on PIF4 function.

The authors smartly demonstrated that protein integrity is fundamental for SMAX1 function, since the N-terminal domain mediates the interaction with PhyB while the C-terminal part contains the sequences required for its nuclear localization. Unexpectedly, SMAX1 protein abundance slowly decreased after exposure to warm temperatures; although this finding contrasts with the proposed promotive role on hypocotyl elongation, thermal degradation of SMAX1 could avoid stem overgrowth that can be deleterious for seedling survival. Accordingly, the expression of *YUC8* peaked after

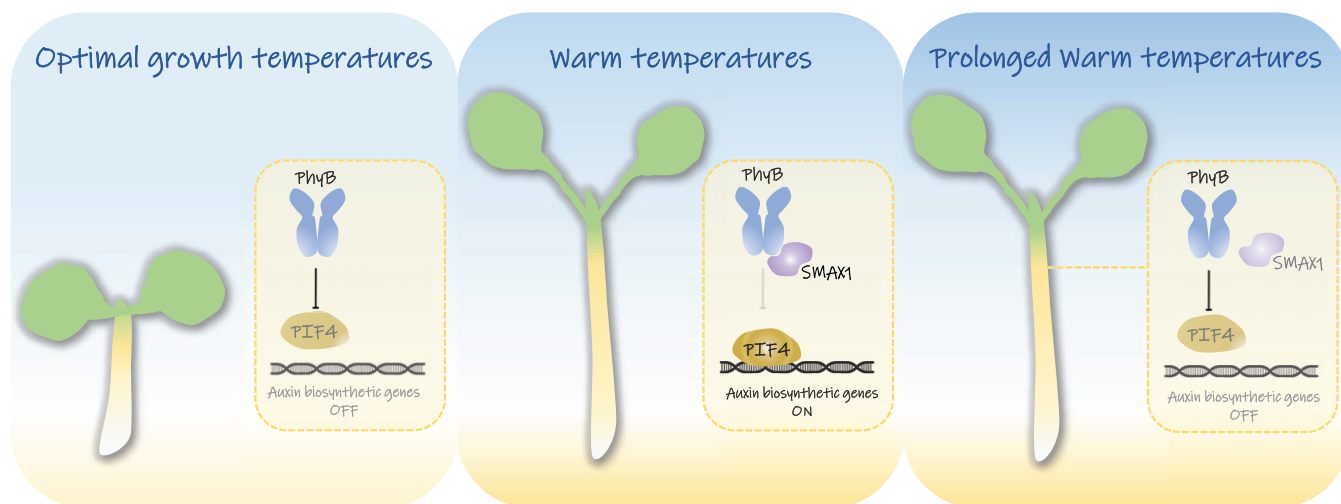


Figure Regulation of hypocotyl elongation in response to warm temperature. At optimal growth temperature, PhyB promotes the degradation of the transcription factor PIF4. At warmer growth temperature, SMAX1 reduces the repressive function of PhyB on PIF4, thus promoting hypocotyl elongation via transcriptional activation of auxin biosynthetic genes. After prolonged warm exposure, the thermal degradation of SMAX1 prevents hypocotyl overgrowth by indirectly repressing auxin synthesis. Figure credits: M. Osnato (modified schematic model from figure 8).

24 h at 28°C but dropped afterwards, thus preventing excessive hypocotyl elongation in response to prolonged warm exposure (Figure).

To conclude, living organisms react in different ways to climatic warming but a common adaptive strategy relies on the dissipation of excessive heat and protection of delicate tissues by increasing the size of specific organs. These morphological alterations should be tightly controlled as they might be beneficial in the short term but detrimental in the long term. What happens to life on earth if temperatures keep rising? Can living organisms continuously adapt to a changing environment?

References

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