Plants control their size through the action of several phytohormones. One class of growth-promoting hormones is the brassinosteroids (BRs), the polyhydroxylated steroid hormones of plants. Here, we present the Arabidopsis-specific proteins that are the founding members of key BR signaling pathway components found in all plants. The genetic studies that identified these components are unique to Arabidopsis owing to its rapid generation time, sophisticated genetics, and facile transformation protocols, thereby highlighting the importance of a reference plant for understanding fundamental processes in all land plants.

This record contains information specific to the Arabidopsis Brassinosteroid Signaling Pathway.

During the past decade, substantial progress has been made in elucidating the molecular mechanisms underlying brassinosteroid (BR) signaling in Arabidopsis thaliana (1) (Fig. 1). Several saturated forward genetic screens designed to uncover loci required for BR responsiveness identified a large number of recessive alleles of only a single gene, called BRI1 (brassinosteroid insensitive 1) (1, 2). Plants with bri1 mutations show similar defects to those of BR biosynthetic mutants: The plants are dark-green dwarfs with epinastic leaves and have delayed senescence, reduced fertility, and reduced apical dominance (3). When grown in the dark, these mutants display many features of light-grown seedlings, suggesting that BRs can uncouple light signals from light-dependent processes (4). Thus, BRI1 is a major nonredundant component of BR signaling. BRI1 is a member of the largest class of predicted receptors in plants, the leucine-rich-repetor receptor kinases (LRR-RKs) (5).

BRI1 is localized to the plasma membrane and has a predicted extracellular domain with an N-terminal signal peptide followed by 24 imperfect leucine-rich repeats. The binding site for active BRs is the atypical LRR, LRR21, and a 70–amino acid “island” found between LRRs 20 and 21 (6). BRI1 has a single transmembrane domain and an intracellular serine/threonine kinase domain, which is kept in its basal state by the C-terminal tail of 41 amino acids (7). In the absence of steroid, BKI1, a plasma membrane-associated protein, interacts directly with the kinase domain of BRI1 to negatively regulate the signaling pathway (8).

Binding of BRs to preformed BRI1 homo-oligomers leads to the dissociation of BKI1 from the plasma membrane (7, 8). Subsequently, BRI1 autophosphorylates and associates with a second LRR-RK called BAK1 (BRI1-associated receptor kinase1) (9–11). BAK1 has a short extracellular domain composed of five LRRs and lacks the critical 70–amino acid “island” domain responsible for BR-binding. It has been proposed that the physical interaction between BRI1 and BAK1 leads to the formation of a signaling-competent hetero-oligomer (9–11).

The signals transmitted from the plasma membrane-localized BRI1-BAK1 hetero-oligomer negatively regulate the activity of a glycogen synthase kinase 3 (GSK-3), called BIN2 (12, 13). Although the mechanism is as yet uncharacterized, inactivation of BIN2 leads to the dephosphorylation of BES1 and BZR1, members of a new family of plant-specific transcription factors (14–17). BES1, and likely other family members, are further dephosphorylated through the activity of a nuclear-localized kelch-containing protein phosphatase BSU1 (18). Current data suggest that dephosphorylated BES1 is then able to form homo- or heterodimers with the basic helix-loop-helix (bHLH) transcription factor BIM1, to bind to E-box elements in the promoters of BR-regulated genes (19). Dephosphorylated BZR1 binds to a novel element in the promoters of BR biosynthetic genes to repress their expression (16).

Because BES1 is 89% identical to BZR1, it is expected that BES1 and BZR1 will have both activating and repressing activities (20).

Other proteins have been identified that interact genetically or physically with BRI1, but their precise functions are currently unknown. In vitro and in vivo, BRI1 associates with TTL, a transhysterin-like protein (21). Overexpression of TTL causes slight dwarfing, suggesting that it may play a negative role early in the BR signaling pathway (21). A suppressor screen using a weak allele of bri1 identified a secreted and active carboxypep-
tidase, called BRS1 (22, 23), although its molecular target in the BR signaling pathway is unknown. Biochemical studies identified TRIP-1 as a BRI1 interactor (24); however, the knockdown in expression of TRIP family members yields a pleiotropic phenotype that is slightly reminiscent of those observed for BR biosynthetic or signaling mutants (25).

Pathway Details
URL: http://stke.sciencemag.org/cgi/cm/stkecm;CMP_19349
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References

Arabidopsis Brassinosteroid Signaling Pathway
Youssef Belkhadir, Xuelu Wang and Joanne Chory (November 28, 2006)
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