RESEARCH ARTICLE

Transposon Tagging of the Defective embryo and meristems Gene of Tomato

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The shoot and root apical meristems (SAMs and RAMs, respectively) of higher plants are mechanistically and structurally similar. This has led previously to the suggestion that the SAM and RAM represent modifications of a fundamentally homologous plan of organization. Despite recent interest in plant development, especially in the areas of meristem regulation, genes specifically required for the function of both the SAM and RAM have not yet been identified. Here, we report on a novel gene, Defective embryo and meristems (Dem), of tomato. This gene is required for the correct organization of shoot apical tissues of developing embryos, SAM development, and correct cell division patterns and meristem maintenance in roots. Dem was cloned using transposon tagging and shown to encode a novel protein of 72 kDa with significant homology to YNV2, a protein of unknown function of Saccharomyces cerevisiae. Dem is expressed in root and shoot meristems and organ primordia but not in callus. The expression pattern of Dem mRNA in combination with the dem mutant phenotype suggests that Dem plays an important role within apical meristems.

INTRODUCTION

In plants, organogenesis is continuous and occurs in apices throughout the entire life cycle. This process is achieved by the action of apical meristems, which are groups of stem cells that are established early in embryogenesis and maintained in the tips of shoots and roots. Because apical meristems are almost entirely responsible for the elaboration of plant architecture, they have been a major subject of observational, experimental, and genetic studies (described in Steeves and Sussex, 1991; Meyerowitz, 1997). We are now beginning to elucidate the genes involved in meristem regulation and to understand their function (Meyerowitz, 1997).

In angiosperms, the shoot apical meristem (SAM) is usually a small dome of cells that consists of a peripheral zone in which leaves are initiated and a central zone in which the peripheral zone cells are replenished. The central zone contains cells that divide slowly, whereas the peripheral zone contains cells that divide rapidly (Lyndon, 1990; Steeves and Sussex, 1991). Superimposed upon this zonation are three clonally distinct cell layers (Poethig, 1987): L1 (forming the epidermis), L2 (forming the mesoderms), and L3 (forming the pith and vascular tissue). These cell layers generate the whole shoot. The L1 and L2 layers in the SAM are maintained by anticlinal cell divisions. Occasional cell divisions occur that result in the insertion of cells derived from one layer into the adjacent layer. These cells adopt a fate appropriate to their new layer, thus suggesting that positional information, rather than cell lineage, is the major factor influencing cell fate decisions during plant development. How cells in meristems communicate with each other has not yet been determined; however, recent results indicate roles for protein trafficking (Lucas et al., 1995) and extracellular signaling (Clark et al., 1997).

The root apical meristem (RAM), in contrast to the SAM, is an internal area of cells and is responsible for the production of cells for both the root and the root cap. The RAM is therefore surrounded on all sides by its derivatives. At the center of the root meristem is a region of cells known as the quiescent center—a population of cells that has a very long generation time. Surrounding the quiescent center are initials cells, which divide more rapidly and whose progeny differentiate into the basic cell types of the root and root cap. The cells of the quiescent center are proposed to act as replacements for the more rapidly dividing apical initials. Cell division patterns within the Arabidopsis root are almost invariant, which results in a root comprised of several clonally distinct...
files of cells (Dolan et al., 1993). Lateral roots are not initiated at the root apex but rather are initiated from an internal layer of cells called the pericycle. Experimental evidence suggests that the root tip inhibits the formation of lateral roots (McCully, 1975).

Despite their differences, the basic organization of the SAM and RAM is similar: both meristems are layered structures that contain a central zone of quiescent or slowly dividing cells. In addition, experiments using surgically isolated meristems have shown that the SAM and RAM are autonomous in their development (Ball, 1952; Feldman and Torrey, 1976). These observations have led to the conclusion (Steeves and Sussex, 1991) that the differences between the SAM and RAM are superimposed upon a fundamentally homologous plan of organization and that the root and shoot systems probably represent evolutionary modifications of an “ancestral meristem” in response to different environments. Mutations that specifically affect both the SAM and RAM may therefore represent lesions in genes whose functions have been conserved throughout the evolution of apical meristems from the ancestral meristem.

In this study, we describe a recessive mutant of tomato, defective embryo and meristems (dem), that is affected in the development of both shoot and root apical meristems. Dem was cloned by using the transposable element Dissociation (Ds) as a tag and shown to encode a novel protein with a region of significant homology to a yeast protein of unknown function. Dem is expressed in SAMs and RAMs, axillary meristems, and organ primordia during adult plant growth. Although the exact function of Dem remains unclear, our initial observations suggest that it plays an important role within apical meristems and organ primordia.

RESULTS

**dem Mutants Have Disrupted Apical Meristems**

A total of 150 families carrying independent transpositions of the maize transposon Ds in tomato were generated. Approximately 25 seeds from each family were sown in flats, and seedlings were screened for mutant phenotypes. dem mutants were found in one family, N174 (Figure 1A). Testcross and F₂ analysis showed that the mutation was recessive and that mutant progeny occurred at a frequency of 10 to 15%. Self-pollination of heterozygotes revealed that the dem mutants had a highly variable number of small, slightly concave, abnormal cotyledons and no SAM. Wild-type seedlings were normally dicot (Figure 1B). Of 110 mutants inspected, two were monocot, 20 were dicot, 65 were tricot, and 23 were tetracot (Figures 1C to 1F).

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**Figure 1.** Seedling and Embryo Morphology Is Disrupted by the dem Mutation.  
(A) Three-week-old dem and wild-type (wt) plants. dem plants have neither elongated roots nor a shoot.  
(B) Wild-type dicot seedling.  
(C) dem monocot.  
(D) dem dicot.  
(E) dem tricot.  
(F) dem tetracot.
Figure 2. *dem* Seedlings Have No Apical Meristem.

(A) and (B) SEM of the SAM (sam) and a cotyledon (c) of a wild-type seedling and the cotyledon and shoot apical region of a *dem* mutant, respectively. One cotyledon has been cut off in (A) and (B) to facilitate viewing. Bar in (A) = 150 μm; bar in (B) = 719 μm.

(C) An expanded view of the *dem* apical region boxed in (B). Bar = 76 μm.

(D) Section through the wild-type shoot apex.

(E) Section through the *dem* shoot apex. No typical SAM can be seen. The adaxial tissues of the cotyledons are disorganized.

(F) SEM of a *dem* root, showing a lateral root (LR) and root tip (RT). Bar = 712 μm.

(G) Section through the wild-type root apex, showing a typical root meristem.

(H) Section through the *dem* root apex, showing that the *dem* root meristem (boxed) is disorganized.
Apical growth of dem seedlings was terminated soon after germination, and no true leaves were initiated (Figure 2). Scanning electron microscopy (SEM) studies (Figures 2A to 2C) showed that the apical region between the cotyledons of dicot dem seedlings usually contained no SAM or leaf primordia. Sections through dem apices (Figures 2D and 2E) confirmed that no organized SAM was present in dem seedlings but rather that tissue with a disorganized cell arrangement formed. This disorganization continued from the axis of the cotyledons into the adaxial half of the cotyledons. Cell organization in the abaxial half of cotyledons appeared to be normal. dem roots terminated after 3 or 4 mm of growth, and lateral roots, which also aborted after a short period of extension (Figure 2F), were initiated. dem roots were also very hairy; however, it is not possible to predict whether this is a direct effect of the mutation. Sections through a dem root show that although many of the outer cell files are correctly maintained, cells in the center of the root apex are disorganized compared with the wild type (boxed in Figures 2G and 2H). No clear cell files were observed in the central cylinder of a dem root.

Isolation of the Dem Gene by Transposon Tagging

Sequences flanking the Ds element in a dem mutant were cloned using inverse polymerase chain reaction (IPCR) (Thomas et al., 1994) and sequenced. Using this sequence, two primers, dem3' and dem5', were designed. When used in combination with primer B34 (Thomas et al., 1994), they could be used to map the Ds element in relation to the dem phenotype. In tests of 200 individuals of a segregating population using triplex PCR, Ds was found to segregate with the dem phenotype (Figure 3A), demonstrating close linkage between the mutant phenotype and a Ds insertion. A transposase source, stabilized Activator (sAc), was crossed onto a dem heterozygote, and an F1 plant containing both sAc and Ds was self-pollinated. Approximately 75% of the dem

Figure 3. Linkage of Ds to the dem Mutation and Somatic Reversion of dem.

(A) Linkage of the Ds insertion to the dem mutation was demonstrated using a PCR zygosity test: a 220-bp fragment was amplified from the preinsertion allele, and a 154-bp fragment was amplified from the Ds insertion allele. PCR with DNA from stable mutant seedlings only produced a 154-bp fragment, indicating that these seedlings are homozygous for the Ds insertion and that the Ds is closely linked to the dem mutation. Wild-type plants were either heterozygous or homozygous for the preinsertion allele. NT, untransformed; WT, wild type.
(B) and (C) Transposase-dependent somatic reversion of the dem phenotype confirmed that the Ds insertion is the cause of the dem mutation. For further details, see Methods and Results. Somatic revertants also initiated leaves that could not develop properly. mut, mutant; wt, wild type; C1, C2, and C3 indicate cotyledons.
mutants in this segregating population contained sAc. After a period of up to 2 months, all of these mutants reverted and formed shoots from between their cotyledons. These shoots were either fully wild type or chimeric. The chimeric shoots contained both wild-type and mutant tissues and were rather unusual in appearance (Figures 3B and 3C). After a period of time, chimeric shoots became fully wild type in appearance. In contrast, dem mutants that did not contain sAc never formed shoots, even after several months. The shoots of somatic revertants yielded fruit that contained viable seed. Seeds from somatic revertants were planted, and seedlings were scored for the dem phenotype: the majority of these seedlings were wild type, demonstrating that the dem mutation is germinally unstable in the presence of the transposase gene. The close linkage of Ds with the dem phenotype and the sAc-dependent somatic and germline instability of the dem phenotype strongly implicate Ds as the cause of the dem mutation.

An 8-bp target site duplication is typical of Ds insertion, and many Ds excision alleles retain this duplication or have deletions/substitutions of one or two nucleotides (Sauder and Nevers, 1985). To confirm that the dem mutation was caused by a Ds insertion, DNA from germlinal revertants was prepared, and the sequence alterations expected from Ds excision were analyzed. All sequenced Dem revertant alleles contained sequence alterations consistent with Ds excision (Figure 4A). This result confirms that the dem mutation is a result of a Ds insertion into the dem locus. The Ds insertion allele of dem was designated demPs.

During the course of the analysis of germlinal revertants, a sAc– Ds– plant was identified that gave rise to ~10% mutant progeny. This allele of dem was later sequenced and found to contain a 7-bp insertion at the Ds insertion site that causes an early frameshift in the Dem open reading frame (ORF). This allele was designated dem–2. Plants homozygous for dem–2 displayed a phenotype identical to demDs, demonstrating that demPs is probably a null allele. The phenotypic analysis described above was performed with mutants homozygous for demPs.

In a separate experiment, dem–7 heterozygotes containing sAc were crossed onto demPs heterozygotes, and several somatic revertants were identified. DNA was extracted from wild-type tissues of these mutants. The sequences surrounding the site of Ds insertion were amplified by polymerase chain reaction (PCR) using oligonucleotides dem3′ and dem5′, with one being kinase labeled. PCR products were then size fractionated by PAGE. All revertant alleles represented either perfect excision events or insertions/deletions of +3, +6, or –3 nucleotides (Figure 4B). These sequence alterations restored the Dem reading frame and resulted in the addition or loss of one or two amino acids in the Dem protein. In one case, a deletion of 18 nucleotides (leading to a deletion of six amino acids in the Dem protein) was identified. These results are consistent with the idea that Ds insertion occurred in the Dem coding sequence and that only excision events that do not alter the reading frame will reinstate wild-type gene function. Amino acid residues around this area are therefore not essential for the function of the Dem protein.

PCR tests showed that demPs is fully transmitted through male and female gametes. The observed segregation distortion (10 to 15% mutant rather than 25% mutant) is due to decreased viability of dem embryos (M.E.C. Reyes and B.J. Carroll, unpublished data). DNA gel blotting experiments using low- and high-stringency washes demonstrated that Dem is present as a single copy in the tomato genome (Figure 4C).

Dem Encodes a Novel Protein

Cloning and sequencing of the flanking DNA of this mutant line revealed that the Ds element had inserted in a large ORF. The cloned flanking sequences were used to screen a cDNA library. One full-length Dem cDNA clone was isolated and sequenced (Figure 5; GenBank accession number...
Y13632). This cDNA contained one long ORF with an in-frame stop codon in the 5’ leader sequence. Translation of the ORF predicted a charged protein of 71,919 D with a pI of 5.58 (Figure 5).

A search of the PROSITE database showed that the predicted mature N-terminal sequence of Dem, MGANHS, conforms to the consensus sequence for N-myristoylation, suggesting that Dem may be attached by a lipid anchor to a cellular membrane. BLAST (Altschul et al., 1997) searches using the Dem peptide sequence identified two potentially homologous proteins (Figure 6A): CYPRO4 from artichoke thistle (GenBank accession number P40781; 93% identical and 98% similar; P = 5.4 × 10^{-26}) and YNV2 from Saccharomyces cerevisiae (GenBank accession number P40157; 33% identical and 51% similar over 150 residues; P = 1.7 × 10^{-11}). Both proteins are of unknown function. Two Arabidopsis expressed sequence tags (F19919 and N96644) with strong homology to the 3’ and 5’ ends of Dem were also identified (Figure 6B).

**DISCUSSION**

Dem is expressed in all regions of the plant in which organized cell divisions take place. These regions include apical meristems, organ primordia, and leaflet primordia. However, Dem is not expressed in callus. Furthermore, loss of Dem function causes disorganization of both the shoot and root apex and in the adaxial tissues of cotyledons. These observations suggest that Dem is required for the organization or maintenance of meristems and primordia. Dem mutants are morphologically distinct from those previously reported to be affected in basic body planning (Jurgens et al., 1991; Mayer et al., 1991), SAM development (Caruso, 1968; Meyerowitz, 1997), and root development (Benfey and Schiefelbein, 1994) and may represent a novel category of mutants that are affected in a basic aspect of meristem regulation.
The shoot apex of dem embryos lacks a shoot meristem and has a highly variable number of cotyledons that contain disorganized cells. During normal embryogenesis, two cotyledons are initiated on the apical flanks of a globular stage embryo, and the SAM becomes morphologically apparent between the emerging cotyledons (Jurgens et al., 1991; Mayer et al., 1991). In this context, we foresee a role for Dem in the organized cell divisions that accompany the transition between the globular and heart stages of embryogenesis. Without correct cell divisions, the normal signals that coordinate cotyledon number and apical development may be perturbed. This would lead to a deregulation of cotyledon number, altered cell division planes, and the failure to form or maintain a SAM. Because the true relationship between the cotyledons and the SAM in wild-type plants is not known (Kaplan, 1969; Barton and Poethig, 1993; Endrizzi et al., 1996), it is impossible to predict whether the SAM simply is not established in dem embryos or is established but not maintained.

In the dem root apex, cell divisions within a central zone of cells are disorganized, and several of the central cell files in the root are not correctly formed. dem roots terminate after a short period of growth but have the ability to initiate determinate lateral roots. The simplest explanation of the dem root phenotype is that the dem mutation makes roots determinate; primary roots and lateral roots can be formed, but they cannot be maintained. The fact that both terminal and lateral root meristems are determinate in dem mutants suggests that the dem SAM may also be determinate. These observations suggest that Dem may play a role in meristem maintenance and that the dem SAM is consumed in embryogenesis during the formation of cotyledons.

A possible clue for Dem function comes from the observation that two other mutants with altered cotyledon number have altered hormone levels. The Arabidopsis mutant pinoid (Bennett et al., 1995) frequently produces tricot seedlings and has defects in auxin transport. The mutant altered meristem program1 also has a highly variable cotyledon number and altered cytokinin levels. Furthermore, polar auxin transport has been shown to be critical for pattern formation during embryogenesis (Liu et al., 1993), leaf and floral organ phyllotaxy (Meicenheimer, 1981; Okada et al., 1991), compound leaf development (Avasarala et al., 1996), meristem maintenance (Avasarala et al., 1996), and root meristem organization (Kerk and Feldman, 1995). Thus, Dem may be involved in cellular responses to hormone gradients that organize all apices and organ primordia.
Figure 8. In Situ Distribution of Dem mRNA.

(A) and (B) SAM and cotyledons of 12-day-old seedlings. Signal was observed in the meristem and leaf primordia, using antisense (A) but not sense (B) Dem probes.

(C) to (E) SAM and stem from 4-week-old plants. Signal was observed in meristems and leaf primordia, using antisense (C) and (D) but not sense (E) probes.

(F) Cross-section showing Dem expression in the adaxial tissues of young leaves.

(G) and (H) Dem expression was detected in leaflet primordia, using sense (G) but not antisense (H) probes.

(I) Stem/cotyledon axis. Dem expression is detected in axillary meristems.

(J) and (K) Cross-section through inflorescence showing an emerging floral meristem and developing flowers. Staining is observed in floral meristems and in organ primordia as they emerge, using Dem antisense (J) but not Dem sense (K) probes.

(L) and (M) Sections through root tips. Staining was observed in root tips, using antisense (L) but not sense (M) probes.

Digoxigenin labeling is visible as brown staining. All hybridizations used 10-μm-thick sections and digoxigenin-labeled Dem probe. ab, abaxial; ad, adaxial; am, axillary meristem; c, cotyledon; fm, floral meristem; llp, leaflet primordium; lp, leaf primordia; m, meristem; pp, petal primordium; s, stem; se, sepal; sp, sepal primordium; st, stamen primordium.
A notable feature of the expression pattern of Dem, at least in shoot apices, is that it is apparently coincident with the expression of tKn1, a gene encoding a KNOTTED1-related homeodomain protein of tomato (Hareven et al., 1996). KNOTTED1-related proteins are believed to maintain cells in an undifferentiated state within meristems (Smith et al., 1992) and in the leaf and leaflet primordia of tomato (Hareven et al., 1996). Similar to Dem, knotted1-related genes of maize are not expressed in callus tissue (Smith et al., 1992) and are expressed in vascular strands (Smith et al., 1992; Jackson et al., 1994). Also, in Arabidopsis, mutations in STM, a Knotted1 homolog, result in seedlings with no apparent SAM (Long et al., 1996). These observations suggest that Dem may be required for correct cell division patterns within the domain of Knotted expression.

In summary, we have identified a mutant, dem, that plays an important role in the maintenance or function of both the SAM and RAM. We have cloned the Dem gene by transposon tagging and shown that it is expressed in all areas of the plant in which organized cell division is taking place. The conceptual translation of the Dem cDNA provides little evidence regarding the function of the Dem protein. The lack of apparent nuclear localization sequences or DNA binding motifs suggests that it is not a nuclear transcription factor. The presence of myristoylation consensus motifs makes it tempting to speculate that Dem may be anchored to a cellular membrane. The homology of Dem to a yeast protein raises the possibility that Dem is a cellular component that has evolved to become an essential gene for organized cell divisions that occur in meristems and primordia during plant development.

**METHODS**

**Transgenic Plant Material and Generation of the defective embryo and meristems Mutant**

Transgenic tomato (Lycopersicon esculentum) cultivar Moneymaker carrying maize transposable elements was used for all experiments. A total of 150 transposants was generated from a single Dissociation (Ds) T-DNA line (1561E) by selection for excision and reinsertion of Ds after testcrossing a 1561E/10512I double heterozygote to wild-type plants (Carroll et al., 1995). The 10512I line carries the transposase gene (sAc) linked to β-glucuronidase (GUS). Seedlings carrying a transposed Ds were self-pollinated, and the progeny were screened for mutations. Family N174 carries a single transposed Ds and includes mutants exhibiting the defective embryo and meristems (dem) phenotype.

**Reversion of the dem Mutant in the Presence of the Transposase**

To demonstrate instability of dem in the presence of a transposase, a Dem heterozygote was crossed to the transposase line 10512I (Carroll et al., 1995). F2 double heterozygotes for the Ds insertion and the transposase gene were identified by a polymerase chain reaction (PCR) test (identifying demDs; see below) and histochemical staining for GUS (the marker for the transposase gene). F2 double heterozygotes were selfed, and the F3 generation was screened for GUS-positive mutant seedlings. GUS-positive mutants were observed for somatic instability of the mutant phenotype. Somatic F2 revertants were testcrossed to an untransformed tester, and the progeny were screened for germinal wild-type and mutant excision alleles at the Dem locus, as described below.

**Cloning the Dem cDNA**

Fragments of the Dem gene were cloned by inverse PCR (iPCR) (Thomas et al., 1994) and used to screen a λgt10 cDNA library constructed using seedling mRNA. We purified six positives from 5 × 107 plaques, and one full-length Dem cDNA was sequenced on both strands. RNA and genomic DNA extraction and analysis were performed as described previously (Keddie et al., 1996).

**PCR Test for Ds Zygosity at the Dem Locus**

The mutant line was maintained as a heterozygote. To detect zygosity for the Ds insertion in Dem, we developed a simple triplex PCR test (Thomas et al., 1994) with intact leaf tissue (Klimyk et al., 1993; Carroll et al., 1995). Based on the sequences flanking both sides of the Ds in dem, oligonucleotide primers dem5’ (5’-TTTTGTCCTCTTATTTATGCTTTGAG-3’) and dem3’ (5’-TTTACATGTTGTTGGAACACTGCGA-3’) were designed to amplify a 220-bp preinsertion fragment. dem5’, in combination with primer B34 (5’-ACGTCGTACGGATTTCCCAT-3’), which primes from sequences at the end of Ds, amplifies a 154-bp fragment corresponding to the Ds insertion in the dem gene. By using PCR with these three primers, we performed zygosity tests for the Ds insertion in Dem on individual seedlings.

**PCR Footprint Analysis of Dem Revertants**

Footprint analysis was done using oligonucleotides dem3’ and dem5’. PCR products from wild-type and germinal revertant plants were cloned and sequenced. In addition, a screen for new excision alleles was performed by crossing sAc+ dem17 heterozygotes with demDs heterozygotes. The seeds from this cross were germinated, and wild-type plants were discarded. After ~1 month, ~50% of the mutants initiated a shoot from between their cotyledons, and growth was resumed. To analyze the size of footprints left in new dem alleles, either dem3’ or dem5’ was kinase labeled with γ-labeled [3P]-ATP and used with the other primer to amplify excision alleles. PCR products were denatured and separated on a 6% polyacrylamide gel (Figure 4B).

**Microscopy and in Situ Hybridization**

Samples for light microscopy were prepared using a microwave procedure. Tissue was fixed twice in formaldehyde acetic acid (FAA) at 37°C for a total of 30 min, dehydrated at 67°C in 70% ethanol and then 100% ethanol for 75 sec each, treated in 2-propanol at 75°C for 90 sec, and then embedded in molten Paraplast (Pelco, Reading, CA) at 67°C for ~3 hr in a 3440 MAX Laboratory microwave (Pelco). A full version of this protocol can be obtained from the National Science
Foundation Center home page (www.plantbio.berkeley.edu). Samples were serially sectioned, stained in safranin O and orange gold to highlight densely cytoplasmic cells, and viewed on an Axioshot microscope (Carl Zeiss, Inc., Thornwood, NY). Samples prepared for scanning electron microscopy (SEM) were fixed in FAA, dehydrated in ethanol, dried in a critical point dryer, sputter coated with palladium to 20 nm, and viewed on a SIS 130 scanning electron microscope (ISI, Philadelphia, PA).

In situ RNA hybridization was performed using methods described by Coen et al. (1990). An internal 559-bp EcoRI fragment of the Dem cDNA was subcloned into pBluescript SK+ (Stratagene, La Jolla, CA). T7- and T3-primed digoxigenin-labeled RNA probes were made using digoxigenin RNA labeling mix (Boehringer Mannheim) and hydrolyzed at 60°C for 30 min in 100 mM carbonate buffer, pH 10.2. A minimum of three samples were examined per experiment, and sense strand controls were always included.

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