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Review

BLADE-ON-PETIOLE genes: Setting boundaries in development and defense

Madiha Khan, Huasong Xu, Shelley R. Hepworth*

Department of Biology, Carleton University, 1125 Colonel By Drive, Ottawa, Ontario, Canada K1S 5B6

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ABSTRACT

BLADE-ON-PETIOLE (*BOP*) genes encode an ancient and conserved subclade of BTB-ankryin transcriptional co-activators, divergent in the NPR1 family of plant defense regulators. Arabidopsis BOP1/2 were originally characterized as regulators of leaf and floral patterning. Recent investigation of BOP activity in a variety of land plants provides a more complete picture of their conserved functions at lateral organ boundaries in the determination of leaf, flower, inflorescence, and root nodule architecture. BOPs exert their function in part through promotion of lateral organ boundary genes including *ASYMMETRIC LEAVES2*, *KNOTTED1-LIKE FROM ARABIDOPSIS6*, and *ARABIDOPSIS THALIANA HOMEOBOX GENE1* whose products restrict growth, promote differentiation, and antagonize meristem activity in various developmental contexts. Mutually antagonistic interactions between BOP and meristem factors are important in maintaining a border between meristem-organ compartments and in controlling irreversible transitions in cell fate associated with differentiation. We also examine intriguing new evidence for BOP function in plant defense. Comparisons to NPR1 highlight previously unexplored mechanisms for co-ordination of development and defense in land plants.

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* Corresponding author. Tel.: +1 613 5202600; fax: +1 613 5203539.
E-mail address: shelley_hepworth@carleton.ca (S.R. Hepworth).

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1. Overview

BTB-ankryin proteins are plant-specific transcriptional co-activators. They are so called because of two conserved protein-protein interaction motifs: a BTB/POZ (for Broad Complex, Tramtrack, and Bric-a-brac/POX virus and Zinc finger) domain at the N-terminus and four ankryin motifs near the C-terminus. The Arabidopsis (*Arabidopsis thaliana*) genome encodes six BTB-ankryin proteins with functions in development and defense (Fig. 1A).

The first BTB-ankyrin protein to be characterized was Arabidopsis NON-EXPRESSOR OF PATHOGENESIS-RELATED GENES1 (NPR1) based on loss-of-function mutations that abolished systemic acquired resistance, a salicylic acid-induced broad-spectrum defense against pathogens [1,2]. NPR3 and NPR4 are paralogs that act primarily to modulate NPR1 abundance for appropriate deployment of systemic acquired resistance [2,3]. NPR2 has a minor role in salicylic acid perception [4]. BLADE-ON-PETIOLE1 (BOP1) and BOP2 comprise a separate subclade in the phylogenetic tree (Fig. 1A). Redundant developmental functions for these genes were reported by three independent groups. *BOP1* was identified through isolation of a *bop1-1* dominant-negative mutant with leafy petioles [5,6] and also based on characterization of an activation-tagged line with enlarged floral bracts [7]. We used a reverse genetics approach to identify *bop1 bop2* defects in leaf and floral patterning and abscission [8]. The connection between these diverse phenotypes is now starting to emerge.

Meta-analysis of sequence data suggests BTB-ankryin proteins originated prior to the emergence of land plants. Whereas the genomes of Chlorophyte green algae, including *Chlamydomonas* and *Volvox*, do not encode BTB-ankryin proteins, all land plants with sequenced genomes including primitive mosses encode homologs of both NPR1 and BOP1/2 (Table S1; [9]). Diversification in mosses suggests that the pairing of BTB and ankyrin domains happened earlier, presumably within a Charophyte algal lineage that gave rise to land plants [10]. The ancestral BTB-ankryin protein may have held functions in both defense and development.

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In this review, we summarize recent advances providing insight into the conserved function and mechanism of BOP transcription factors in plant development, drawing on studies in Arabidopsis, moss, tobacco, and legumes. We also examine new evidence of a role for BOPs in disease resistance induced by methyl jasmonate.

2. Developmental function in a basal land plant

A glimpse into the ancient developmental function of *BOP* genes comes from their characterization in a moss, *Physcomitrella patens*, used as a model for basal land plants [11]. During the juvenile phase of its development, germination of a haploid spore produces a linear array of cells that branch and form a filamentous network called a protonema. Meristematic cells at tip of the protonema divide and extend the network. The first filaments consist of green chloronemal cells. As the plant matures, apical cells in the filament tip transition from chloronemata to caulonemata fate in an auxin-dependent manner. Apical cells at the tip of young caulonemata side branches are selectively responsive to cytokinin, which promotes their differentiation into a reproductive bud meristem. Division of the bud produces the gametophore, an inflorescence-like shoot with leaflets at the base and male (atheridia) and female (archegonia) reproductive organs at the apex [11,12]. PpBOP gain-of-function accelerates the transition to reproductive development by promoting bud meristem formation [11].

The *Physcomitrella* genome contains three *BOP* genes (Table S1; [11]). The abundance of *PpBOP1* and *PpBOP2* transcript is negatively regulated by a moss-specific microRNA (miRNA) *Pp-miR534a* that directs cleavage near the junction between the third and fourth ankryin repeats [11]. Disruption of the *Pp-miR534a* genomic locus stabilizes *PpBOP1/2* transcripts in caulonemata branch tips causing premature budding. *Pp-miR534a* and *PpBOP1/2* have inverse patterns of expression and opposite responses to cytokinin. Whereas *Pp-miR534a* is primarily transcribed in protonemata and the young gametophore, *PpBOP:GUS* expression accumulates in dividing caulonemata branch tips that have bud-forming competence [11]. *miRNA534a:GUS* expression is depleted in caulonemata side tips supplied with cytokinin whereas expression of *PpBOP:GUS* is enhanced and accelerated. These data show that cytokinin promotes *PpBOP1/2* transcript accumulation in caulonemata branch tips and accelerates reproductive phase transition [11]. BOPs in angiosperms have a similar role in promotion of the floral meristem (see Section 5.1; [7,13,14]).

Moss *BOPs* may also have a role in patterning the gametophore, based on *PpBOP1/2* expression at the base of young antheridia and apex of axillary hairs that originate from the base of leaflets. Lack of morphological defects in *ΔPpbop1 ΔPpbop2* double mutants suggests functional redundancy with *PpBOP3* whose mRNA is predicted to be stable due to a mismatch in the *miRNA* recognition site [11]. Confirmation of a patterning role for moss BOPs awaits construction of the triple mutant.

3. Lateral organ boundaries

BOP genes in seed plants have now been studied in the eudicots Arabidopsis, tobacco, pea, and the model legume, *Medicago truncatula*, affording a more detailed understanding of their activities, which predominate at lateral organ boundaries.

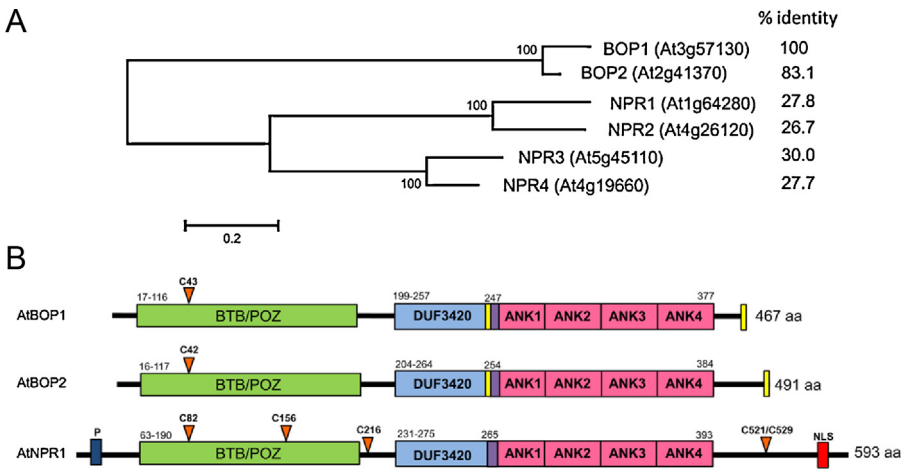

Fig. 1. Phylogenetic tree and structural domains of Arabidopsis BTB-ankryin proteins. (A) Phylogenetic tree. Protein sequences were aligned using ClustalW. A maximum likelihood tree was generated with Mega 5.2.1 (www.megasoftware.net). Branch lengths are proportional to the amount of inferred evolutionary change. Bootstrap values were calculated from 100 replicates. Arabidopsis genome identifier numbers are as indicated. Percent amino acid identities were based on ClustalO output (www.ebi.ac.uk/Tools/msa/clustalO). (B) Comparison of NPR1 and BOP1/2 domain structures. Green, BTB/POZ domain (Pfam: 00651); Blue, DUF3420 domain (Pfam: 11900); Pink, ankryin (ANK) domain (Pfam: 00023). Purple, represents overlap between DUF3420 and the first ANK domain. Yellow, polyhistidine tract; Orange triangles, regulatory Cys residues based on NPR1 [130,148,149]; Blue, P, NPR1 phosphorylation at serine 11/15 is required to promote its rapid turnover during systemic acquired resistance [154]; Red, NLS, nuclear localization signal [128].

Lateral organ boundaries are specialized junctions in the plant that separate emerging lateral organs from the meristem or plant body [15,16]. All of the aerial parts of a plant, including the leaves, shoots, and internodes are generated by the shoot apical meristem. As differentiating lateral organ primordia emerge from the meristem, they are partitioned from surrounding undifferentiated cells by a narrow band of small, cytoplasmically dense cells with a slow rate of cell division. These characteristics facilitate the formation of a groove that divides emerging lateral organs from the meristem [15]. Partitioning creates three distinct transcriptional compartments in the shoot apex: the meristem, the primordia, and the boundary, whose borders are maintained by complex genetic interactions [16].

3.1. BOP expression in developing embryos

BOP1/2 expression at lateral organ boundaries is established during embryogenesis shortly after initiation of the shoot apical meristem. Lateral organ boundary identity is governed by NAC (NO APICAL MERISTEM, ARABIDOPSIS THALIANA ACTIVATING FACTOR1, 2 and CUP-SHAPED COTYLEDON2 (CUC2)) domain transcription factors. In Arabidopsis, *CUC1*, *CUC2*, and *CUC3* function redundantly in this role [16]. *CUC1* and *CUC2* expressed in globular-stage embryos are essential in establishing the embryonic shoot apical meristem through activation of the class I KNOTTED1-like homeobox (*KNOX*) gene *SHOOT MERISTEMLESS* (*STM*) [17]. STM in the meristem controls the position of the boundary by restricting *CUC* expression to the axil of cotyledons in conjunction with auxin-based signals from the embryonic leaf [16,17]. *BOP1/2* are activated in embryos at torpedo stage and are confined by late torpedo stage to the axil of cotyledons, the leaf-meristem boundary ([6,18], Khan and Hepworth, unpublished data). Post-germination, *BOP1/2* are transiently expressed in leaf primordia and restricted to the boundary when primordia first appear as morphologically distinct from the meristem [7,13,18]. This pattern suggests that cues from the meristem and the emerging leaf confine *BOP* expression to the boundary. Microarray data indicate that induction of STM in seedlings promotes *BOP2* expression [19]. In *stm* mutants, *BOP1/2* expression expands to the junction between the fused cotyledons showing that STM spatially restricts *BOP* expression to the lateral organ boundaries in embryos [20]. Although the precise relationship between *BOP1/2* and *CUC* genes is yet unclear, BOP1/2 probably function downstream and/or in parallel with *CUC* genes given their slightly later activation in developing embryos. There is no evidence of change in expression of *CUC* genes in *bop1 bop2* mutants, nor are organ separation defects enhanced in *bop1 bop2 cuc* triple mutant seedlings (Hepworth lab, unpublished data). These findings suggest that BOP1/2 do not play a primary role in establishment of the initial boundary.

3.2. BOP expression at lateral boundaries

The expression patterns of Arabidopsis *BOP1/2* define them as markers of the lateral organ boundary [6–8,13,18,21]. While the boundary has an initial function in separating emerging lateral organs from the meristem and adjacent primorida, the subsequent patterning of boundaries or "junctions" in the plant is often overlooked as a determinant of architectural diversity. Axillary meristems that produce lateral branches and flowers and allow recovery from injury arise from the boundary as do specialized organs such as stipules (appendages at the base of leaves) and nectaries (secretory organs at the base of floral organs). Lateral organ boundaries at defined positions are specialized in detachment or release: abscission mediates the detachment of spent or diseased organs from the plant body whereas dehiscence mediates the release of seeds and pollen from fruit and anther compartments, respectively [15,16].

3.3. Functional redundancy

Most species contain two or three *BOP* orthologs (Table S1). Arabidopsis *BOP1* is expressed at lower levels than *BOP2*, but segregation analyses show that their functions are redundant [8]. Single *bop1* loss-of-function mutants develop an occasional petiole-borne leaflet suggesting a slightly greater requirement for BOP1 in leaves [6,7,22]. Stem-leaf fusions during bolting are seen occasionally in single *bop2* loss-of-function mutants, suggesting a slightly greater requirement for BOP2 in reproductive development [22]. Orthologs in most species show greater amino acid similarity to BOP2 suggesting that its requirement is more universal (Table S1).

Loss-of-function studies were instrumental in establishing that BOP activities promote the fate and determinacy of axillary

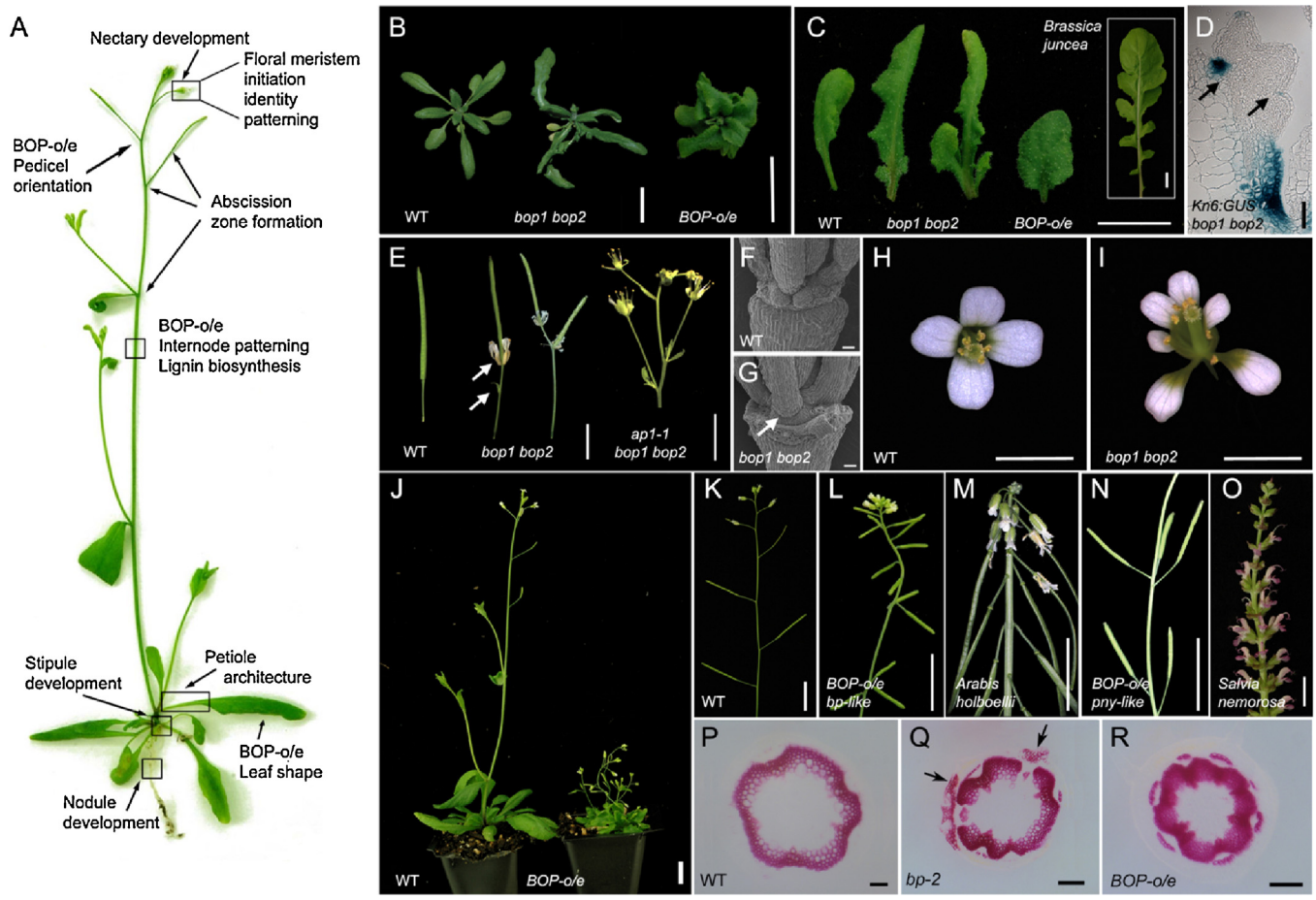

Fig. 2. Morphological variation caused by Arabidopsis BOP1/2 loss- and gain-of-function. (A) Overview. (B and C) Rosette leaves in *bop1 bop2* are elongated with leafy petioles, compare to *Brassica juncea* (inset). BOP-o/e leaves are wide with a short petiole. Scale: 2 cm (B), 1 cm (C), 1.5 cm (inset). (D) A longitudinal section at the base of a young *bop1 bop2* leaf shows multiple tiny leaflets delineated by expression of *KNAT6* (arrows). Scale: 50 μm. (E) *bop1 bop2* flowers have inflorescence-like characteristics including bracts (arrow) and branching due to defects in floral-meristem identity. Phenotypes are enhanced by an *ap1-1* mutation. Floral-organs remain attached in *bop1 bop2* mutants (arrow) due to lack of abscission zone anatomy. Scale: 0.5 cm. (F–G) Nectaries at the base of stamens are absent in *bop1 bop2* mutants (arrow in G). Scale: 400 μm. (H–I) *bop1 bop2* flowers are asymmetric due to extra abaxial floral organs. The abaxial sepal in wild-type is typically replaced by two petalloid organs that grow outward as "wings". Scale: 2 mm. (J-O) BOP-o/e causes a spectrum of changes in inflorescence architecture that mimic variations in nature. Scale: 1 cm. (J) Strong BOP-o/e lines are short and bushy. (K) WT inflorescence. (L) Moderate BOP-o/e in the Landsberg *erecta* accession of Arabidopsis mimics a *bp* phenotype with downward-pointing fruits, compare to (M) *Arabis holboellii*. (N) Moderate BOP-o/e in the Columbia accession of Arabidopsis mimics a *pny*-like whorled arrangement of clustered flowers, compare to (O) *Salvia nemorosa*. (P–R) Cross-sections from the base of the primary stem were stained for lignin with phloroglucinol–HCl [18]. (P and Q) *BOP* ectopically expressed in *bp* stems causes abnormal deposition of lignin in the epidermis (arrows) and underlying cortex above gaps in the vascular ring. (R) *35S:BOP*-o/e stems overproduce lignin. Scale: 100 μm.

meristems, restrict growth, and control the specialized patterning of lateral organ boundaries in a variety of species and developmental contexts (Fig. 2A). Given the importance of growth asymmetry, differentiation, and determinacy in morphogenesis, it is unsurprising that both loss and gain of BOP function exert dramatic changes in plant architecture. The next part of the review summarizes BOP activities and interactions in the determination of leaf, flower, and inflorescence architecture, and in floral-organ abscission.

4. Leaf patterning

Leaf architecture is broadly classified as simple versus compound. Simple leaves have a single undivided blade. Compound leaves of typical eudicots have a divided blade composed of several pairs of leaflets attached to a central stalk, the rachis. In both cases, a narrow petiole joins the blade to the stem [23,24]. As implied, the *BOP* genes were named after their mutant phenotype in leaves. In the dominant negative *bop1-1* point mutant or *bop1 bop2* double T-DNA insertion mutants, simple Arabidopsis leaves acquire compound-like characteristics (Fig. 2B and C; [5,7,8]).

Interconversion between simple and compound leaves has occurred repeatedly in evolution in strong correlation with reactivation of meristematic activity in developing leaves [23,24]. Leaf morphogenesis comprises initiation, primary morphogenesis, and secondary morphogenesis phases [24,25]. Auxin-dependent signaling in leaf founder cells is the initial trigger of down-regulation of meristematic *KNOX* genes (*STM* and the closely related *BREVIPEDICELLUS* (*BP*)), required in selection of determinate leaf fate [23,24]. Repression is permanent in simple leaves. In most species with compound leaves, reactivation of *KNOX* genes occurs during an extended primary morphogenesis phase that facilitates leaflet development on the rachis [23–25].

While mutations that simplify or even convert compound leaves to simple are well known, mutations that convert simple leaves to compound are still lacking [24]. This makes it interesting that *bop1 bop2* leaves have compound-like morphology. First: proximal-distal growth of the petiole is prolonged causing an elongated leaf form (Fig. 2B and C; [5,8]). This is reminiscent of the extended primary morphogenesis phase of compound leaf development wherein the marginal blastozone (a meristematic region at the base of the blade) facilitates leaflet initiation [25]. Second: the *bop1 bop2* petiole is abaxialized, producing a rachis-like structure. Cross-sections through the petiole reveal phloem encircling xylem and a pith-like interior similar to the rachis of a tomato compound leaf

[5,22,26]. Third: *bop1 bop2* petioles initiate leaflets and/or continuous blades (Fig. 2C; [5–8]). Multiple tiny leaflets delineated by expression of the lateral organ boundary gene *KNOTTED1-LIKE FROM ARABIDOPSIS THALIANA6* (*KNAT6*) can be seen in cross-sections through the base of young leaves similar to the pattern of *CUC3* in compound leaves (Fig. 2D; [27,28]). Recent progress has clarified how BOP1/2 in the petiole repress *KNOX* genes and inhibit blade outgrowth in maintaining simple leaf shape (summarized in Fig. 3A).

4.1. Repression of KNOX genes

Three-amino acid loop extension (TALE) homeodomain proteins play central roles in the functioning of plant meristems. The TALE superfamily comprises KNOX and BELL1-like (BELL) subclasses [23,29]. Four class I *KNOX* genes with overlapping functions provide meristem activity in Arabidopsis: *STM*, *BP* (formerly known as *KNAT1*), *KNAT2*, and *KNAT6* [23,29]. Whilst all four genes are ectopically upregulated in *bop1 bop2* leaves, misexpression of *KNAT6* has the greatest functional impact (Fig. 2D, [5,22,30], Xu and Hepworth, unpublished data). Single inactivation of *KNAT6* partially rescues the *bop1 bop2* leaf phenotype. Moderate but incomplete rescue is achieved by the additional inactivation of *BP* and *KNAT2*, indicating the involvement of other factors, potentially *STM* [30].

Stable repression of *KNOX* genes in species with simple leaves depends on the activities of orthologous MYB transcription factors, represented by ASYMMETRIC LEAVES1 (AS1) in Arabidopsis [23,31,32]. AS1 functions in a complex with the LATERAL ORGAN BOUNDARIES (LOB) domain factor ASYMMETRIC LEAVES2 (AS2) [23]. AS1 and AS2 bind to separate sites on the promoters of *BP* and *KNAT2*, where they interact through looping to direct repression of transcription via recruitment of Polycomb-repressive Complex2 [33,34]. Despite high levels of *KNOX* expression in *as1* and *as2* leaves, there is no blade-on-petiole phenotype [32]. Leaves in *as1* have a short petiole and broad rumpled blade [e.g. 31, 32]. Leaves in *as2* are often dissected by a single leaflet at the blade-petiole interface [e.g. 32, 35]. Time-course experiments indicate that leaflet formation in Arabidopsis requires the misexpression of *KNOX* genes during the primary morphogenesis stage of leaf development, corresponding to the interval where an active marginal blastozone promotes rachis extension and leaflet initiation at the base of compound leaves [36]. These data suggest that *bop1 bop2* mutation prolongs the primary morphogenesis phase of leaf development or mimics its properties in conjunction with *KNOX* misexpression.

One way that BOP1/2 repress *KNOX* genes is through direct activation of *AS2* (Fig. 3A; [20]). Promoter deletions supported by chromatin immunoprecipitation analysis delineated a 600-bp region located between 2.6 kb and 3.2 kb upstream of the *AS2* translation start site that is responsive to BOP1 [20]. Expression of a *BOP1pro:AS2* fusion gene was sufficient to complement leafy outgrowths in 11% of *bop1-1* and 5% of *bop1 bop2* lines. *BOP1pro:AS2* also restored wild-type blade morphology in *as2-1* mutants, even though *BOP1* is primarily transcribed in the petiole. This may indicate a function for BOP1/2 in the blade. Alternatively, the establishment of a stable repressive state for *KNOX* chromatin in newly initiated leaves may be sensitive to *AS2* depletion [33,34].

Synergistic enhancement of meristematic activity in *bop1 bop2 as1* and *bop1 bop2 as2* petioles indicates that BOP1/2 repression of *KNOX* genes is not exclusively through AS2 (Fig. 3A; [22]). *BOP* overexpression (BOP-o/e) upregulates several other LOB domain genes, including *LOB/AS2-LIKE4* and *LOB DOMAIN36/AS2-LIKE1*, whose functions partially overlap with *AS2* based on similar gain-of-function phenotypes [22,37–39]. However, BOP1 activation of these genes is indirect and loss-of-function fails to rescue BOP-o/e phenotypes [20,22]. The LOB domain factor JAGGED LATERAL ORGANS and CINCINNATA (CIN)-LIKE TCP (TEOSINTE BRANCHED1,

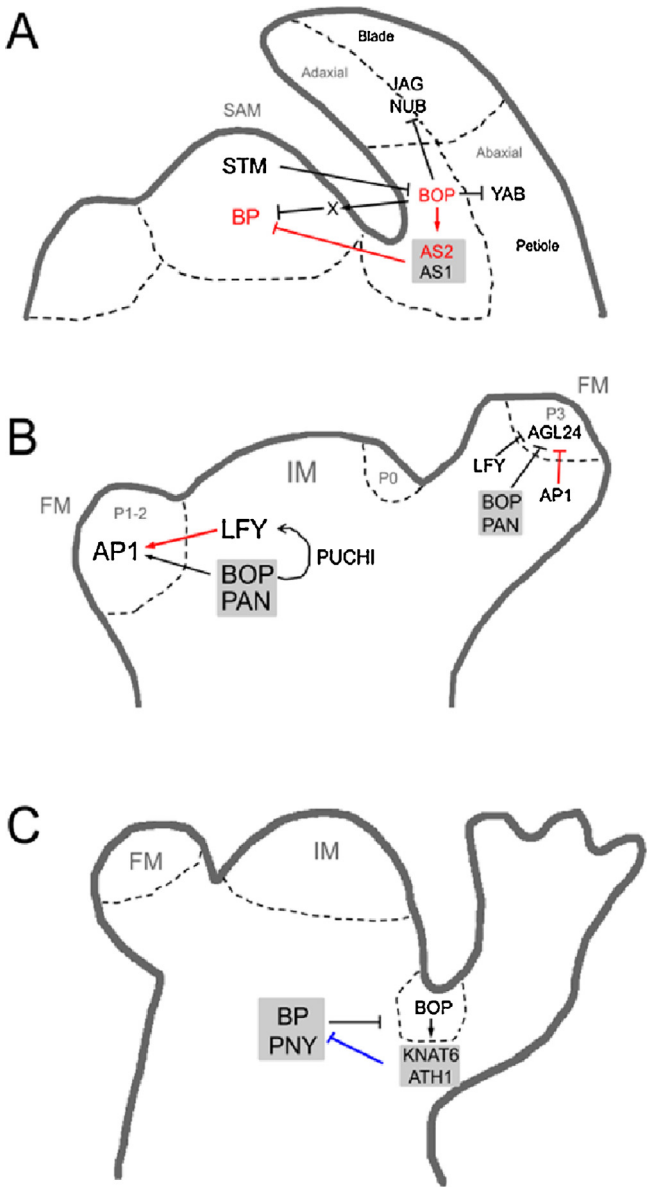

Fig. 3. Summary of genetic interactions for BOPs in leaves, the floral meristem, and internodes. Arrows, transcriptional activation. T-bars, transcriptional repression. Grey boxes, complex formation. Red denotes direct transcriptional regulation. Blue denotes post-translational regulation of activity. (A) BOPs in the petiole promote simple leaf shape by repressing the meristem activity of class I KNOX genes, promoting adaxial fate, and excluding blade-promoting transcription factors. BOPs directly activate *AS2* whose product forms a complex with AS1 for direct repression of *BP*. BOP1/2 repression of KNOX genes is also mediated by a second unknown pathway. BOPs promote adaxial fate by activation of *AS2* and by repression of *YAB* abaxial fate determinants. BOPs inhibit blade outgrowth by excluding JAG-like factors from the petiole. (B) BOP1/2 promote floral-meristem development in conjunction with flower-specific factors. BOPs function redundantly with PUCHI in promoting *LFY* expression in lateral meristems. At stages 0–2, BOP1/2 and LFY promote meristem proliferation and confer floral fate through activation of *AP1*. At stage 3, BOP1/2, LFY, and AP1 activities converge in promoting determinacy by blocking the continued expression of inflorescence meristem identity genes including *AGL24*. BOP1/2 in the floral meristem is recruited to DNA by interactions with the TGA bZIP factor PAN. (C) In the inflorescence, BP and PNY in the stem cortex restrict *BOP1/2* expression to the axil of floral pedicels. BOPs promote the expression of lateral organ boundary genes *KNAT6* and *ATH1* whose products form a complex that opposes BP-PNY activity. Ectopic BOP-o/e in the stem restricts growth, inhibits internode elongation, and accelerates lignin deposition, phenotypes that likely reflect the endogenous function of BOPs at lateral organ boundaries specialized in abscission and/or dehiscence. SAM, shoot apical meristem; FM, floral meristem; IM, inflorescence meristem. P, floral primordia (stages as indicated).

CYCLOIDEA, and PROLFERATING CELL FACTOR1) transcription factors are newly identified components of the AS1-AS2 based *KNOX* repression complex and candidates for BOP activation in leaves. Similar to *bop1 bop2*, mutations in these genes cause synergistic enhancement of meristematic activity in *as2* petioles [40–42].

4.2. Adaxial leaf polarity

Lateral outgrowth of the blade requires a functional leaf margin, a boundary where adaxial and abaxial fates are juxtaposed [43]. Mutations that impair adaxial leaf fate can result in patches of abaxial tissue misplaced on the adaxial side, capable of forming new boundaries that support adventitious blade outgrowth [43]. This was proposed as a mechanism to account for residual blade-on-petiole phenotype in *bop1 bop2 bp knat2 knat6* mutants [22]. Within this context, BOP1/2 promote adaxial leaf fate. Petiole cross-sections in *bop1 bop2* show partial (55%) or complete (22%) abaxialization of vasculature (xylem surrounded by phloem) with enhancement provided by mutations in *as1* and *as2* together with adaxial misexpression of abaxial fate determinants [22]. Nevertheless, inactivation of abaxial fate determinants *YABBY3* and *FILAMENTOUS FLOWER* in *bop1 bop2* only weakly suppress blade outgrowth [22,30].

4.3. Blade outgrowth

Once a leaf margin is established, blade outgrowth is dependent on downstream factors that delay differentiation and promote cell proliferation. Arabidopsis petioles express a number of lateral organ boundary markers (e.g. *BOP1/2*, *AS2*, low levels of *KNAT6*) suggesting that petioles are an extension of the boundary where growth is restricted. Analysis of cell division markers supports this view, showing a shift in proliferative activity from the blade-petiole junction in wild-type to the leaf base in *bop1 bop2*, which is consistent with petiole acquisition of blade character [44].

JAGGED (JAG) and its paralog NUBBIN (NUB) are important positive regulators of blade outgrowth [45–47]. JAG gain-of-function promotes ectopic development of floral bracts and leafy petioles [45,46] similar to *bop1 bop2* mutants. This identifies JAG as a potential target of BOP regulation [7,8]. LYRATE is the tomato ortholog of JAG. *LYRATE* overexpression results in a *bop1 bop2*-like phenotype in tomato with ectopic development of blade on the petiole, rachis, and petiolule of leaflets [48]. *JAG* expression is restricted to the blade in wild-type leaves but expands to the petiole in *bop1 bop2* mutants [7]. This indicates that BOP1/2 in the petiole directly or indirectly exclude blade-promoting transcription factors important in maintaining simple leaf shape (Fig. 3A). *LYRATE* inactivation simplifies tomato compound leaves, but this is not seen in *jag bop1 bop2* triple mutants, consistent with continued expression of *NUB* [7,47]. A third candidate for BOP regulation is *LEAFY PETIOLE*, which encodes an EREBP/AP2-type transcription factor, whose gain-of-function transforms petioles into blades [49].

Other candidates of BOP regulation in leaves include downstream targets of the YABBY pathway: *CIN-TCP* and *YUCCA* auxin biosynthesis genes [50]. *CIN-TCP* genes are active during the primary morphogenesis stage of leaf development in Arabidopsis and tomato [51,52]. Down-regulation of CIN-TCP activity delays cell cycle arrest thereby increasing the complexity of leaves whereas up-regulation conditions smaller leaves with reduced complexity [51–53]. Blade expansion is similarly blocked by the progressive inactivation of auxin-biosynthetic *YUCCA* genes highlighting a well-established requirement for polar auxin transport in blade outgrowth [54]. Microarray experiments will be useful in identifying which of these potential BOP targets is biologically relevant.

4.4. Compound leaf development in legumes

Legume species including pea (*Pisum sativum*) and *Medicago trunculata* use meristematic activity provided by orthologs of Arabidopsis *LEAFY* (*LFY*) in place of *KNOX* genes for elaboration of compound leaves [24,25]. A pair of leafy stipules flanks the base of compound pea leaves. The central rachis initiates a pair of proximal leaflets, two pairs of distal tendrils, and a terminal tendril. Prolonged expression of *UNIFOLIATA (UNI)* enhances pea leaf complexity whereas inactivation of *UNI* or its counterpart *SINGLE-LEAFLET1* in *M. truncatula* results in a simple leaf with a single terminal leaflet [55–57].

Legume orthologs of *BOP2* were recently identified as *COCHLEATA* (*COCH*) in pea and *NODULE ROOT* (*NOOT*) in *M. truncatula* [58]. BOP activity in these genomes is encoded by a single locus whose inactivation alters leaf patterning via defects in stipule development [14,58–61].

Stipules flank the base of leaves in ~25% of angiosperm species but are absent in *bop1 bop2* mutants [44]. Stipules are also reduced or absent in *noot* mutants and at early nodes in pea *coch* mutants. At later *coch* nodes, stipules are converted to leaflets or an entire compound blade, signifying loss of determinacy and conversion to leaf fate. This is consistent with *COCH* expression in stipule primordia [14,56,58,59]. The phenotype is explained in part by transient and ectopic expression of *UNI* in compound *coch* stipules during the period in which a marginal blastozone is active in the converted stipule [56]. *uni* mutations partially suppress the *coch* stipule phenotype [56,60,61]. In Arabidopsis, BOP interactions with LFY also play a role in proliferation and patterning of the floral meristem ([7,13]; see Section 5).

5. Flower development

5.1. Specification of floral meristems

Floral inductive signals acting on the shoot apical meristem result in acquisition of inflorescence meristem fate and new patterns of aerial development. In Arabidopsis, internodes are elongated and axillary meristems proliferate in the axils of leaves whose development is suppressed, resulting in the production of an inflorescence. Axillary meristems are indeterminate at early nodes, giving rise to secondary inflorescences. Axillary meristems at subsequent nodes become determinate through specification of floral fate [62,63].

LFY and APETALA1 (AP1) are the major regulators of floral-meristem identity in Arabidopsis [63,64]. *BOP1/2* are co-expressed with *LFY* and *AP1* during the early stages of flower development. Triple mutant analyses determined that BOP1/2 function redundantly with LFY and AP1 in initiating and patterning flowers (diagrammed in Fig. 3B; [7,13]). This role is unsurprising given that floral meristems are formed in the axil of cauline leaves at the lateral organ boundary [15,65].

LFY expression in floral anlagen (stage 0 floral primordia) is the earliest marker of floral fate [65,66]. During stages 1 and 2, *LFY* is transcribed in the adaxial part of the primordium [65,66] where its activity stimulates meristem proliferation and confers floral fate through direct activation of floral-meristem identity genes including *AP1* [63,64,67]. *BOP1/2* expression mirrors *LFY* until late stage 2 [13]. There is some evidence that BOP1/2 promote *LFY* expression (Fig. 3B). This comes from analyses of PUCHI, an EREBP/AP2-type transcription factor expressed in lateral meristems [68]. Weak floral-meristem identity defects in *puchi* mutants are enhanced by *bop1 bop2* inactivation, together with diminished *LFY* expression [68]. Loss-of-function *bop1 bop2* also causes subtle defects in floral-meristem identity signified by enlarged floral

bracts, a delay in the node of first flower, the occasional absence of cauline leaves at the base of lateral branches, and branched flowers (Fig. 2E; [7,13,22]). Enlarged floral bracts and branched flowers are also found in *coch* mutants, indicating conserved function in pea [14,58].

Analyses of Arabidopsis triple mutants indicate that BOP1/2 and LFY act separately in initiating floral meristems. Mutations in *lfy* delay the node of first flower. When flowers are eventually produced, a delay in proliferation of the floral meristem permits enlargement of the subtending floral bract, also seen in *bop1 bop2* [7,8,62,65]. In *bop1 bop2 lfy-1* triple mutants, bracts are larger and more numerous relative to *lfy-1* as is the number of "empty" bracts without an axillary meristem [7,13]. *AP1* transcripts in the triple mutant are less abundant than in *lfy-1*, indicating that BOP1/2 and LFY independently promote floral-meristem identity via activation of *AP1* (Fig. 3B; [13]).

The *AP1* promoter integrates signals from multiple flowering-time pathways. A LFY binding site is essential for proper photoperiodic induction of *AP1* [63,69]. *AP1* is also activated by a complex of the bZIP FD and FLOWERING LOCUS T (FT) in response to photoperiod, temperature, and hormonal signals [70]. BOP1/2 are unlikely to act through either pathway since *AP1* transcript accumulation in *lfy-1* and *ft-1* is further reduced by loss-of-function *bop1 bop2* [13]. SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) proteins directly activate *AP1* expression in response to endogenous and age-related signals [71,72]. BOP1/2 function in this pathway remains untested. Alternatively, BOP1/2 might activate *AP1* directly based on interactions with TGA (TGACG-motif binding) bZIP transcription factors [8,13]. Mutants in TGA8/PERIANTHIA (PAN) have weak floral-meristem identity defects similar to *bop1 bop2* including floral bracts, missing cauline leaves, and branched flowers [13]. At a later point in development, BOP1/2 and PAN form a nuclear complex that controls sepal number [8,13]. BOP1-GFP is enriched at two potential TGA binding sites in the *AP1* promoter close to binding sites for LFY, FD, and SPL3/9 based on chromatin immunoprecipitation assays [13]. Given that *pan ap1* double mutants are not enhanced in floral meristem identity defects, PAN may function redundantly with other TGAs expressed in the developing flower [13,73,74].

5.2. Meristem determinacy

BOPs promote floral meristem determinacy at two steps. At late stage 2, BOP1/2, LFY and AP1 activities converge in blocking the continued expression of inflorescence meristem identity genes (Fig. 3B; [13,64]). Prolonged expression of *AGAMOUS-LIKE24 (AGL24)* in *ap1-1* and *lfy* flowers is associated with "branching" caused by the ectopic initiation of second-order floral meristems in the axils of sepals [64]. Branching complexity and misexpression of inflorescence identity genes is dramatically enhanced in *bop1 bop2 ap1* and *bop1 bop2 lfy* triple mutants with phenotypes suppressed by loss-of-function *agl24* (Fig. 2E; [13]). These genetic interactions suggest a role for BOP1/2 in maintaining determinacy through repression of *AGL24* [13]. The pea ortholog of AP1 is PROLIFERATING INFLORESCENCE MERISTEM (PIM) [75]. Flowers in *pim coch* double mutants are similarly replaced by highly branched leafy shoots [76].

At late stage 3, activation of *AGAMOUS* (*AG*) in the central whorls of the flower directs termination of the floral meristem. PAN functions with LFY in maintaining *AG* expression by binding to a conserved bZIP element in the *AG* second intron [74,77]. Genetic evidence suggests that BOP1/2 is part of this pathway. Prolonged meristematic activity in *pan-2 lfy* mutants results in extra whorls of floral organs and unfused carpels, also seen in *bop1 bop2 lfy* flowers [13,77].

5.3. Floral patterning

Typical eudicot flowers are composed of sepals, petals, stamens, and carpels arranged in four concentric whorls. The ABC model describes how floral organ identities are established by the overlapping activities of three classes of homeotic genes, termed A, B, and C [78]. Floral-meristem identity factors LFY and AP1 provide A-class activity by promoting sepal and petal identities and by antagonizing C-class function in the outer whorls [78]. Similar A-class activity for BOP1/2 was revealed by triple mutant analyses with *lfy* and *ap1*. For instance, inactivation of *bop1 bop2* in a weak *lfy-2* mutant background produced flowers having a strong *lfy-1* phenotype. Outer whorl organs in *bop1 bop2 lfy-1* were enhanced in carpelloid character similar to *ap1 lfy-1* double mutants [13]. These interactions confirm floral-meristem identity patterning functions for BOP1/2 in the flower.

5.4. Nectaries

BOP1/2 are strongly expressed in the nectaries that arise at the base of stamens [21,79]. Nectaries in *bop1 bop2* are underdeveloped and lack characteristic landmarks such as parenchymal and secretory tissue and modified stomata (Fig. 2F and G; [21]). This is another example of the requirement for BOPs in the differentiation of axillary organs.

5.5. Floral symmetry

The arrangement of floral organs in most eudicot flowers is tetramerous or pentamerous. The option for dorsal-ventral (adaxial-abaxial) asymmetry is superimposed on this pattern [80]. Loss-of-function *bop* mutations in Arabidopsis, pea, and *M. truncatula* all increase the number of floral organs and perturb dorsal-ventral growth patterns, altering flower symmetry [8,14,58].

Arabidopsis *bop1 bop2* mutations convert floral patterning from tetramerous to partially pentamerous and change overall symmetry from radial to bilateral [8]. Wild-type flowers are radially symmetric with four sepals, four petals, six stamens, and two fused carpels (Fig. 2H). Radial symmetry in *bop1 bop2* flowers is broken by outgrowth of the floral bract and by the ectopic initiation of organs on the abaxial (ventral) side of the floral meristem. Unlike wild-type, the adaxial (dorsal) sepal emerges first, followed by four additional sepals spaced equidistantly in the whorl. The two abaxial sepals are typically converted to wing-like petals that extend outward, accentuating the appearance of asymmetry (Fig. 2I; [8]). In some flowers, an extra stamen forms on the abaxial side [8]. The mutation of *PAN* causes a similar shift to pentamerous patterning based on additional abaxial organs [8,81]. This is because BOP1/2 and PAN form a nuclear complex and function in the same genetic pathway to control sepal number [8,13].

Wild-type peas have asymmetric flowers subtended by bracts. Patterning is pentamerous with five sepals fused at their bases in the outer whorl. The corolla is composed of three types of petals arranged asymmetrically along the dorsal-ventral axis: a large standard on the dorsal side, two lateral wing petals, and a pair of fused ventral keel petals. Ten anthers fused into a tube and one central carpel complete the flower [14].

Typical *coch* flowers are dorsalized with enlarged floral bracts and supernumerary organs in all whorls [14,58,61,82]. Defects in dorsal-ventral asymmetry are best seen in the petal whorl. In a typical *coch* flower an extra standard develops ectopically on the dorsal side of the floral meristem, replacing the keel petals. An additional pair of wing petals forms laterally, creating a symmetrical flower with six corolla petals [58]. Extra floral organs occur in all whorls in strong *coch* mutants, along with variety of organ

fusion and mosaic phenotypes, suggesting an enlarged floral meristem with disrupted whorl boundaries [14,82]. The complexity of *coch* flowers is reduced by mutation of pea *LFY* (*uni-tac*) [82] suggesting that differentiation in *bop1 bop2* and *coch* floral meristems is potentially delayed, providing founder cells for additional organs. This fits with the role of BOP1/2 in attenuating meristem activity in other developmental contexts.

The patterning changes in *bop1 bop2* and *coch* mutants seem to be concentrated on the abaxial (ventral) side of the floral meristem. *BOP1/2* expression in the Arabidopsis floral meristem is symmetrical until late stage 2 when it becomes concentrated at the abaxial base of the flower—its boundary with the cryptic bract [13]. Dorsal-ventral polarity in legumes is governed by CYCLOIDIA-like TCP growth regulators dorsally expressed in the floral meristem, as shown earlier in *Antirrhinum* [83–85]. Dorsalization of *coch* flowers suggests that *COCH* might inhibit ventral expression of CYCLOIDIA-TCP genes to maintain asymmetry. Now that *COCH* has been identified, these models can be tested. Flower modification in *noot* mutants is mild. Additional petals and stamens are formed along with changes in petal character [58].

6. Inflorescence architecture

Spatial regulation of *BOP1/2* expression is an important determinant of inflorescence architecture. Parameters such as the timing, length, and pattern of internode elongation and the orientation of flowers are key variables that contribute to architectural diversity [86]. Elongation of Arabidopsis internodes begins at the transition to flowering, contributing to a regular spiral arrangement of upwardly oriented flowers on the primary stem [87,88]. When elongation is complete, the differentiation of lignified interfascicular fibers provides mechanical support [89,90].

6.1. Internode patterning

Arabidopsis *BOP1/2* expression in the primary stem is excluded from internodes and restricted to lateral organ boundaries in the axils of pedicels [18]. Gain-of-function studies revealed that BOP misexpression in stem tissue alters the length and pattern of internode elongation, pedicel orientation, and accelerates lignin deposition via antagonism of KNOX-BELL meristem factors leading to dramatic changes in plant stature and the display of flowers (Fig. 2A and J–O, diagrammed in Fig. 3C; [7,18,22,91]).

TALE homeodomain proteins are subdivided into KNOX and BELL classes, whose products function as heterodimers [23,29]. Class I KNOX genes *BP* and *STM* are expressed in both meristematic and stem tissues [92,93] where their activities require BELL binding partners [23,29]. In the shoot apical meristem, this function is fulfilled by a minimum of *ARABIDOPSIS THALIANA HOMEOBOX GENE1* (*ATH1*), *PENNYWISE* (*PNY*; also called *BELLRINGER*, *REPLUMLESS*, and *VAMAANA*), and *POUND-FOOLISH* (*PNF*) based on genetic and biochemical data [23,94].

Significant reorganization of TALE homeobox gene expression occurs at the transition to flowering in support of new patterns of aerial development [87,88,93,95,96]. *ATH1* expression is down-regulated in the inflorescence meristem but continues at lateral organ boundaries where its product interacts with KNAT2 and KNAT6 (Fig. 3C; [94,95,97,98]). *BP* expression is also down-regulated in the inflorescence meristem and moves to the stem cortex where its activity promotes internode elongation and vascular patterning (Fig. 3C; [87,93,99,100]). *PNY* expression is maintained in the inflorescence meristem but expands to the stem cortex where its function overlaps with BP (Fig. 3C; [87,96]). *STM* and *PNF* also remain expressed in the inflorescence meristem [88,92]. Mutations in *pny pnf* block both internode elongation and flowering indicating important functions for these genes in meristem competence to flower [88].

The inflorescence architecture of *bp* and *pny* mutants differs markedly from wild-type. *BP* inactivation causes short downward-pointing pedicels whereas *PNY* inactivation alters phyllotaxy causing an irregular, often whorled (verticillate) arrangement of flowers on the primary stem. Both mutations result in shortened internodes, impaired apical dominance, and disrupted vascular patterning, with these phenotypes enhanced in the double mutant [87,99,100].

Inflorescence defects in *bp* and *pny* are the result of *BOP1/2* misexpression in the stem [18]. Quantitative analyses of apical dominance, pedicel angle, internode length, and phyllotaxy showed that inactivation of *BOP1/2* suppresses *bp-1* defects and rescues *pny-40126* defects in full. Patterns of *BOP1/2* misexpression in *bp* and *pny* mutants closely mirror the characteristic defects of each mutant. In *bp* mutants, *BOP1/2* expression expands to the abaxial side of nodes where growth restriction results in downward-pointing pedicels. Misexpression also occurs in narrow strips of epidermis and underlying stem cortex that extend basipetally below nodes. Abnormal cell-types in these tissues are ectopically lignified in mature *bp* stems [18,101]. In *pny* mutants, *BOP1/2* are misexpressed in the stem cortex above and below nodes consistent with growth restriction resulting in clustered fruits. Thus, BP-PNY in the stem confine *BOP1/2* expression to lateral organ boundaries in the axils of pedicels, necessary for wild-type inflorescence architecture (Fig. 3C; [18]).

BP and PNY repress additional lateral organ boundary genes that function in the same genetic pathway(s) as *BOP1/2*: *KNAT2*, *KNAT6*, and *ATH1* (Fig. 3C; [18,91,94,98,102]). Inactivation of *KNAT2* alone has no effect but loss-of-function *knat2 knat6* partially rescues *bp-9* and fully rescues *pny-40126* inflorescence defects similar to *bop1 bop2* [18,101]. Inactivation of *ATH1* partially or fully rescues *pny-40126* defects depending on the allele. Significant rescue of *bp-2* by *ath1* requires the additional inactivation of *KNAT6* [91,98]. These data are interpreted as distinct functions for ATH1 complexes with KNAT2 and KNAT6 in the stem.

BOP1/2 gain-of-function phenocopies *bp* and *pny* mutants because BOP1/2 act downstream of BP-PNY in functional opposition. Strong BOP-o/e lines have short irregular internodes similar to *bp pny* double mutants and broad leaves with a short petiole (Fig. 2B, C and J; [7,18]). In the Landsberg *erecta* accession of Arabidopsis, moderate BOP-o/e confers a *bp* phenotype with downward-pointing of fruits enhanced by the *erecta* mutation (Fig. 2K and L; [18,22]). In the Columbia accession of Arabidopsis, moderate BOP-o/e confers a *pny* phenotype with whorls of clustered fruits (Fig. 2K and N; [18]). Patterns similar to *bp* and *pny* occur in nature (Fig. 2M and O).

While it is clear that BOP1/2 antagonize BP-PNY activity in causing these patterns, the mechanism is complex. Rescue experiments confirm that BOP-dependent changes in inflorescence architecture require the function of downstream target genes: *AS2*, *KNAT6*, and *ATH1* (Fig. 3C; [18,20,22,91]). BOP1 directly activates *AS2* whose product is transcriptional repressor of *BP* [20]. Compatible with this, *BP* transcript abundance is reduced in Landsberg *erecta* BOP-o/e lines with a *bp* phenotype [22]. Pedicel orientation in these lines is partially rescued by loss-of-function *as2*, but there is no rescue of internode elongation [22] nor does *as2* rescue *bp* and *pny* inflorescence defects [18]. This suggests the involvement of additional factors. Subsequent experiments identified a functional requirement for KNAT6 and ATH1 whose transcripts are elevated in Columbia BOP-o/e internodes. Inactivation of one genomic copy of *KNAT6* or *ATH1* in these lines is sufficient to rescue BOP-o/e defects in internode elongation and leaf shape [18,91]. ATH1 loss- and gain-of-function phenotypes are similar to those of BOP1/2 suggesting a close functional relationship: *ath1* and *bop1 bop2* are defective in

floral-organ abscission and *35S:ATH1* and *35S:BOP1* overexpressing lines are late flowering with short internodes [8,18,95,97,103]. In contrast, *35S:KNAT6* overexpressing lines have a lobed leaf phenotype similar to *35S:BP* [93,104]. This suggests that KNAT6 activity is modified by co-misexpression of *BOP1/2* and interaction with ATH1. Given that *BP* and *PNY* expression is unchanged in Columbia BOP-o/e lines, BOP1/2-dependent antagonism of BP-PNY via ATH1-KNAT6 is best explained by a post-transcriptional mechanism [18]. BP-PNY and ATH1-KNAT6 heterodimers with affinity for the same binding sites might antagonistically regulate the expression of common target genes to determine growth patterns in the inflorescence.

6.2. Lignin biosynthesis

The collective downstream targets of KNOX and BELL transcription factors include genes that modulate the abundance of cytokinins and gibberellins and that encode enzymes for cell wall remodeling and lignin biosynthesis [23,101,105,106]. Gibberellins play a well-established role in elongating internodes [107]. When this process is ended, differentiation of lignified interfascicular fibers completes the vascular ring and provides mechanical support [89,90,108]. BP and its orthologs repress secondary stem development, consistent with their role in maintaining indeterminancy [101,106]. BOP1/2 and BP have opposing roles in the maturing stem where they function as antagonistic regulators of lignin biosynthesis [18].

Interfascicular fibers prematurely differentiate in *bp* stems [101]. Cross-sections reveal gaps in the vascular ring underlying narrow strips of epidermis and adjacent cortex where lignin is ectopically deposited (Fig. 2P and Q; [18,101]). Wild-type patterning is partially restored by *bop1 bop2* loss-of-function [18]. Mutations in *knat6*, *knat2*, and *ath1* also suppress *bp* stem defects by varying degrees [18,91] confirming the functions of these genes in a genetic pathway common with BOP1/2. The stem has a thicker vascular ring with reduced interfascicular space in *pny* mutants. Patterning is restored to wild-type by *bop1 bop2* loss-of-function [18,87]. BOP-o/e stems have an expanded pattern of lignification similar to *bp pny* double mutants, consistent with a promotive role for BOPs in lignin biosynthesis (Fig. 2R; [18,87]).

KNOX genes are active in the vascular cambium of woody species where they stimulate or maintain meristematic activity required for lateral growth [90]. Genetic and biochemical data suggest that *BP* directly represses lignin biosynthesis genes in the stem [101]. *In vitro*, BP binds to the promoter of three lignin biosynthesis genes whose expression is elevated in *bp* stems [101]. Several other lignin biosynthesis genes whose transcripts are upregulated in *bp* stems (*PAL1*, *C4H1*, *4CL1*, *C3H1*, *CAD5*, and *PRXR9GE*) were restored to near wild-type levels by *bop1 bop2* mutation [18,101]. Four of these same genes are dramatically up-regulated in BOP-o/e stems consistent with promotion of lignin biosynthesis. Of these, the class III peroxidase-encoding *PRXR9GE* expressed in lateral organ boundaries (data unpublished, Khan and Hepworth) stands out as the best candidate for direct regulation, its transcripts showing a 15- to 20-fold increase in *bp* and *BOP*-o/e internodes [18]. Peroxidases are predicted to activate monolignol subunits for polymerization in the final step of lignin assembly. Polymer assembly may be an important regulatory node for developmental control of lignin deposition [101].

6.3. Hormonal controls

The metabolic targets of BOP1/2 that inhibit internode elongation are still unknown. Short internodes are characteristic of defects in gibberellin biosynthesis [107]. However, GA_{20}-oxidase transcript levels in BOP-o/e stems appear to be unaffected and spray treatments with GA_3 failed to rescue internode elongation [18]. This suggests that BOP1/2 do not simply repress gibberellin biosynthesis. Overproduction of jasmonic acid inhibits stem elongation in tobacco by antagonizing gibberellin production and promotes secondary development in Arabidopsis stems [108,109]. It will be interesting to examine BOP interactions with jasmonic acid since BOP1/2 appear necessary for its perception in plant defense [110].

7. Abscission

BOPs are essential in abscission, a process that merges potential functions in development and defense [21,111,112]. Primary abscission zones at the site of detachment of leaves, floral organs, and fruits differentiate simultaneously with lateral organs at the boundaries where they are connected to the plant body (Fig. 2A). The abscission zone is typically comprised of several layers of small cytoplasmically dense cells that acquire responsiveness to separation-inducing signals that promote cell wall dissolution. After separation, exposed cells on the plant body undergo secondary differentiation, generating a protective surface rich in suberin and lignin that resists dehydration and pathogen attack [112,113].

7.1. Abscission zone formation

BOP activity in Arabidopsis and tobacco (*Nicotiana tabacum*) is essential for differentiation of abscission zones [21,111]. In Arabidopsis, only the floral organs abscise but vestigial abscission zones differentiate at the base of cauline leaves and pedicels [21]. *BOP1/2* are expressed at abscission zones prior to other abscission-related gene markers and their expression is maintained throughout development [18,21]. The correct spatial and temporal expression of several later-acting genes in the abscission pathway is not disrupted in *bop1 bop2* mutants suggesting separate or redundant control of their transcription at the lateral organ boundary [21,114]. *NtBOP2:GUS* in tobacco plants is expressed in the receptacle where overexpression of antisense *NtBOP2* blocks differentiation of the corolla abscission zone and delays petal senescence in transgenic flowers [111]. *NtBOP2* restricts growth in the differentiating corolla abscission zone by inhibiting longitudinal cell expansion [111].

7.2. Cell separation

Additional downstream functions of BOPs in abscission are likely. Studies in tobacco indicate that transcripts of the cellulase gene *NtCEL5* and expansion gene *NtEXPA5* expressed in the abscission zone are reduced in NtBOP2-AS transgenic flowers [111]. Prior to shedding, cells in the abscission zone become responsive to an integrated set of hormonal, developmental, and/or environmental signals leading to abscission. Responsiveness to these signals requires a pair of leucine-rich receptor-like kinases, HAESA and HEASA-LIKE2, whose ligand is a secreted peptide called INFLORESCENCE DEFICIENT IN ABSCISSION (IDA) [113]. Genetic and expression data support the model that IDA-dependent signaling upregulates *KNAT2/KNAT6* in the abscission zone just prior to organ detachment and promotes the final steps of abscission. Premature abscission in *35S:IDA* overexpressing plants is suppressed by *knat2 knat6* revealing that IDA signaling requires KNAT2 and KNAT6. Conversely, BP in the abscission zone delays abscission. In *bp* mutants, the abscission zone is enlarged and organs are prematurely shed [115]. Inactivation of *knat2 knat6* suppresses these defects, suggesting that ectopic KNAT2 and KNAT6 activities are conserved as a cause of *bp* defects [18,102,115]. BOP1/2 likely contribute to the promotion of *KNAT6* and potentially of *ATH1* in the abscission zone but this is difficult to test genetically because *bop1 bop2* mutation

blocks the earlier step of abscission zone formation [21]. Mutations in *ath1* and *knat2 knat6* delay but do not abolish abscission [95,115]. Collectively, antagonism between BP and BOP1/2 via KNAT6-ATH1 emerges as a conserved module in development.

Ethylene is the major signal for abscission but jasmonic acid provides additional positive input [112,116]. Genes required for cell wall hydrolysis and modification, jasmonic acid biosynthesis, lignin biosynthesis, and pathogenesis-related (PR) defense proteins are all found to be induced in microarray studies of abscission in citrus, tomato, and Arabidopsis [117–121]. The idea that hormonal signals prime the site of future abscission for resistance to pathogens by way of lignin deposition and activation of *PR* genes is intriguing. A role for BOP1/2 in this process as effectors of jasmonic acid or ethylene signaling is an unexplored possibility.

8. Patterning of fruits

Preliminary evidence suggests that an antagonistic interaction between KNOX-BELL and BOP1/2 activities also governs the architecture of fruits. The Arabidopsis silique is a fruit pod composed of two valves joined at their margins to a meristematic replum that generates seeds attached on the interior. The valves are homologous structures to leaves. The valve margins that separate the valves from the replum are lateral organ boundaries specialized in dehiscence, comprised of a separation layer adjacent to a layer of lignified cells required in pod shatter [122,123]. Numerous genes with functions in shoot apical meristem maintenance and leaf patterning play homologous roles in fruit patterning [123]. BP and PNY provide meristematic function in the replum and set the expression boundaries of genes that confer valve identity [124,125]. In strong *pny* alleles, the replum is replaced by cells with valve margin identity [124]. Inactivation of *bop1 bop2* or *knat2 knat6* rescues replum formation in *pny* fruits, consistent with their co-expression in the valve margins [18,102]. Given the functions of BOP1/2 acting via KNAT6-ATH1 in the stem and abscission zones, this same module is likely to pattern the fruit.

9. Signaling mechanism

The NPR1 signaling mechanism serves as a paradigm for BOPs. BOPs and NPR1 share homologous functional domains that support a similar mode-of-action (Fig. 1B). Both have a BTB/POZ domain at the N-terminus and two ankyrin repeats near the C-terminus that mediate interaction with TGA bZIP transcription factors [8,126,127]. BTB-ankryins also have an uncharacterized domain of unknown function (DUF3420) adjacent to the first ankyrin motif (Fig. 1B). Divergent C-termini for BOP1/2 and NPR1 reflect differences in post-translational regulation. The NPR1 C-terminus contains a bipartite basic nuclear localization signal [128] and a novel Cys-containing transactivation domain made functional by binding to the defense hormone salicylic acid (Figs. 1B, S1; [129,130]). BOP1 also functions as a transcriptional co-activator in the nucleus but this activity is salicylic acid-independent [4,20].

Supplementary material related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.plantsci.2013.10.019.

9.1. Interaction with TGA bZIP factors

TGA bZIP proteins are a distinct subclade in the bZIP superfamily [131]. The Arabidopsis genome encodes ten TGA factors subdivided into five clades based on sequence and function: Class I comprises TGA1 and TGA4, Class II comprises TGA2, TGA5, and TGA6, Class III comprises TGA3 and TGA7, Class IV comprises TGA9 and TGA10 and Class V comprises TGA8/PAN ([132]; Table S2). Genetic studies indicate that TGA factors operate with a high degree of functional redundancy with overlapping roles in defense, stress responses, and development ([132]; Table S2). NPR1 exerts most or all of its function via TGA factors based on genetic evidence: systemic acquired resistance is abolished in *tga2 tga5 tga6* triple mutants recapitulating the *npr1* mutant phenotype [133]. Biological relevance has been established only for BOP interactions with PAN in the floral meristem but physical interactions are weakly detected between BOPs and most TGAs in a yeast heterologous system [8,13,111]. Presumably, other TGAs with expression in the floral meristem, lateral organ boundaries, and petioles mediate BOP function in these tissues. Most TGAs are broadly expressed making it difficult to narrow the list [134]. Studies in other species provide clues. RNAi-mediated inhibition of the tobacco class II factor TGA2.1 induces stamen to petal conversions [135]. Mutation of maize *LIGULELESS2* (most related to *AtTGA9*) is involved in the establishment of the maize leaf blade-sheath boundary [136]. Maize KNOTTED1 directly regulates *LIGULELESS2* in establishing this boundary [137]. *liguleless2* mutants are also delayed in their transition from vegetative to reproductive development [138]. These phenotypes are reminiscent of petiole defects and of impaired commitment to flowering in *bop1 bop2* mutants of Arabidopsis [7,8,13]. There are no obvious defects in *tga9* loss-of-function mutants, but anther development is blocked in *tga9 tga10* double mutants highlighting the potential for functional redundancy [139].

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Arabidopsis Class I TGA factors are unique in having confirmed roles in defense and development. Loss-of-function *tga1 tga4* mutants are deficient in basal resistance to pathogens [140–142]. TGA1 is also pinpointed as a potential regulator of fiber differentiation based on transcript profiling in developing stems [89]. *TGA4* is strongly expressed in leaf vasculature and petioles [143] with *tga1 tga4* loss-of-function causing “pinwheel” leaf curvature similar to *bop1 bop2* (Fig. 2B; [142]). TGA4 was identified biochemically as interacting with CONSTANS and binding to the promoter of its immediate target gene *FT*, which promotes flowering [70,143]. TGA1/4 are particularly interesting given their potential as a bridge between defense and development.

9.2. Transactivation

Salicylic acid-dependent activation of defense genes by NPR1 was elucidated using *PR1* as a marker gene. TGA2 in resting cells functions as a transcriptional repressor of *PR1* [129]. The C-terminus of NPR1 contains a novel Cys-containing transactivation domain [129,130]. $Cys^{521/526}$ residues in the C-terminus interact with salicylic acid via the transition metal copper (Cu) (Figs. 1B, S1). Salicylic acid binding triggers a conformational change that releases the transactivation domain from an inhibitory interaction with the N-terminal BTB/POZ domain [130], which in turn interacts with the repression domain of TGA2 to negate its function [144]. It remains to be seen if NPR1 orthologs from other species use this mechanism since $Cys^{521/526}$ are not conserved [130]. Cu-binding motifs are diverse and can be based on co-ordination with histidine or methionine [145]. Polyhistidine tracts of five or more residues also have affinity for nickel and Cu transition metals [146]. Alignment of BOP orthologs shows conservation of two such histidine repeats: one at the C-terminus and one in the DUF3420 domain. Both tracts are conserved in eudicots, but only the DUF4320 tract is conserved in monocots (Fig. S1). These residues might function as metal-binding domains for post-translational control of BOPs based on analogy with NPR1. It would be interesting to test if these motifs allow direct perception of hormones with potentially dual

roles in defense and development. Jasmonic acid and ethylene are good candidates based on their involvement in abscission, stem elongation, and defense. Indeed, especially since Cu ions mediate ethylene binding to its receptor [147].

9.3. Nuclear localization

Nuclear localization is a key control point for NPR1 and potentially BOPs. NPR1 in the cytoplasm is an oligomer held together by intermolecular disulphide bonds utilizing Cys^{82} and Cys^{216} residues in the N-terminus (Figs. 1B, S1; [148]). A biphasic change in intracellular redox potential induced by pathogen attack triggers reduction of these bonds by thioredoxin and releases active NPR1 monomers that translocate to the nucleus [148,149]. Nuclear localization is also driven by exogenous salicylic acid and by oxidizing conditions induced by nitric oxide or ascorbate deficiency [130,141,148,150]. The reaction is reversible with reestablishment of NPR1 oligomers dependent on nitrosylation of Cys^{156} in the BTB/POZ domain [149]. BOP1/2 and its orthologs conserve only Cys^{82} in the BTB/POZ domain (Figs. 1B, S1). Nevertheless, work in multiple species confirms that BOP fusions to green fluorescent protein reside in both the cytoplasm and nucleus, suggesting biological relevance for this pattern [8,11,20,111]. If NPR1 serves as a paradigm, BOP accumulation in the nucleus may be enriched by hormone perception and/or by redox changes in the cell.

9.4. Redox regulation

Redox regulation also controls TGA transcription factor activity [132]. $Cys^{260/266}$ residues in the C-termini of TGA1 and TGA4 form intramolecular disulphide bridges that block interaction with NPR1. Reduction of these bonds in salicylic acid-treated plants restores TGA1 interaction with NPR1 [151]. Multiple TGAs with functions in defense and development interact physically and genetically with CC-type (ROXY-type) glutaredoxins that are predicted to reduce disulphide bonds or remove glutathione conjugated to cysteine in proteins [73,132,152]. Significant examples are seen in development. Double mutants *roxy1 roxy2* and *tga9 tga10* have similar defects in anther development. Direct interaction of TGA9/10 with ROXY1/2 suggests that ROXY-dependent modification of Cys residues in TGA9/10 is required for function [139,153]. Inhibitory interactions are also possible. Flowers in *pan* and *bop1 bop2* mutants contain extra petals, whereas *roxy1* mutants initiate fewer petals [8,81,151]. This suggests that ROXY1 modification of PAN inhibits petal initiation, possibly by disrupting interactions with BOP1/2. Equivalent Cys residues in TGA1 and PAN are crucial for function [73,151]. These data point to conserved functional interactions between ROXY-type glutaredoxins, BTB-ankryin proteins, and TGA factors in defense and development that have yet to be fully explored.

9.5. Protein stability and role of paralogs

Paralogs of NPR1 have an important role in fine-tuning defense responses. NPR1 monomers are constitutively cleared from the nucleus of resting cells in preventing the inappropriate activation of defense genes [2,154]. A high turnover of NPR1 in the nucleus following exposure to salicylic acid is equally important for the full induction of defense genes and the establishment of systemic acquired resistance [2,154]. NPR3 and NPR4 are salicylic acid-responsive adaptors for NPR1 recruitment to a CULLIN3-based E3 ubiquitin ligase complex that mediates degradation [3]. NPR3 bound to salicylic acid has increased affinity for NPR1, facilitating its rapid turnover during an immune response. Conversely, NPR4 bound to salicylic acid loses affinity for NPR1, promoting clearance of NPR1 from resting cells [3]. In accord, *npr3* and *npr3 npr4* mutants accumulate NPR1 to higher levels and are insensitive to induction of systemic acquired resistance. Stabilization of NPR1 in these mutants also blocks necrosis, which is required to stop the spread of pathogen from the initial site of infection [2,3]. Based on these data, BOP-NPR1 interactions may play a role in fine-tuning the defense response or in mediating cross-talk between salicylic acid and jasmonic acid or ethylene-mediated defenses.

10. Plant defenses

Basal resistance based on preformed structural and chemical barriers including the plant cuticle, cell wall, and anti-microbial compounds is the first line of defense against pathogens [155]. Penetration of these barriers stimulates localized responses geared to the life-style of the pathogen. In general, salicylic acid-induced defenses are most effective against biotrophic and hemi-biotrophic pathogens, which require nutrients from living tissue to complete their life cycle. Jasmonic acid/ethylene-induced defenses are typically most effective against herbivorous insects and necrotrophic pathogens, which kill the host tissue prior to feeding [155–157].

A new report indicates that BOP1/2 are essential for resistance induced by methyl jasmonate [110]. Initial characterization of *bop1 bop2* mutants showed no change in resistance to the hemi-biotrophic pathogen *Pseudomonas syringae maculicoli ES4326* [8]. Further study confirmed that *bop1 bop2 npr1-3* triple mutants were only slightly more susceptible to *Pseudomonas syringae tomato DC3000* (*Pto*) than *npr1-3* indicating little or no role for BOP1/2 in salicylic acid perception [4].

Two types of long-lasting broad-spectrum disease resistance occur in plants [157]. It is well-established that NPR1 is essential for systemic acquired resistance [2,157]. Beneficial soil microorganisms (arbuscular mycorrhizal fungi and certain rhizobacteria) exposed to the roots trigger a second form NPR1-dependent defense called induced systemic resistance which relies on jasmonic acid/ethylene signaling [155,158,159]. Induced systemic resistance differs from systemic acquired resistance in that it is salicylic acid-independent. Induced resistance is associated with depression of natural host defenses in aid of microbial colonization, and with "priming" for enhanced pathogen defense. Priming refers to the faster, stronger activation of defensive genes upon exposure to pathogen [155,158,159].

A promotive role for NPR1 in systemic acquired resistance and induced systemic resistance is both interesting and enigmatic since salicylic acid and jasmonic acid/ethylene signaling in plant defense tends to be mutually antagonistic [155–157]. Genetic studies show that NPR1 mediates negative crosstalk between salicylic acid and jasmonic acid signaling, functioning in the cytoplasm by an unknown mechanism to inhibit jasmonic acid-responsive gene expression [155,160,161].

Historically, a role for NPR1 in induced systemic resistance was established using the point mutant *npr1-1* [158,159]. Plants treated with methyl jasmonate have a priming response similar to induced systemic resistance [110]. NPR1 is now shown as dispensable for this response using an *npr1-3* truncation mutant. Rather, methyl jasmonate-induced resistance is abolished in *bop1 bop2* mutants and enhanced in plants overexpressing BOP1 or BOP2. The *npr1-1* product is proposed to negate BOP1/2 function in methyl jasmonate-induced resistance by acting as a dominant negative repressor. This activity derives from its high levels of expression in the plant compared to BOP1/2 and an ability to disrupt BOP interactions with TGA2, TGA5, and TGA6. This was shown indirectly in yeast by monitoring for changes in BOP interaction with TGAs caused by co-expression of *npr1-1* [110]. Compatible with this finding, Class II TGAs are required for both salicylic acid and jasmonic acid/ethylene defenses [132,133,162].

The *npr1-1* allele used in previous studies contains an H334A substitution in the third ankryin repeat rendering defects in both salicylic acid and jasmonic acid perception. The *npr1-3* allele contains a nonsense mutation at residue 400 resulting in deletion of the C-terminal regulatory region and is defective only in salicylic acid perception [1,110,160]. Collectively, these data are the first evidence that BOPs have a role in plant defense. On this basis, it is worth testing if BOP interactions with NPR1 or class II TGAs are regulated by salicylic acid or methyl jasmonate. Such interactions may be a means of curbing NPR1 function in promoting jasmonic acid/ethylene-induced resistance to necrotrophs, induced systemic resistance, and/or symbiosis with beneficial microbes.

11. Nodulation

Legumes are well-studied for their ability to establish symbiotic relationships with nitrogen-fixing rhizobia [58,163]. BOP2 orthologs *NOOT* and *COCH* from *M. truncatula* and pea transcribed in roots are essential for maintenance of nodule meristem identity and size (Fig. 2A; [58,164]). Despite defects in nodule identity, rhizobium infection and symbiosis in *noot* mutant plants is not adversely affected [58].

M. truncatula and pea have persistent tip-growing nodule meristems that originate from cell divisions in the inner cortical and pericycle cells adjacent to xylem poles in the root [163]. Nodule organogenesis has many parallels with the regeneration of adventitious shoots from lateral root primordia. In both cases, new meristems are derived from root pericycle cells adjacent to xylem poles [163,165,166]. Both processes are activated by cytokinin in response to signaling by WUSCHEL-like homeodomain proteins and entail the repression of lateral root fate [58,163,165,166]. In line with this, *BOP2* is induced by cytokinin downstream of WUSCHEL and plays an essential role in the establishment of adventitious shoot meristems from lateral root initials (Chatfield and Hepworth, unpublished results). These similarities suggest that the specialized role of BOPs in nodulation may be derived from a more conserved role in shoot regeneration from lateral root initials. Further experiments are needed to address the function of BOPs in the root.

12. Concluding remarks

BOP genes encode a distinct subclade of BTB-ankryin transcriptional co-activators, divergent in the NPR1 family of plant defense regulators. Combining data from Arabidopsis, moss, tobacco, pea, and *M. truncatula* provides a more complete understanding of their conserved roles in development. The emerging picture is that BOPs function at lateral organ boundaries—uniquely patterned transitional zones in the plant that separate determinate lateral organs from the apical meristem or plant body and that give rise to axillary meristems. BOPs as part of the boundary modulate growth, differentiation, and determinacy, accounting for diverse loss- and gain-of-function phenotypes impacting leaf, inflorescence, flower, fruit, and root nodule architecture (Fig. 2A). Loss-of-function studies indicate that BOPs inhibit longitudinal cell expansion as well as promote the differentiation of abscission zones and axillary organs including stipules, flowers, nectaries, and root nodules. Gain-of-function studies indicate that BOPs promote the expression of lateral organ boundary genes, including *AS2*, *KNAT6*, and *ATH1*, whose products inhibit growth, promote differentiation, and antagonize meristem activity in various developmental contexts. Mutually antagonistic interactions between BOPs and meristematic factors are an important link in maintaining borders between meristem-organ compartments and in controlling irreversible transitions in cell fate associated with abscission and lignin biosynthesis.

Many questions remain. Genomics approaches will be useful in clarifying how BOPs regulate both development and defense. Comparison of transcriptional targets in different species and in different tissues can be used to identify direct target genes that are unique or shared in different developmental contexts. While these are likely to include genes that promote lateral organ boundary or floral-meristem identity, will they also include genes that modulate hormonal pathways or that promote secondary cell wall biosynthesis, abscission, or plant defense? Promoter analyses of target genes from different functional classes can be used to examine if BOP range of function is due to interactions with different TGA factors and/or interactions with transcription factors of other classes. Striking parallels in the mechanism of BOP and NPR1 transcription factor activity remain to be tested. What is the significance of shared interactions with TGA bZIP factors and ROXY-type glutaredoxins? Do BOPs directly perceive jasmonate or ethylene signaling molecules to regulate events in development and defense? New data in these areas will help to connect diverse functions of *BOP* genes at lateral organ boundaries, in flowering, and potentially, as part of the complex circuitry that co-ordinates defense and development in land plants.

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