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An *Elongated-D* Mutation Acts Downstream of the Photosensory Signaling Pathways

Ji Hyun Jeon[1], Mi Kwon[1], Hojin Ryu[2], Vitnary Choe[1], Ildoo Hwang[2], Ok Sun Lee[1], and Sunghwa Choe[1],*

[1]Department of biological sciences, College of natural sciences, Seoul National University, Seoul 151-747, Korea

[2]Department of life sciences, Pohang University of Science and Technology (POSTECH), Pohang 790-784, Korea

Brassinosteroids (BRs) stimulate plant growth through a receptor kinase-mediated signaling pathway. Contrast to the conventional BR dwarf phenotypes, tall plant stature can be induced due to a gain-of-funtion mutation in a gene for positive regulator or a loss-of-function mutation in a negative regulator in BR signaling pathways. We obtained a list of long hypocotyl mutants from Arabidopsis Biological Resources Center (ABRC), and made double mutants with a BR receptor mutant *bri1-5*. Among the mutants tested, *elongated-D* (*elg-D*) caused a significant suppression of a dwarf phenotype seen in bri1-5. Thus, we hypothesized that *elongated-D* has a dominant mutation in one of the BR signaling components. Using a map-based cloning method, we identified that *ELG* encodes for BRASSINOSTEROID INSENSITIVE1-ASSOCIATED KINASE1 (BAK1). The *elg-D* mutation caused a substitution of Asp at the 123th amino acid residue for Ile, which is located in an extracellular Leucine-rich repeat domain of BAK1. To understand the nature of this mutation, we expressed *ELG/BAK1* using a protoplast transient expression system. We found that an LRR domain of ELG/BAK1 is heavily glycosylated and processed, and eventually led to a protein degradation, whereas the BRI1 protein was stable and was not further processed. To test if this modification of ELG/BAK1 is necessary for their activity as a co-receptor for BRs, we separated the two domains, an extracellular LRR and an intracellular kinase domain, and expressed independently both in protoplasts and transgenic plants. Detailed analysis of the phenotypes shown by the transgenic lines should reveal a role for these domains in BR signaling pathways.