RESEARCH COMMUNICATION

MAX4 and RMS1 are orthologous dioxygenase-like genes that regulate shoot branching in Arabidopsis and pea

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Shoot branching is inhibited by auxin transported down the stem from the shoot apex.Auxin does not accumulate in inhibited buds and so must act indirectly. We show that mutations in the MAX4 gene of Arabidopsis result in increased and auxin-resistant bud growth.Increased branching in max4 shoots is restored to wild type by grafting to wild-type rootstocks, suggesting that MAX4 is required to produce a mobile branch-inhibiting signal, acting downstream of auxin. A similar role has been proposed for the pea gene, RMS1. Accordingly, MAX4 and RMS1 were found to encode orthologous, auxin-inducible members of the polyene dioxygenase family.

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Variation in shoot branching is an important cause of diversity in plant form. Individual species have a characteristic branching pattern, which can change through the life cycle in response to developmental cues and to environmental conditions (Cline 1991; Beveridge et al. 2003). Branching control therefore requires the integration of many signals, both known and unknown.

Shoot branches arise from axillary meristems that form in the axils of leaves on the primary shoot axis. The axillary meristems themselves initiate leaves to form a bud. Bud growth can arrest but has the potential to reactivate to produce a shoot branch. Removal of the primary shoot apex results in activation of arrested axillary buds. The ability of the shoot apex to repress axillary bud growth is termed apical dominance. Thimann and Skoog (1933) reported that a compound, derived from the shoot apex, and later identified as auxin (indole-3-acetic acid), could inhibit the growth of lateral buds when applied to the stump of a decapitated plant. Subsequent work has provided multiple lines of evidence in support of auxin-mediated bud inhibition in planta. However, a second messenger must relay the auxin signal into the bud because apically derived auxin is not transported into buds (Morris 1977) and exogenous auxin applied directly to buds does not inhibit their growth (Cline 1996).

One model proposes that the effect of auxin on bud growth is mediated by cytokinin. Cytokinin can directly promote bud growth (Cline 1991); transgenic plants with increased auxin levels have reduced cytokinin levels (Ek-löf et al. 2000), and cytokinin export from roots increases after decapitation, with this increase being abolished by application of auxin to the decapitated stump (Bangerth 1994). However, there is also good evidence for novel regulators of bud growth downstream of auxin. The ramosus mutants (rms1 to rms5) of pea (for reviews, see Beveridge 2000; Beveridge et al. 2003) have increased lateral branching, but this phenotype can be almost completely rescued by grafting a wild-type (WT) rootstock to an rms1, rms2, or rms5 mutant scion. Such grafting studies show that RMS1 and RMS5 are required for the production of a graft transmissible signal that moves from root to shoot and inhibits branching (Foo et al. 2001; Morris et al. 2001). This mobile signal is unlikely to be auxin or cytokinin because, as well as increased branching, the rms1 and rms5 mutants have reduced root-derived cytokinin and have at least WT auxin levels and transport (Beveridge et al. 1997; Morris et al. 2001). This is exactly the opposite of the prediction for a bushy plant and may be the result of feedback regulation of auxin and cytokinin levels. It is possible that the RMS1/RMS5-dependent long distance signal is a second messenger for auxin. The lateral buds of rms1 shoots can only respond to the inhibitory effects of apical auxin when grafted to WT rootstocks (Beveridge et al. 2000).

To identify genes that regulate bud growth in Arabidopsis, we screened mutagenized populations for plants with increased branching and have identified four loci, mutations at which result in more axillary growth, named max1 to max4 (Stirnberg et al. 2002). In this paper, we describe the phenotype of the max4 mutant and the cloning of the MAX4 gene, and show that this gene is orthologous to RMS1.

Results and Discussion

Isolation and genetic characterization of the max4 mutants

We have identified a class of Arabidopsis mutants with more axillary branches and placed them in four complementation groups named max1 to max4 (Stirnberg et al. 2002). Four independent recessive alleles were found at the MAX4 locus in the Columbia [Col] ecotype. Two alleles (max4-1 and max4-2) were isolated from the Sainsbury Laboratory Arabidopsis Transposant (SLAT) collection [Tissier et al. 1999], and two alleles (max4-3
and max4-4) were isolated from the AMAZE population (Wisman et al. 1998). There were no apparent differences in the severity of phenotype conferred by these alleles, and the max4-1 allele was chosen for detailed phenotypic analysis, following two rounds of back-crossing to WT.

The max4-1 mutant has increased shoot branching

Mature max4-1 mutant plants grown under a 16-h long-day photoperiod have a bushy appearance at maturity as a result of increased growth of the buds in rosette leaf axils (Fig. 1A). In WT plants, all the cauline nodes and the most apical rosette nodes produced buds that developed into lateral inflorescences and, on average, the uppermost 5.1 ± 0.2 rosette nodes produced an elongated inflorescence >4 mm in length (Fig. 1B). A basipetal gradient of inflorescence length was observed, with branches arising in the youngest leaf axils having the greatest mean branch lengths. This gradient continued into older rosette nodes, where buds remained smaller than 4 mm in length. In most plants (n = 7/10) a weak acropetal gradient of bud growth was also observed, although these buds remained very small throughout the life of the plant. Between the acropetal and basipetal gradients were one to three nodes that carried tiny buds, or no bud visible to the naked eye (data not shown).

As in WT, all the cauline nodes of mature max4-1 plants produced buds that developed into elongating inflorescences with the same basipetal gradient (Fig. 1A). However, a greater number of rosette buds developed into inflorescences compared with WT, on average 8.5 ± 0.4 (Fig. 1B). An acropetal gradient of bud growth was also observed in most of the max4-1 plants (n = 8/10), but in contrast to WT, these buds grew out to form short inflorescences. Therefore, both the basipetal and acropetal patterns of bud activity are similar to WT in long-day-grown max4-1 mutants, but the mutant buds are more likely to grow out.

We found no evidence that, unlike the supershoot mutant (Reintanz et al. 2001; Tantikanjana et al. 2001), but similar to the Arabidopsis max1 and max2 mutants (Stirnberg et al. 2001) and the axr1 auxin-resistant mutant (Stirnberg et al. 1999), the max4 mutations affect the number of axillary meristems formed at each node. Rather, the defect appears to be specifically in bud outgrowth. Furthermore, like max1, max2, and axr1, max4 affects both the acropetal and basipetal gradients in a similar way.

MAX4 expression in the roots is sufficient for WT shoot branching

To determine the site of action of MAX4, we performed reciprocal hypocotyl grafting experiments (Turnbull et al. 2002). The self-grafted control plants reproduced branching phenotypes similar to intact controls, indicating that the grafting process does not affect branching (data not shown). Graft combinations with either a WT scion or a WT rootstock showed WT shoot branching patterns (Fig. 1C), indicating that, although the MAX4 gene can act in the shoot to inhibit branching, expression in the root is sufficient for WT shoot branching levels. These data suggest that the MAX4 gene is required for the production of a graft-transmissible inhibitor of shoot branching.

Auxin responses in the max4-1 mutant

We assayed the response of max4-1 axillary buds to auxin using a split plate assay (Chatfield et al. 2000). Bud outgrowth from excised cauline nodes placed between divided agar sections in a Petri dish is inhibited by applied auxin. In this system, without hormone treatment, buds of WT and max4-1 grew out with similar kinetics, with elongation commencing 2 d after node excision (Fig. 2A). Apical auxin inhibited WT bud outgrowth for an average of 6 d, whereas max4-1 mutant buds were partially resistant to apical auxin, being inhibited for an average of only 4 d.
Molecular characterization of the MAX4 gene

The max4-1 and max4-2 lines were found to contain single transposon insertions that cosegregated with the mutant phenotypes (data not shown). DNA flanking the transposon in max4-1 and max4-2 was isolated by inverse PCR and both amplified fragments were found to be identical in sequence to parts of the same predicted gene (At4g32810; Fig. 3). When At4g32810 was sequenced from the max4-3 and max4-4 alleles, it was found to contain a four-base insertion and a two-base deletion, respectively [Fig. 3]. These changes are predicted to result in premature termination of the encoded protein and are consistent with transposon excision footprints (Cardon et al. 1993). The fact that four independent max4 alleles have mutations in this one gene provides strong evidence that this is the MAX4 gene.

MAX4 belongs to the polyene chain dioxygenase family

Database searches using the predicted protein sequence of MAX4 (Altschul et al. 1990) show that it is a member of the polyene chain dioxygenase superfamily, and is likely to be localized to plastids (Emanuelsson et al. 2000). A phylogenetic analysis of family members from plants, animals, and bacteria is shown in Figure 4A [constructed using ClustalX 1.8 program; Thompson et al. 1997]. MAX4, RMS1 [see below], and a rice sequence (OsMAX4) form a well-supported clade. The abscisic acid (ABA) biosynthetic protein, VP14, falls within another strongly supported clade that includes Arabidopsis carotenoid-cleaving dioxygenases (CCDs, also called NCEDs) probably involved in ABA biosynthesis (AtCCD2, AtCCD3, AtCCD5, AtCCD6, and AtCCD9; for review, see Seo and Koshiba 2002). The representatives of the animal RPE65, BETA DIOX1 and BETA DIOX2 proteins, form a third well-supported clade; and a fourth discrete group of proteins with similarity to bacterial lignostibene dioxygenases is represented here by two Sphingomonas paucimobilis proteins. The AtCCD1 protein, which is not involved in ABA biosynthesis but cleaves beta carotene [Schwartz et al. 2001], and AtCCD7 and AtMAX4 are grouped on long branches.

These results suggest the attractive hypothesis that MAX4 encodes a carotenoid-cleaving dioxygenase involved in the synthesis of a mobile branch-inhibiting substance. This substance is very unlikely to be ABA because of the lack of ABA-related phenotypes in max4 mutants [data not shown] and the lack of requirement for ABA in auxin-mediated inhibition of bud outgrowth (Chatfield et al. 2000). Taken together, these data support the hypothesis that MAX4 is involved in the syn-
thesis of a novel hormone that acts downstream of auxin to inhibit shoot branching.

The MAX4 gene is orthologous to the RMS1 gene of pea

The similar phenotypes conferred by max4 and the pea rms1 mutants prompted us to test whether any of the RMS genes were orthologous to MAX4. We isolated the pea ortholog of MAX4 using degenerate primers based on amino acid alignment between MAX4 and the deduced amino acid sequences from two Medicago truncatula ESTs showing high homology with MAX4 [60% (77/127) and 62% (60/96) identity]. A pea gene was isolated encoding a 561 amino acid protein showing 68% identity with MAX4 across the 518 amino acids at the C terminus. Comparison of the RT–PCR-amplified cDNA and the corresponding genomic DNA revealed five introns for PsMAX4 at the same position as in the Arabidopsis gene and of comparable sizes (740/918; 263/122; 88/110; 78/91; 70/89).

We mapped the PsMAX4 sequence using a recombinant inbred line mapping population (Laucou et al. 1998). PsMAX4 was found to map to the top of linkage group III at the same position as RMS1 (Blixt 1976). In an F2 population of 95 individuals [M3T-884 x Tordag] segregating for rms1, complete cosegregation was observed between PsMAX4 and RMS1. Southern analysis using PsMAX4 as a probe revealed a 12-kb band present for WT progenitors Weitor and Raman and absent for mutant alleles rms1-2 and rms1-3 [Fig. 4B]. The map position of PsMAX4, its deletion in two independent rms1 alleles, and the similar phenotypes of max4 and rms1 mutants provide strong evidence that RMS1 and MAX4 are true orthologs.

Expression of the MAX4 and RMS1 genes

Transcripts for MAX4 and RMS1 are present at very low levels and are not readily detectable on Northern blots. However, they can be amplified by PCR from all tissues tested [data not shown]. These widely distributed but very low expression levels for MAX4 transcripts are supported by Affymetrix gene chip data, publically available from the Nottingham Arabidopsis Stock Centre. To determine more precisely the location of MAX4 transcription, we constructed promoter–GUS fusions using 2.7 kb of DNA upstream of the MAX4 translational start site. This construct was introduced into WT Col plants, and GUS expression was analyzed using the chromogenic substrate X-Gluc. Strong expression was consistently observed in root tips [Fig. 5A]. Other tissues such as hypocotyls and petioles occasionally showed very weak expression in some plants from each line [data not shown]. Similarly, weak expression was variably observed in nodal sections associated with young axillary buds [Fig. 5C]. No GUS staining was observed in the buds themselves, consistent with the remote site of action of MAX4 predicted by the grafting studies.

Because MAX4 and RMS1 are predicted to act downstream of auxin, we tested whether the transcription of either gene is affected by auxin. We used RT–PCR to investigate the expression of the RMS1 gene in pea using the classical apical dominance test involving decapitation and replacement of the apex by exogenous auxin [Fig. 5D]. Decapitation caused a substantial drop
Shoot branching in Arabidopsis and pea

Materials and methods

Plant growth conditions
Arabidopsis plants were grown in 4-cm square compartments (P40, Cookson Plantpak) containing F2 compost treated with Intercept 70WG [both Levington Horticulture] in a growth room with 16 h light (white light at 70 µmole/m² s⁻¹). Pea seedlings were grown under glasshouse conditions extended to 18 h light (incandescent light providing -3 µmole/ m² s⁻¹ at pot top), as described by Morris et al. [2001].

Phylogenetic analysis
Sequences for phylogenetic analysis were retrieved from the NCBI database [see Supplemental Material]. Sequences were aligned with ClustalX software [Thompson et al. 1997]. The alignment was analyzed by eye, and regions with a low confidence of alignment were removed using Bioedit software [Hall 1999]. The phylogenetic tree was generated using Neighbour Joining and a distance matrix with correction for multiple substitutions. Bootstrapping values were generated with n = 10,000. TreeView [http://taxonomy.zoology.gla.ac.uk/rod/treeview.html] was used to visualize the tree.

Gene cloning
The MAX4 gene was isolated using inverse PCR [iPCR] of max4-1 and max4-2 DNA [see Supplemental Material for details]. The CMV35S: MAX4 cdNA construct was made using standard techniques [Sambrook et al. 1989, Supplemental Material]. The MAX4 promoter–GUS fusion construct was generated by PCR amplification of a 2.7-kb region upstream of the ATG including BamH1 and XbaI restriction sites in the primers. These sites were used to clone the fragment into the pBI101.1 vector. This plasmid was transformed into Agrobacterium and then into plants using the floral dip method of Clough and Bent [1998]. Two typical lines containing a single site of transgene insertion were taken to homozygosity and used for detailed analysis.

Isolation of the pea MAX4 homolog PsMAX4
Degenerate primers were designed in consensus regions between the Arabidopsis MAX4 protein and the deduced amino acid sequence of two Medicago truncatula genes showing high homology with MAX4 [see Supplemental Material]. These primers were used to amplify PsMAX4 from both cdNA and genomic DNA. The amplification products were used to determine the sequence of the pea cdNA and the positions of introns in the gene [Brunel et al. 1999].

Mapping of PsMAX4
For mapping the PsMAX4 sequence, a cleaved amplified polymorphic sequence (CAPS) marker was designed to detect a DraI restriction site polymorphism between the two parents of our mapping population of 139 recombinant inbred lines [Lauco et al. 1998], Tére'se and line K586 isogenic to Torsdag, see Supplemental Material]. This CAPS marker was also used for cosegregation analysis between PsMAX4 and the pea branching gene RMS1 in a population of 95 F2 individuals from a cross between the rms1-10 mutant line M3T-884, obtained from Tére'se by EMS mutagenesis [Rameau et al. 1997; Symons and Murtet 1997], and Torsdag. Linkage analysis was carried out using the MAPMAKER/EXP 3.0 computer program [Lincoln et al. 1992]. Southern blot analysis of the two radiation-induced rms alleles was carried out using standard techniques [see Supplemental Material].

PsMAX4 expression study in pea
Fourteen-day-old cv. Torsdag plants [five leaves expanded, counting acropetally from the cotyledonary node as zero] were left intact or were decapitated below the apex (internode 5) and IAA in lanolin was applied to the decapitated stump, as described by Beveridge et al. [2000]. Six hours after treatment, internode 4 was collected. Total RNA was extracted using a modification of the hot-phenol method [Kreig 1996]. RT-PCR and Southern blotting were used to determine the abundance of RMS1 transcripts in these samples. For details, see Supplemental Material.

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Figure 5. Root tip expression of the GUS reporter protein driven by a 2.7 kb of DNA upstream of the MAX4 gene. (A) Untreated 5-day-old seedlings. (B) Seedlings transferred to 1 µM 1-NAA for 24 h. (C) Nodal section showing expression close to a young bud, but not in the bud. (D, top panel) RMS1 gene expression in internode 4 of 14-day-old plants, with 5 leaves expanded, that were intact [I] or 6 h after decapitation [D], treated with 0, 500, or 3000 mg/L⁻¹ IAA to the decapitated stump. [Bottom panel] Act gene expression was monitored as a control.

in RMS1 expression within 6 h after treatment. This reduction was not only prevented by replacement of the apex with 500 and 3000 mg/L exogenous IAA, but RMS1 expression was up-regulated compared with intact controls. In contrast, similar experiments in Arabidopsis using both RT–PCR and the promoter::GUS reporter lines failed to detect any up-regulation of MAX4 transcript levels in stem sections in response to apical auxin [data not shown]. However, 24 h after transfer of promoter::GUS seedlings to auxin-containing media [1 µM NAA], up-regulation of GUS expression was observed in regions of the root distal to the apparently constitutive root tip expression pattern [Fig. 5B]. No such up-regulation was detected following 6 h exposure [data not shown].

These data suggest that auxin may regulate shoot branching partly through transcriptional up-regulation of RMS1/MAX4. In pea, this up-regulation occurs at the node and may be sufficiently rapid to inhibit bud growth in response to apical auxin. In Arabidopsis, however, no up-regulation was detected at the node, despite the fact that this is a major site for AXR1 and auxin action in the regulation of bud growth [Booker et al. 2003]. This, combined with the observation that MAX4 action in the root is sufficient for WT branching, suggests an additional role for auxin downstream of the RMS1/ MAX4-dependent signal. For example, auxin may regulate the transport of the RMS1/MAX4-dependent signal into axillary buds. A full test for this hypothesis awaits the identification of the novel MAX4/RMS1 dependent, branch-inhibiting signal.
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