Final Report
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PI: Jianming Li
University of Michigan

The proposed research had two main objectives: 1) investigating the molecular mechanism by which BRs activate the BRI1-containing steroid receptor; and 2) to investigate the molecular mechanism of BRI1 function. During the course of this project, several research papers were published from other laboratories, which reported studies similar to our proposed experiments. We therefore changed our research direction and focused our research efforts on 1) molecular genetic studies of several extragenic suppressors of a weak *bri1-9* mutant (which were named as *EMS-mutagenized bri1 suppressor* or *ebs*) and 2) biochemical characterization of the protein products of the cloned *EBS* genes. This switch turned out to be extremely successful and led to a surprising discovery that the dwarf phenotype of the well-studied *bri1-9* mutant is not due to the failure of the bri1 receptor to bind the plant steroid hormone but rather caused by the retention of a structurally-imperfect but biochemically-competent bri1-9 and its subsequent degradation in the endoplasmic reticulum. This initial discovery coupled with subsequent cloning and further studies of additional *EBS* genes significantly increased our understanding of the protein quality control mechanisms in plants, a severely under-studied research topic in plant biology. Our DOE-supported project resulted in publications of 8 research papers (see the publication list) and training of three postdoctoral research fellows (***black italicized names***), three graduate students (underlined names), and a total of 8 undergraduate students (two being coauthors on two of the listed papers, **black underlined names**).

The publication list:

1. **Li J**, Jin H (2007) Regulation of brassinosteroid signaling. *Trends Plant Sci.* **12**, 37-41.
2. Jin H, Yan Z, ***Nam KH***, **Li J** (2007) Allele-specific suppression of a defective brassinosteroid receptor reveals an essential role of UDP-glucose:glycoprotein glucosyltransferase for a high-fidelity ER quality control. *Mol. Cell* **26**, 821-830.
3. Shan L., He P., **Li J.**, Heese A., Peck S. C., Nurnberger T., Martin G. B., and Sheen J. (2008). Bacterial effectors target the common signaling partner BAK1 to disrupt multiple MAMP receptor-signaling complexes and impede plant immunity. *Cell Host & Microbe* **4**, 17-27.
4. ***Hong Z***, Jin H, Tzifira T, **Li J** (2008) Multiple mechanism-mediated retention of a defective brassinosteroid receptor in the endoplasmic reticulum. *Plant Cell* **20,** 3418-3429.
5. ***Hong Z***, Jin H, Fitchette A-C, Xia Y, **Monk AM**, Faye L, and **Li J**. (2009). Mutations of an alpha-1,6 mannosyltransferase inhibit endoplasmic reticulum–associated degradation of defective brassinosteroid receptors in Arabidopsis. *Plant Cell* **21**, 3792-3802.
6. **Li J** (2010) Regulation of the nuclear activities of brassinosteroid signaling. *Curr Opin Plant Biol* **13**, 540-547.
7. ***Hong Z*** and **Li J** (2012) The protein quality control of plant receptor-like kinases in the endoplasmic reticulum. In Receptor-Like Kinases in Plants. Eds F. Tax & B. Kemmerling (Springer-Verlag Berlin Heidelberg). pp 275-307.
8. ***Hong Z***, Kajiura H, ***Su W***, Jin H, **Kimura A**, Fujiyama K, and **Li J** (2012) An evolutionarily conserved glycan signal to degrade aberrant brassinosteroid receptors in Arabidopsis. *Proc. Natl. Acad. Sci. USA*, **109:**11437-11442.