REVIEW ARTICLE

Brassinosteroid actions in plants

Jianming Li and Joanne Chory
Howard Hughes Medical Institute, Plant Biology Laboratory, The Salk Institute for Biological Studies, 10010 N. Torrey Pines Road, La Jolla, CA 92037, USA

Received 8 July 1998; Accepted 6 October 1998

Abstract

Recent studies on dwarf mutants of Arabidopsis, tomato and pea have provided convincing evidence that brassinosteroids are a unique class of plant hormones that are essential for normal plant growth. Detailed metabolic analyses of these mutants, coupled with thorough molecular and biochemical studies of their corresponding genes and gene products, are essential for a better understanding of brassinosteroid biosynthesis and its regulation. Molecular and genetic approaches have been undertaken to dissect the brassinosteroid signalling pathway, leading to the identification of a putative brassinosteroid receptor and a few brassinosteroid-response genes. Further studies should expand our knowledge on how brassinosteroids are perceived and transduced to regulate plant development.

Key words: Brassinosteroïd, biosynthesis, signal transduction, dwarf, receptor kinase.

Introduction

Steroid hormones are crucial for embryonic development and adult homeostasis in animals (Evans, 1988). Similarly, the insect steroid hormone, ecdysone, triggers a key regulatory cascade of gene expression controlling many developmental processes such as moulting, metamorphosis and reproduction (Thummel, 1995). In plants, many steroids have been identified, but only one class of steroids, called collectively brassinosteroids, have wide distribution throughout the plant kingdom and unique growth-promoting activity when applied exogenously (Mandava, 1988; Fujioka and Sakurai, 1997). Physiological studies done in several laboratories have demonstrated that brassinosteroids can induce a broad spectrum of cellular responses such as stem elongation, pollen tube growth, leaf bending and epinasty, root inhibition, induction of ethylene biosynthesis, proton-pump activation, xylem differentiation, and regulation of gene expression (Mandava, 1988; Clouse and Sasse, 1998). In addition, useful agricultural applications have been found such as increasing yield and improving stress resistance of several major crop plants (Cutler et al., 1991). Despite this extensive research, a definitive proof that these steroids are essential for normal plant growth has been lacking.

A clear role for these steroids in plant growth and development came from an entirely different field of studies: genetic analysis of plant photomorphogenesis. In plants, immediate post-germinative growth usually occurs beneath the soil surface and newly germinated seedlings need to bring their leaves into full sunlight so that they can start photosynthesis to provide the essential raw materials needed for long-term growth. An etiolated seedling, with an apical hook, closed cotyledons, and a rapidly elongating hypocotyl, can reach the soil surface quickly without damaging its young leaves. Once the seedling is in full sunlight, its growth pattern changes: the cotyledons expand and new leaves develop, accompanied by the induction of many genes involved in photosynthetic function, while the hypocotyl, whose elongation is dramatically inhibited, becomes thicker and acts as a physical support to hold newly developed leaves. Such a developmental switch from one suited to an underground environment to one suited to full sunlight is often referred to as photomorphogenesis, or de-etiolation (Cosgrove, 1986).

In order to understand the molecular mechanisms regulating this process, a genetic screen was initiated to identify Arabidopsis mutants that display many characteristics of light-grown plants when grown in the dark (Chory et al., 1989). So far, mutants of over 18 loci have been isolated and characterized (Wei and Deng, 1996).
Among these mutants are recessive alleles of a gene called DET2. Loss of-function mutations in DET2 have pleiotropic effects on Arabidopsis development (Chory et al., 1991). In the dark, det2 mutants are short, have thick hypocotyls, accumulate anthocyanins, have open, expanded cotyledons, develop primary leaf buds, and inappropriately express light-regulated genes. In the light, det2 mutants are dark-green dwarfs, have reduced apical dominance and male fertility, display altered photoperiodic responses, show delayed chloroplast and leaf senescence, and respond improperly to fluctuations in their light environment. Such phenotypic differences between det2 mutants and wild-type Arabidopsis plants indicate that DET2 plays an important role throughout Arabidopsis development.

Brassinosteroid biosynthesis

DET2 encodes a protein that exhibits significant sequence identity with mammalian steroid 5α-reductases that are involved in animal steroid metabolism (Li et al., 1996). In addition, a conservative substitution of a glutamate (Glu204), which is absolutely conserved among all mammalian steroid 5α-reductases and is essential for the activity of a type 2 human 5α-reductase (Russell and Wilson, 1994), completely abolishes the in vivo activity of DET2 protein, strongly suggesting that DET2 might be involved in the biosynthesis of plant steroids such as brassinosteroids. Consistent with this hypothesis, brassinosteroid treatment rescued the growth defects of both dark and light-grown det2 mutants (Li et al., 1996; Fujioka et al., 1997). When expressed in mammalian cells, DET2 protein was able to 5α-reduce several animal steroids including testosterone and progesterone. Moreover, the expression of human genes encoding steroid 5α-reductases complemented a det2 mutation, demonstrating that DET2 is a functional homologue of mammalian steroid 5α-reductases (Li et al., 1997). det2 mutants accumulated only 8–15% of the wild-type level of campestanol and less than 10% of the wild-type levels of other brassinosteroids and failed to convert [3H]labelled campesterol to [3H]labelled campestanol, showing that det2 is a brassinosteroid-deficient mutant. A further proof came from rescue experiments in which all intermediates of the brassinosteroid biosynthetic pathway after the DET2 reaction were capable of rescuing det2 mutant phenotypes. Taken together, these results establish that DET2 is a brassinosteroid biosynthetic enzyme catalysing an early step in the brassinosteroid biosynthetic pathway (Fig. 1; Fujioka et al., 1997).

In addition to the DET2 gene, there are several putative brassinosteroid biosynthetic genes that have been cloned recently in Arabidopsis. Both CPD and DWF4 encode cytochrome P450 proteins that share sequence similarities with mammalian steroid hydroxylases and feeding experiments with various brassinosteroids suggest that they act at the C-23 and C-22 hydroxylation steps, respectively, in the biosynthetic pathway (Fig. 1; Szekeres et al., 1996; Choe et al., 1998a). Both CBB3 (Kauschmann et al., 1996) and DWF3 (Choe et al., 1998b) are allelic to CPD. The Arabidopsis DWF1/DIM1/CBB1 protein (Feldmann et al., 1989; Takahashi et al., 1995; Kauschmann et al., 1996) has strong homology with several unknown proteins of pea, human, and C. elegans (J. Li and J. Chory, unpublished data), and is suggested to be an FAD-dependent oxidoreductase (Mushegian and Koonin, 1995). Quantitative analysis of endogenous brassinosteroid levels in dim1 mutants suggests that the dim1 mutation might block the conversion of 24-methylenecholesterol to campesterol (Fig. 1; Clouse and Sasse, 1998). Mutations in each of these genes result in phenotypes that are very similar to those of det2 mutants and these mutants can also be rescued by brassinosteroid treatment. Given the fact that mutations in each of these genes affect many aspects of light-regulated Arabidopsis development, the recent molecular genetic analyses of these dwarf mutants have provided convincing evidence that brassinosteroids are a unique class of plant hormones that are essential for normal plant growth and development.

The identification of additional brassinosteroid-deficient dwarf mutants in both Arabidopsis and other plant species further confirms the importance of these steroids in plant development. Three novel Arabidopsis dwarf mutants including dwf5, dwf7 and dwf8 were recently found to be defective in brassinosteroid biosynthesis (Choe et al., 1998b). Results from feeding experiments and analyses of endogenous brassinosteroid levels have indicated that both dwf5 and dwf7 mutants are defective in the formation of 24-methylenecholesterol, a precursor to campesterol, while the dwf8 mutation is suggested to block a late step after 3-dehydrosterasterone in the brassinolide biosynthetic pathway (Fig. 1; Choe et al., 1998b). The lkb mutant of pea is a brassinosteroid-deficient dwarf that shows normalization of internode elongation upon application of a range of brassinosteroids (Nomura et al., 1997) and a recent study suggested that the lkb mutation, like the Arabidopsis dim1 mutation, blocks the conversion of 24-methylenecholesterol to campesterol (Fig. 1; Clouse and Sasse, 1998). Another pea mutant, lk, is believed to be defective in the conversion of campesterol to campestanol and the corresponding gene LK might encode a pea homologue of the Arabidopsis DET2 (Fig. 1; Yokota et al., 1997). The DWARF gene of tomato was recently cloned by transposon tagging and shown to encode a cytochrome P450 with 38% sequence identity to the Arabidopsis CPD (Bishop et al., 1996). Preliminary feeding experiments suggest that the tomato DWARF is also a brassinosteroid biosynthetic enzyme, acting later in the biosynthetic pathway than CPD (Clouse
Fig. 1. Proposed pathways for brassinolide biosynthesis. The early C-6 oxidation pathway is on the left side and the late C-6 oxidation pathway is on the right side. Steps of phytosterol biosynthesis leading to the formation of campesterol are included and known mutations that block either biosynthesis of or response to brassinosteroids are indicated (X). Question marks indicate uncertainty about exact biochemical lesions and the broken arrows denote multiple steps. This diagram is adopted from Clouse and Sasse (1998) and Choe et al. (1998a).
Another tomato mutant, dpy, was found to be deficient in C-23 hydroxylation of both cathasterone and 6-deoxocathasterone, implying that DPY is the tomato homologue of CPD (Fig. 1; Clouse and Sasse, 1998).

The brassinosteroid-deficient mutants and the cloned brassinosteroid-biosynthetic genes provide an important tool for investigating not only the physiological functions but also the biosynthetic pathways of brassinosteroids. The detailed metabolic analyses of det2 mutants coupled with the biochemical characterization of DET2 protein aided the identification of a 3-oxo, Δ4–5 steroid, 3-dehydro-Δ4–5-campesterol, as a direct substrate of the DET2 reductase (Fig. 1; Li et al., 1997; Fujioka et al., 1997). Such a study also concluded that Arabidopsis must have a plant orthologue of the mammalian 3β-hydroxysteroid dehydrogenase/Δ5-4-Δ4-5 isomerases to catalyse the conversion of campesterol to 3-dehydro-Δ4–5-campesterol and the conversion of 3-dehydrocampestanol to campestanol (Fig. 1). Indeed, recent database searches indicate that the Arabidopsis genome contains at least two genes for such an enzyme (J Li and J Chory, unpublished results). Quantitative analysis of campestanol levels in three presumed null alleles of det2 has suggested that there is a second steroid 5α-reductase in Arabidopsis that plays a minor role in brassinosteroid biosynthesis (Fujioka et al., 1997), which might explain the relatively weak phenotypes of det2 mutants compared to cpd/cbb3/dwf3 mutants. Further molecular and genetic studies should aid in the identification of this second reductase and the elucidation of its physiological function during plant development.

The existence of various brassinosteroid-deficient mutants allowed the testing of the possibility that brassinolide is not synthesized via a simple linear biosynthetic pathway. Recently, two pathways, the so-called early C-6 oxidation and late C-6 oxidation pathways, were proposed for the biosynthesis of brassinolide (Fig. 1; Fujioka and Sakurai, 1997). Feeding experiments with intermediates of both pathways provided strong genetic evidence that both pathways are operating in Arabidopsis (Fujioka et al., 1997; Choe et al., 1998a). A recent study with dwf4 mutants suggests that 22-hydroxyl-campesterol might be a starting point for a new subpathway since this compound is able to rescue dwf4 mutations (Choe et al., 1998a). Similarly, 6α-hydroxycampesterol could also be a starting point for a different subpathway whose intermediates act as ‘bridging molecules’ between the early and late C-6 oxidation pathways (Choe et al., 1998a). Identification of new brassinosteroid biosynthetic intermediates in vivo are needed to confirm whether these hypothesized subpathways exist in plants. One simple explanation for plants having multiple pathways of brassinosteroid biosynthesis is that these subpathways might be differentially regulated by various environmental or developmental signals. Feeding experiments using det2 and dwf4 mutants have consistently shown that brassinosteroids in the late C-6 oxidation pathway are more effective in rescuing light phenotypes whereas the brassinosteroids in the early C-6 oxidation pathways show stronger activity in promoting hypocotyl elongation of dark-grown seedlings (Fujioka et al., 1997; Choe et al., 1998a). Further measurements of endogenous brassinosteroid intermediates of different subpathways and detailed analysis of expression patterns of key brassinosteroid biosynthetic genes under different environmental conditions or at different developmental stages should help to understand the regulation of brassinosteroid biosynthesis.

Metabolic studies with wild-type Arabidopsis and det2 seedlings indicate that the reaction catalysed by DET2 is a major rate-limiting step in the brassinosteroid biosynthetic pathway (S Fujioka, personal communication). Consistent with such an observation, overexpression of DET2 in Arabidopsis results in transgenic plants that are significantly larger than wild-type plants in the light, while underexpression of DET2 using an antisense DET2 construct leads to an allelic series of plants ranging from det2-like dwarfs to wildtype in stature (Chory and Li, 1997). These results support the early claims that brassinosteroid applications could increase crop productivity. Likewise, the 22-hydroxylation reaction has also been hypothesized to be a rate-limiting step (Fujioka et al., 1995). Therefore, DWF4 might be another limiting activity in the biosynthesis of brassinolide (Choe et al., 1998a). Biochemical, metabolic and transgenic studies are needed to confirm whether DWF4 does catalyse a rate-limiting reaction in the brassinosteroid biosynthetic pathway. If so, it will be interesting to see whether overexpression of both DET2 and DWF4 genes will have a great effect on the biomass of transgenic plants.

Analysing the expression of the cloned brassinosteroid biosynthetic genes can provide hints to where brassinosteroids are synthesized and how brassinosteroid biosynthesis is regulated. DET2 appears to be constitutively and ubiquitously expressed throughout Arabidopsis development and in response to different light conditions (Li and Chory, 1997; D Friedrichsen and J Chory, unpublished observations). In contrast, the expression of CPD is confined to cotyledon and leaf primordia in dark-grown seedlings and the adaxial parenchyma of expanding leaves in light-grown plants. CPD expression is not detected in either actively elongating cells of hypocotyls and roots, or pollen and seeds where the highest brassinolide levels have been detected (Mathur et al., 1998). Such an expression pattern of CPD might suggest that brassinosteroids are synthesized in both cotyledons and leaves and are then transported to the organs that require high levels of brassinosteroids for normal growth. Alternatively, there might be enzymatic machineries that reduce the levels of active brassinosteroids to relieve a feedback inhibition on
CPD expression in either cotyledons or expanding leaves. Several different enzyme reactions have been implicated in brassinosteroid inactivation such as hydroxylation, glycosylation, acylation, and side-chain degradation (Fujioji and Sakurai, 1997; Adam et al., 1996). Both the transport and metabolism of 14C-labelled 24-epibrassinolide have been studied in cucumber and wheat seedlings (Fujioji and Sakurai, 1997). When applied to roots, 24-epibrassinolide is readily taken up and transported to leaves; in contrast, when applied to leaves, its transport is quite slow. It was later shown that this compound is quickly metabolized in leaves, but not in hypocotyls and roots. As such, these results are consistent with the second hypothesis.

**Brassinosteroid signal transduction**

Having established the essential role of brassinosteroids in plant growth and development, the most interesting question to ask is how plants recognize these steroids and transduce their signals to regulate a wide spectrum of developmental processes. To understand the molecular mechanism by which brassinosteroids regulate plant development, it is necessary to identify components of the brassinosteroid response pathway, including receptor(s), intermediates, and targets. Currently, both molecular and genetic approaches have been taken to investigate the molecular mechanisms of brassinosteroid signalling.

The molecular approach is to clone brassinosteroid-regulated genes, understand their functions, map their corresponding brassinosteroid-responsive cis-acting elements and identify trans-acting regulatory factors mediating brassinosteroid-regulated gene expression. It has been demonstrated that brassinosteroid-induced responses require de novo protein synthesis (Mandava, 1988) and brassinosteroid-treatment induces synthesis of both mRNAs and proteins (Clouse, 1996). Using a differential hybridization method, a brassinosteroid up-regulated (BRU1) gene was identified from elongating soybean epicotyls (Zurek and Clouse, 1994). BRU1 encodes a protein that showed significant homology to various xylloglucan endotransglycosylases (XETs) which have been implicated in cell wall loosening during cell elongation (Clouse, 1996). A later study proved that BRU1 is indeed a functional XET (Oh et al., 1998). The expression level of BRU1 correlates with the extent of brassinosteroid-promoted stem elongation and the accumulation of the BRU1 transcript parallels the brassinosteroid-mediated increases in plastic extensibility of the cell wall (Zurek and Clouse, 1994; Zurek et al., 1994).

Moreover, a linear relationship has been observed between brassinosteroid concentrations and extractable XET activities in brassinosteroid treated soybean epicotyls (Oh et al., 1998), strongly suggesting an involvement of BRU1 in brassinosteroid-stimulated stem elongation. Surprisingly, brassinosteroid regulates BRU1 expression at a post-transcriptional rather than a transcriptional level (Zurek and Clouse, 1994). A brassinosteroid-regulated XET has also been identified in Arabidopsis (Xu et al., 1995). The TCH4 gene encodes an XET whose expression is increased within 30 min of brassinosteroid treatment, with a maximum at 2 h. In addition to brassinosteroids, this gene is also regulated by touch, darkness, temperature shock and auxin. In contrast to soybean BRU1, the brassinosteroid-regulated TCH4 expression occurs at the transcriptional level (Xu et al., 1995). The brassinosteroid-responsive element has been mapped to a 100 bp fragment of the TCH4 promoter (Clouse and Sasse, 1998) and linker-scanning mutagenesis is underway to identify the specific brassinosteroid response element(s) for this gene.

A recent study has shown that expression of CPD is specifically down-regulated by brassinolide although its transcription is not affected by other plant hormones including auxin, ethylene, gibberellins, cytokinin, jasmonic acid, and salicylic acid (Mathur et al., 1998). Such a brassinosteroid-induced repression of CPD transcription is sensitive to the protein synthesis inhibitor cycloheximide, indicating a requirement for de novo synthesis of a negative transcriptional regulator (Mathur et al., 1998). Mapping of brassinosteroid-response element(s) in the CPD promoter is necessary to find transcriptional factors regulating CPD gene expression.

The genetic approach to the identification of components of the brassinosteroid signalling pathway is the isolation and characterization of Arabidopsis mutants deficient in brassinosteroid responses. Screens for hormone-insensitive mutants in Arabidopsis have proven to be fruitful in the study of plant hormone action. As examples, screens for mutants that are insensitive to high levels of exogenously applied auxins, ethylene, gibberelins, and abscisic acid have led to the identification of loci involved in signalling from these plant hormones. Among the ethylene-insensitive loci are ETR1 and ETR2, which encode ethylene receptors (Chang et al., 1993; Sakai et al., 1998).

An Arabidopsis brassinosteroid-insensitive mutant (bril) was originally identified by a root-growth inhibition assay (Clouse et al., 1996). bril seedlings display similar pleiotropic phenotypes to brassinosteroid-deficient mutants, yet can not be rescued by brassinosteroid treatment. The bril mutants showed marked insensitivity only to brassinosteroids but retain complete sensitivities to auxins, cytokinins, gibberelins, abscisic acid, and ethylene. A second brassinosteroid insensitive mutant cbh2, has been isolated independently in a genetic screen for cabbage-like Arabidopsis mutants (Kauschmann et al., 1996) and was later found to be allelic to bril. Brassinosteroid insensitivity in the cbh2 mutant has also been observed at the molecular level. The brassinosteroid-
induced expression of two XET genes, TCH4 and meri5, is missing in the mutant, although GA-induced meri5 expression is still observed (Kauschmann et al., 1996). A different genetic screen, designed specifically for brassinosteroid-response mutants with brassinosteroid-deficient phenotypes, has also been performed. The screen involved two steps: a primary screen for det2 or cpd-like dwarf mutants followed by a secondary screen for dwarfism that could not be rescued by brassinosteroid treatment. This screen yielded 18 bin (brassinosteroid insensitive) mutants that were later found to be new alleles of the BRI1/CBB2 locus. Recently, three dwf2 alleles were found to be allelic to bri1 (Clouse and Sasse, 1998) and two new bri1 alleles were identified from a collection of dwarf mutants at the Arabidopsis Biological Resource Center (The Ohio State University, Ohio, USA) and a private collection of T-DNA insertion lines (J Li and J. Chory, unpublished data), thus bringing the total number of bri1 alleles to 25. It is somewhat surprising that all brassinosteroid-insensitive mutants isolated in four different genetic screens are all alleles of a single gene. This suggests that BRI1 is the only unique and specific component in the brassinosteroid signalling pathway and other components of the pathway are either redundant (mutants would have no obvious phenotype) or shared with other signalling cascades (mutants would be lethal). Brassinosteroid-insensitive mutants have also been identified in both pea (Ika, Nomura et al., 1997) and tomato (cu-3, Clouse and Sasse, 1998) (Fig. 1).

The almost identical phenotypes between bri1 and cpd, the most severe brassinosteroid-deficient mutant isolated to date, strongly suggests that BRI1 encodes an early component of the brassinosteroid response pathway, most likely a brassinosteroid receptor. Recently, the Arabidopsis BRI1 gene was cloned by chromosome walking (Li and Chory, 1997) and found to encode a protein that has significant sequence identity to a family of plant leucine-rich-repeat (LRR) receptor-like kinases including Xa21 of rice (Song et al., 1995), ERECTA and CLV1 of Arabidopsis (Torr and al., 1996; Clark et al., 1997). The predicted BRI1 protein contains several distinct domains: a signal peptide, a putative leucine-zipper motif, 25 leucine-rich-repeats, a 70 amino-acid island buried between the 21st and 22nd LRR, and a cytoplasmic kinase domain that has serine/threonine kinase activity when expressed in E. coli or animal cells (J Li, C Joazeiro and J Chory, unpublished data).

The finding that BRI1 is a LRR-containing receptor kinase is quite a surprise. First, while a transmembrane receptor kinase is a central theme in many signal transduction events (Ullrich and Schlessinger, 1990), no such receptor kinase has ever been found to be involved in a steroid signal transduction pathway. All known steroid receptors belong to a superfamily of nuclear receptor proteins that are ligand-dependent transcription factors that regulate gene expression (Beato et al., 1995); however, cell surface steroid receptors are known to be present in animal cells mediating non-genomic effects of steroid hormones (McEwen, 1991) and protein tyrosine phosphorylation has been implicated in such a membrane-initiated steroid signalling pathway (Tesarik et al., 1993; Mendoza et al., 1995).

Second, while many LRR-containing proteins are involved in signal transduction pathways, LRRs are believed to mediate protein–protein interactions and, in many cases, provide binding sites for protein ligands (Kobe and Deisenhofer, 1994). So far, no LRR has ever been found to interact with small compounds. Interestingly enough, the predicted BRI1 protein contains a unique 70 amino-acid island buried between the 21st and 22nd LRRs. The importance of this island in BRI1’s function is self-evident by sequencing two bri1 alleles, both of which have a missense mutation in this domain (Li and Chory, 1997; J Li and J. Chory, unpublished results). In contrast, of 19 bri1 alleles that have been sequenced, no allele contains a mutation in the LRRs themselves. It is worthwhile to note that mutations in LRRs have been identified in both erecta and clv1 alleles (Torri et al., 1996; Clark et al., 1997). Careful biochemical studies are needed to determine whether BRI1 is a brassinosteroid receptor, and if so, whether the island is directly involved in binding brassinosteroids.

BRI1’s kinase domain must be essential for the BRI1’s function. Of the 19 bri1 alleles sequenced to date, 15 have mutations in this domain (Li and Chory, 1997; J Li and J. Chory, unpublished results). This domain must interact with other downstream signalling components to transduce the steroid signal. One protein that might interact with BRI1’s kinase domain is a type 2C phosphatase (KAPP) which has been implicated to be involved in both RLK5 and CLV1-mediated signalling pathways (Stone et al., 1994; Williams et al., 1997). Indeed, a fusion protein between glutathione S-transferase (GST) and BRI1’s kinase domain interacts with this phosphatase in vitro (J Li and J. Chory, unpublished results). Transgenic experiments are underway to find the physiological relevance of such an interaction.

Conclusion and perspectives

To understand completely how brassinosteroids act in plants, more components of the brassinosteroid response pathways need to be isolated and characterized. It might be possible to identify proteins involved in brassinosteroid signal transduction that directly interact with BRI1’s kinase domain by a yeast two-hybrid screening or interactive cloning strategy. Although four different genetic screenings have resulted in the identification of only one brassinosteroid signalling component, BRI1, at least two other genetic screens can be used to identify new compon-
ents involved in brassinosteroid signal transduction. One is a transgene-based screening using a well-characterized brassinosteroid-regulated promoter fused to a reporter gene. Such a genetic screen has been used successfully to identify mutants affecting phytochrome responses and chloroplast-nuclear communication (Li et al., 1994, 1995; Susak et al., 1993). The other screen is to identify mutants that suppress or enhance either brassinosteroid-deficient or brassinosteroid-response mutations. Results from these studies will certainly expand our knowledge of how steriods regulate plant development. A better understanding of the brassinosteroid signal transduction pathway will make it possible to manipulate plant growth genetically in order to improve crop productivity.

Acknowledgements

Work on brassinosteroids in our laboratory is supported by the United States Department of Agriculture. JC is an investigator of the Howard Hughes Medical Institute and JL is an American Cancer Society postdoctoral fellow.

References


