IN BRIEF

A Tripartite Growth Regulatory Cascade of Basic Helix-Loop-Helix Transcription Factors

Plant growth is regulated by a multiplicity of factors, many of which are transduced via hormone signaling. However, how these hormone signals converge to affect growth is not entirely clear (reviewed in Depuydt and Hardtke, 2011). New work from Ikeda et al. (2012) and Bai et al. (2012) demonstrates that the cell elongation component of growth is regulated by a mechanism involving groups of three basic helix-loop-helix (bHLH) transcription factors (some of which lack the basic domain).

*Arabidopsis thaliana* bHLH PACLOBURTRAZOL RESISTANT1 (PRE1) positively regulates cell elongation and interacts antagonistically with IL1 BINDING BHLH PROTEIN1 (IBH1) (see figure), and these activities occur downstream of brassinosteroids (BRs) and during the developmental transition from young to mature organs (Zhang et al., 2009). IBH1 expression is negatively regulated by BR, whereas PRE1 expression is activated by BR and gibberellic acid and repressed by light (Oh et al., 2012; Bai et al., 2012b). Ikeda et al. (2012) and Bai et al. (2012a) independently identified bHLH proteins of a third type that are also involved in this regulation of cell elongation.

Ikeda et al. found that IBH1 acts as a transcriptional repressor, despite its lack of DNA binding. They identified several bHLH proteins, including three related proteins, ACTIVATOR FOR CELL ELONGATION1 (ACE1) to ACE3, as interactors with IBH1. ACE1 was shown to bind DNA and promote the expression of EXPANSIN 8 (EXP8), and ACE1 activity was inhibited by IBH1, whereas the addition of PRE1 relieved this inhibition. These results support a model in which the ACEs promote cell elongation by increasing the expression of factors involved in the process (e.g., expansins that loosen the cell wall) and in turn are negatively regulated through dimerization with IBH1, which itself can be sequestered via dimerization with PRE1.

Bai et al. also found several related bHLH proteins as interacting with IBH1 and explored the role of one of them, HOMOLOG OF BEE2 INTERACTING WITH IBH1 (HB1). Although neither PRE1 nor IBH1 binds DNA, they show that HB1 does and, furthermore, that it binds specifically to the promoter of the EXPANSIN gene EXP1. HB1 promoted EXP1 expression, but IBH1 inhibited HB1 binding to DNA, and this inhibition was released in the presence of PRE1. Importantly, cell elongation in PRE1/ HB1-deficient or IBH1 overexpression lines was less sensitive to BR, gibberellic acid, and temperature, suggesting that these signals converge on this cascade.

Together, these reports underscore the importance of this triple bHLH regulatory cascade in growth regulation in response to a variety of stimuli. Strikingly, ACE1 and HB1 are both members of bHLH subfamily 25 (Carretero-Paulet et al., 2010). Whether other members of this subfamily act in cell elongation and are similarly key regulators remains to be explored.

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REFERENCES


Overexpression of *IBH1* (IBH1-Ox) leads to dwarfism compared with the wild type (Columbia), but overexpression of *PRE1* (PRE1-Ox) suppresses this effect. (Reprinted from Bai et al. [2012a], Figure 1A.)
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