Shoot Meristem Function and Leaf Polarity: The Role of Class III HD–ZIP Genes

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ABSTRACT

The shoot apical meristem comprises an organized cluster of cells with a central region population of self-maintaining stem cells providing peripheral region cells that are recruited to form differentiated lateral organs. Leaves, the principal lateral organ of the shoot, develop as polar structures typically with distinct dorsoventrality. Interdependent interactions between the meristem and developing leaf provide essential cues that serve both to maintain the meristem and to pattern dorsoventrality in the initiating leaf. A key component of both processes are the class III HD–ZIP genes. Current findings are defining the developmental role of members of this family and are identifying multiple mechanisms controlling expression of these genes.

Introduction

A hallmark of land plant evolution has been development of the leaf. Leaves are the principal organ for capture of energy from sunlight and conversion, through photosynthesis, into organic components for growth. In angiosperms, leaves are typically planar, dorsoventrally flattened structures. Dorsoventrality is specified early in development of primordia. In the initiating leaf, the dorsal, or adaxial, side is immediately adjacent to the shoot apical meristem, whereas the ventral, or abaxial, side is farther from the shoot meristem (Figure 1A and 1B). In the mature leaf, the adaxial side is usually the upper sun-exposed side of the leaf and the abaxial side is the lower shaded side of the leaf.

A series of surgical experiments carried out in the 1950s, and elaborated upon more recently, were the initial key to mechanisms that establish leaf dorsoventral patterning [1–3]. Separation of initiating primordia from the meristem by surgical incision generated a radial, abaxial leaf. This suggested, firstly, lateral organ patterning required an interaction between the initiating organ and the shoot apical meristem and, secondly, that in the absence of this interaction loss of dorsoventrality resulted in radial organs.

Markers of Dorsoventrality

The extent to which adaxial and abaxial sides of a mature leaf can be distinguished varies between species; however, in developmental model species such as the dicot Arabidopsis or the monocot maize, many cell types differentiate top from bottom [9–11]. In Arabidopsis, epidermal cells on both sides of the leaf are jigsaw-shaped, but adaxial cells are larger and uniform in size relative to variable abaxial cell size. Trichome density is a useful marker on early juvenile leaves, where adaxial trichomes are much more frequent than abaxial trichomes. In subepidermal cell layers, closely aligned elongate palisade mesophyll cells lie juxtaposed to the adaxial epidermis. Less closely spaced, larger spongy mesophyll cells form abaxial internal tissue (Figure 1C). The palisade and spongy mesophyll tissues are organized for light capture and gas exchange, respectively. Vasculature is also patterned in the dorsoventral dimension with xylem, the water-conducting tissue, adaxial to the organic nutrient–conducting phloem tissue (Figure 1D). Vascular bundles within the stem are also patterned with xylem more central to peripheral phloem. Conceptually, the dorsoventral vascular patterning of the leaf can be translated into a collateral central–peripheral pattern within the stem.

Editor: Nicholas Katsanis, Johns Hopkins University School of Medicine, United States of America

Citation: Byrne ME (2006) Shoot meristem function and leaf polarity: The role of Class III HD–ZIP genes. PLoS Genet 2(6): e89. DOI: 10.1371/journal.pgen.0020089

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Abbreviations: HD–ZIP, homeodomain–leucine zipper; phan, phantastica; se and SE, SERRATE; START, sterol/lipid binding domain Mary E. Byrne is at the Department of Crop Genetics, John Innes Centre, Norwich Research Park, Norwich, United Kingdom. E-mail: mary.bym@bbrc.ac.uk
defined tissue-specific expression patterns within the embryo. (B) Developing vegetative leaf of Arabidopsis. Adaxial trichomes on the larger leaf are one marker distinguishing dorsoventrality. Scale bar is 250 μm. (B) Developing vegetative leaf of Arabidopsis. Adaxial trichomes on the larger leaf are one marker distinguishing dorsoventrality. Scale bar is 250 μm. (C) Diagramatic representation of a leaf cross section with adaxial and abaxial outer epidermal and inner mesophyll marked. (D) Cross section of leaf midvein with adaxial xylem and abaxial phloem cells marked.

**Class III HD–ZIP Genes—The Arabidopsis Family**

Class III HD–ZIP transcription factors have in common a homeodomain DNA binding motif and a leucine zipper dimerization motif (HD-ZIP), and are a subset of a much larger group of plant proteins that also include a sterol/lipid binding (START) domain [12,13] (Figure 2A). Although lipid ligands for a small number of START domain proteins have been identified in animals, none to date have been found for plant START proteins. There are five Class III HD–ZIP genes in Arabidopsis, each encoding a protein in the range of 833–852 amino acids, and sharing between 60% to 85% amino acid homology (Figure 2B). PHABULOSA (PHB) and PHAVOLUTA (PHV) are most closely related to one another, sharing 85% amino acid identity [14]. Likewise, ATHB8 and ATHB15 form a relatively closely related pair, sharing 75% amino acid identity [15]. ATHB15 has also been published under the name CORONA (CNA) [16,17], REVOLUTA (REV), in some previous work published as INTERFASICULAR FIBERLESS1 (IFL1), shares between 60% and 66% amino acid identity with other members of the group [18–20].

**Patterned Expression**

All five Class III HD–ZIP genes in Arabidopsis have well-defined tissue-specific expression patterns within the embryo and shoot. There are several principal reports describing tissue specific expression patterns, as determined by in situ hybridization, for REV [15,18,20], PHB [14], and PHV [15]. For comparative analysis, expression patterns in embryo and shoot for all five HD–ZIP genes have also been reported [17].

**Loss-of-Function Effects**

A visible gross plant phenotype for loss-of-function mutations in Class III HD–ZIP genes has only been described for REV, as redundancy seems to mask the role of other family members in development [18–20,24]. REV mutants have a diverse range of phenotypes. One of the most prominent defects is a failure in initiation and development of secondary meristems in the axis of vegetative rosette and cauline leaves, resulting in plants with few branches (Figure 2C). Flowers display variable phenotypes and may lack organs or form only rudimentary tapered filaments. In the inflorescence stem, there is a loss of xylem and interfascicular layers interconnecting vascular bundles. Despite this loss, there is no defect in vascular dorsoventral patterning.

Loss of PHB and PHV, either as single mutants or together in a double mutant, has no evident phenotypic consequences. However, mutations in both genes enhance rev defects, indicating PHB and PHV are redundant with each other and also with REV [15,17]. Depending on the background, loss of all three genes results in either partially abaxial cotyledons without a shoot apical meristem or a single abaxial cotyledon, or, in the extreme, failure to correctly pattern embryo development. Additional loss of ATHB15 in this background increases the frequency of the more severe phenotype. These patterning defects coincide with the expression pattern of these genes and are consistent with loss of both organ adaxial patterning and shoot central patterning within the embryo.
All HD–ZIP genes function in postembryonic development. Mutations in both PHB and PHV enhance shoot phenotypes of rev [17]. Like PHB and PHV, loss of either ATHB8 or ATHB15 or combined loss of both of these genes has no gross phenotypic effect, although vascular development is slightly perturbed in athb15 mutants. However, mutations in ATHB8 and ATHB15 suppress axillary and floral meristem defects of rev mutants. Together, genetic interactions suggest overlapping redundant as well as competitive interactions between these genes in development [17].

**Dominant Effects**

Mutations in PHB were first reported as temperature-sensitive, semidominant mutants with radial, adaxialized leaves and enlarged meristems [25]. Ectopic meristems surround the adaxial leaf as would be expected if adaxial fate promotes meristem formation or, alternatively, if PHB independently promotes meristem formation. Like PHB, gain-of-function mutations in REV, as in rev-10d and the allele amphivasal vascular bundle 1 (avb1), result in stem fasciation indicative of an increase in meristem size, adaxialized leaves, and conversion of normally collateral vasculature of the stem, where xylem is central to peripheral phloem to amphivasal vasculature with the xylem surrounding phloem [15,26]. All of these phenotypes are not always evident even with identical mutations and genetic background, suggesting that growth conditions may influence expressivity of the phenotype.

All dominant mutations in Class III HD–ZIP genes occur within a defined region around the 3' end of the fourth exon and 5' end of the fifth exon (Figure 3). An allelic series of phb-d mutants revealed either a point mutation at a splice site, resulting in a small percentage of transcripts with a short peptide sequence insertion, or point mutations resulting in an amino acid change [14]. Multiple dominant alleles of phv and rev also have single nucleotide changes within the same region as found in phb-d alleles [14,15,26]. However, as discussed below, changes in amino acid sequence in the dominant mutants do not appear to be significant as the site
of all these mutations is within the predicted binding site for the small regulatory microRNAs, miR165 and miR166 [27,28].

**Regulation by microRNAs**

MicroRNAs are approximately 21 nucleotides in length and are generated from longer precursor transcripts. The precursor transcripts not only form a hairpin loop structure that is recognized and cleaved into a double stranded form carrying the microRNA and a complementary sequence. Ultimately, the microRNA as a single strand is guided to the target transcript, a process involving the small RNA binding proteins of the ARGONAUTE family. Subsequently, target transcripts are either cleaved within the region binding the microRNA or are subject to translational inhibition. Much more detail on this pathway and the genes involved can be found in reviews on the subject [29–31].

The *Arabidopsis* genome encodes two copies of miR165 and seven copies of miR166, which have near perfect match with a sequence conserved within the transcript of all *Arabidopsis* Class III HD–ZIP genes [27] (Figure 3). So far, regulation of Class III HD–ZIP genes by microRNAs appears to occur by transcript cleavage, as is typical for many plant genes targeted by microRNAs. Several lines of evidence support this model [15,26,32,33]. Firstly, REV and PHB cDNA constructs that alter the microRNA binding site but not the corresponding amino acid sequence induce the same phenotypes as the gain-of-function mutations in these genes. Secondly, transcript cleavage products can be generated in in vitro assays and can be detected in plant extracts. Thirdly, in *phb-1D* mutants, the mutant PHB transcript is ectopically expressed and occurs throughout the leaf, whereas constitutive expression of wild-type PHB, using a 35S promoter, does not, in general, induce a phenotype. Although constitutive expression of the Class III HD–ZIP genes does not result in a phenotype, there are two interesting exceptions. Overexpression of PHB did result in adaxialization of early leaves in a small proportion of transformants [33] and overexpression of *ATHB8* induces a phenotype with an increase in vascular xylem [34]. Occasional overexpression phenotypes may reflect dosage-dependent effects between microRNAs and target transcripts.

As might be expected, overexpression of microRNAs results in a reduction in Class III HD–ZIP gene transcripts. Two semidominant mutants, *meristem enlarged1* (*men1*) and *jabba-1D* (*jab-1D*), isolated from activation tagged lines of *Arabidopsis*, have increased levels of *miR166a* and *miR166g*, respectively [35,36]. Although homozygous *men1* are seedling lethal, *men1/+* plants have a range of phenotypes including an enlarged shoot apical meristem and slight downcurling of leaves suggesting weak abaxialization of lateral organs [35]. Meristem, leaf patterning, and vascular defects also appear in the *jab-1D* mutant, and the severity of these phenotypes is stronger in homozygous compared with heterozygous plants [36]. In both *men1/+* and *jab-1D*, overexpression of different miR166 genes has differential effects on expression of individual members of the Class III HD–ZIP genes. In *jab-1D* mutants, PHB, PHV, and *ATHB15* are downregulated. However, REV is upregulated and this accounts for adaxialization of leaves but not meristem defects. Again, differential effects of miR165/166 on Class III HD–ZIP genes may be a consequence of dosage-dependent interactions between microRNA and target and the degree to which expression patterns of these two overlap. This in turn is potentially influenced by regulatory interactions between the different Class III HD–ZIP genes.

**Regulation of Regulators**

One gene involved in microRNA-mediated regulation of PHB is *ARGONATE1* (*AGO1*). *AGO1* is a key component of RNA-mediated gene silencing. In plants, *AGO1* binds microRNAs and is sufficient to mediate cleavage of target transcripts [37,38]. Mutations in *ago1* disrupt many aspects of development, including organ dorsoventral patterning. In plants carrying strong *ago1* mutant alleles, lateral organ development is severely affected, leading to a number of disparate interpretations of the phenotype [39–41]. It is possible that both adaxial and abaxial fates are affected by *ago1* such that leaves in severely affected plants have lost polarity. However, in strong *ago1* mutants PHB expression is expanded throughout the leaf and weak *ago1* alleles have leaf development defects consistent with adaxialization of the leaf, indicating that one role of *AGO1* is regulation of PHB via microRNAs [41].

Another component implicated in microRNA-mediated regulation of PHB is *SERRATE* (*SE*). Mutations in *SE* have a number of effects on shoot development including time to flowering, meristem size, leaf serrations, and dorsoventral patterning defects [42–45]. In severe mutants, leaves and leaf vasculature are adaxialized. se mutants have increased levels of expression of several Class III HD–ZIP genes. In the case of PHB, the domain of expression is expanded to the abaxial side of the leaf and the level of PHB is increased in the abaxial side of the leaf, similar to the pattern of misexpression in the dominant phb-1D mutants. The Class III HD–ZIP genes are likely secondarily affected by *SE* because the level of *miR166* is greatly reduced in *se* mutants [45]. Concomitant with a reduction in *miR166*, the level of the precursor transcript for *miR166* is increased in *se* mutants indicating that *SE* may function in processing miRNA precursor transcripts. *SE*
Dominant mutations in PHB and mutations in AGO1 and SE all result in PHB misexpression with increased levels of transcript in the adaxial domain and ectopic expression in the abaxial domain of young leaf primordia. Changes in adaxial and abaxial expression in these backgrounds suggest miRNAs regulating PHB expression are expressed throughout early leaf primordia. One report shows miR163/166 expression throughout the Arabidopsis shoot [46]. However two other reports, in Arabidopsis and in maize, indicate that these microRNAs are spatially restricted in the shoot apex and may function in patterning [41,47]. A further report examining expression in the embryo indicates dynamic expression throughout development, but in early embryogenesis miR166 is initially abaxial and at the distal tips of initiating cotyledons, an expression pattern closely complementary to that of the HD-ZIP genes [36]. Resolution of the degree to which miR163/166 and HD-ZIP target genes overlap may come from analysis of the expression pattern of sensor constructs where a cell-autonomous reporter gene carries the miRNA target binding site [48]. To add to this picture, Class III HD-ZIP tissue-specific expression appears to be directed simply by the promoter [21,22,49], in which case microRNAs may serve to modulate expression levels, to be directed simply by the promoter [21,22,49], in which case microRNAs may serve to modulate expression levels, particularly at boundaries where regulator and target expression overlap.

The LOB-domain gene ASYMMETRIC LEAVES2, which is required in leaf patterning, appears to negatively regulate miR165. Levels of miR165 are further increased in mutants lacking both ASYMMETRIC LEAVES2 and the RNA silencing pathway gene RDR6, an RNA-dependant RNA polymerase [46]. However, regulation of miR165 by ASYMMETRIC LEAVES2 and RDR6 is likely to be indirect as RDR6 acts in a pathway with three genes, AGO7, SGS3, and DCL4, known to regulate production of a specific class of small RNAs known as trans-acting siRNA [50–55]. Downstream targets of these trans-siRNA pathway genes are ETTIN and ARF3, two AUXIN RESPONSE FACTOR genes required for abaxial fate [50,56,57].

Linking to Chromatin

Aside from cleavage, microRNA targeting to the PHB transcript also influences the methylation status of the PHB locus [58]. High levels of methylation are usually associated with transcriptionally inactive chromatin, and, conversely, low methylation levels are typically associated with transcriptionally active chromatin. In wild-type plants the PHB locus is methylated at the 3’ end of the gene, although methylation levels are low in meristem-enriched tissues where PHB is expressed. The dominant allele, which may no longer effectively bind miR163/166, fails to mediate methylation of the PHB gene. Thus miR163/166 may function, directly or indirectly, in transcriptional as well as posttranscriptional regulation of PHB. The PHV gene is also methylated in the 3’ region indicating Class III HD-ZIP genes as a whole may be subject to multiple levels of regulation. The significance of this regulatory system is yet to be established, but multiple levels of regulation may serve several purposes. Conceivably, microRNA-directed cleavage of transcripts acts as an efficient mechanism for rapid inactivation of transcripts within a cell. Simultaneous or subsequent methylation of the locus would then maintain a stable repressed state in cells where gene expression is no longer required.

Conclusions

The Class III HD-ZIP genes play multiple, possibly interdependent, roles in plant development. Conservation of these genes and expression patterns throughout land plants, in particular in lower land plant species, highlight a critical role in development of the basic plant body [59]. Spatial, temporal, and quantitative regulation of expression appears to involve a number of mechanisms including posttranscriptional and transcriptional gene silencing mediated by microRNAs. The importance of microRNAs as regulators of this gene family is reflected in conservation of miR166 and conservation of Class III HD-ZIP gene function in divergent plant species [27,47,60,61]. An additional layer of regulation may involve modulation of function via a sterol-type ligand. Evaluation of the contribution and interplay of these regulatory mechanisms and the degree to which components of regulation are conserved are clearly going to be subjects of much future research.

Supporting Information

Accession Numbers

Accession numbers from the Genbank genebank (http://www.ncbi.nlm.nih.gov/Genebank) are for: PHABULOSA (PHB), 2G34710; PHAVOLUTA (PHV), 1G50450; ATHB8, 4G32880; ATHB15, 1G52150; and REVOLUTA (REV), 5G60090.

Acknowledgments

I thank Catherine Kidner and Robert Martienssen for critical reading of the manuscript and Peter Etchells for the figure of vasculature. My apologies to colleagues whose research I failed to adequately cite due to space limitations, in particular to those whose work moves beyond the bounds of Arabidopsis.

Funding. MEB is a recipient of a Royal Society Wolfson Merit Award, and her laboratory is funded by the Biotechnology and Biological Sciences Research Council, United Kingdom.

Competing interests. The author has declared that no competing interests exist.

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