

Auxin Biology in Bryophyta: A Simple Platform with Versatile Functions

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Bryophytes, including liverworts, mosses, and hornworts, are gametophyte-dominant land plants that are derived from a common ancestor and underwent independent evolution from the sporophyte-dominant vascular plants since their divergence. The plant hormone auxin has been shown to play pleiotropic roles in the haploid bodies of bryophytes. Pharmacological and chemical studies identified conserved auxin molecules, their inactivated forms, and auxin transport in bryophyte tissues. Recent genomic and molecular biological studies show deep conservation of components and their functions in auxin biosynthesis, inactivation, transport, and signaling in land plants. Low genetic redundancy in model bryophytes enable unique assays, which are elucidating the design principles of the auxin signaling pathway. In this article, the physiological roles of auxin and regulatory mechanisms of gene expression and development by auxin in Bryophyta are reviewed.

Land plants evolved from ancestral charophycean algae approximately 500 million years ago (Morris et al. 2018). Among the extant land plants, bryophytes, including liverworts, mosses, and hornworts, are the basalmost lineages that diverged from other land plants. Recent phylogenetic analyses support the monophyly of bryophytes (Nishiyama et al. 2004; Cox et al. 2014; Wickett et al. 2014; Puttick et al. 2018), and the synapomorphy of “Bryophyta” is the haploid gametophyte-dominant life cycle. Germinated spores develop filamentous protonemata or globular sporelings, which in turn give rise to three-dimensionally (3D) organized gametophores (in leafy liverworts and mosses) or thalli (in thalloid liverworts and hornworts), from which reproductive organs that contain germ cells develop. After fertilization, diploid

sporophytes, consisting of a foot, seta or intercalary meristem, and sporogenous tissue, develop on the haploid bodies (Ligrone et al. 2012) and finally generate haploid spores via meiosis.

The plant hormone auxin is known to be involved in almost all aspects of development through regulation of cell division, expansion, and differentiation in sporophyte-dominant angiosperms (Demeulenaere and Beeckman 2014; Dresselhaus and Schneitz 2014; Landrein and Vernoux 2014; Rios et al. 2014). Classic auxin- and inhibitor-treatment experiments showed that in gametophyte-dominant bryophytes, auxin plays diverse roles in both gametophyte and sporophyte generations. Additionally, recent molecular approaches in model bryophytes and omics data revealed that the core compo-

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nents in auxin biology, including biosynthesis, metabolism, transport, and signaling, are highly conserved among land plants. At present, two model bryophytes, the moss *Physcomitrium* (*Physcomitrella*) *patens* and the liverwort *Marchantia polymorpha*, are widely used in part because of the ease of transformation and gene disruption, low genetic redundancy compared with angiosperms, and simple developmental traits (Rensing et al. 2008, 2020; Ishizaki et al. 2016; Bowman et al. 2017; Lang et al. 2018; Montgomery et al. 2020). These model bryophytes provide unique platforms for investigating auxin biology.

This article provides an overview of auxin biology in Bryophyta, including developmental roles revealed by pharmacological assays, homeostatic regulation revealed by chemical approaches, design principles of the nuclear auxin signaling (NAS) pathway to which genetic studies in model bryophytes contributed, and mechanisms underlying developmental regulation revealed by molecular biological studies. See also Bowman (2016), Kato et al. (2018), and Thelander et al. (2018) for reviews of classical auxin studies in gemma and gemmalings of *M. polymorpha*, recent auxin studies in *M. polymorpha*, and recent auxin studies in *P. patens*, respectively.

PHARMACOLOGICAL EFFECTS OF AUXINS AND THEIR INHIBITORS ON BRYOPHYTE DEVELOPMENT

Mosses spend a long time as protonemata, which consist of functionally distinct, interconvertible feeding (chloronema) and foraging (caulonema) filaments (Cove 2005). Some lateral branches on caulonema give rise to buds through several unique divisions, which in turn form 3D gametophores with stem-leaf structures (Harrison et al. 2009; Kofuji and Hasebe 2014). Exogenously supplied auxin was shown to inhibit protonema branching, induce caulonema differentiation, and promote bud initiation in caulonema together with cytokinin (Johri and Desai 1973; Ashton et al. 1979; Sood and Hackenberg 1979; Thelander et al. 2018). Because auxin transport inhibitors, *N*-1-naph-

thylphthalamic acid (NPA) and 3,4,5-triiodobenzoic acid, inhibit auxin-mediated caulonema differentiation, active transport seems necessary to accumulate sufficient auxin (Cove and Ashton 1984).

Moss gametophores, which harbor stem cell regions at their apices where leaves and reproductive organs arise, develop multicellular rhizoids from the basal epidermis (Sakakibara et al. 2003; Harrison et al. 2009; Kofuji and Hasebe 2014) and show various responses to exogenous auxin depending on the type and concentration of auxin used. Generally, low auxin enhances shoot development by elongating stems and increasing leaves, whereas high auxin arrests growth by stunting stalks and inhibiting leaf formation (Ashton et al. 1979; Bennett et al. 2014b; Lavy et al. 2016). Exogenous auxin generally elongates leaves because of longitudinal hyperelongation of cells (Barker 2011). Rhizoid development is also strikingly stimulated by auxin application (Ashton et al. 1979; Nyman and Cutter 1981; Chopra and Vashistha 1990; Sakakibara et al. 2003).

Various liverworts also show enhanced and/or ectopic rhizoid development in response to exogenous auxin, while excessive auxin sometimes suppresses rhizoid formation (LaRue and Narayanaswami 1957; Ilahi and Allsopp 1969; Allsopp and Ilahi 1970a,b). In the case of *M. polymorpha* (for its life cycle, see Fig. 1), auxin application enhances rhizoid development in sporelings and gemmalings (Rousseau 1950; Tarén 1958; Maravolo and Voth 1966; Allsopp et al. 1968; Ishizaki et al. 2012; Flores-Sandoval et al. 2015), whereas *L*-kynurenine (*L*-Kyn), an indole-3-acetic acid (IAA)-biosynthesis inhibitor, decreases rhizoids in growing thalli (Fig. 1; He et al. 2011; Flores-Sandoval et al. 2015). Under normal growth conditions, gemmae give rise to rhizoids from their ventral side after detaching from gemma cups as a germination process, which is regulated by light via phytochromes (Otto and Halbsguth 1976; Inoue et al. 2016). However, in the presence of moderate concentrations of auxin, rhizoids also emerge from the dorsal sides of the gemmae, even in the dark, suggesting that exogenous auxin compensates for light signals during gemma



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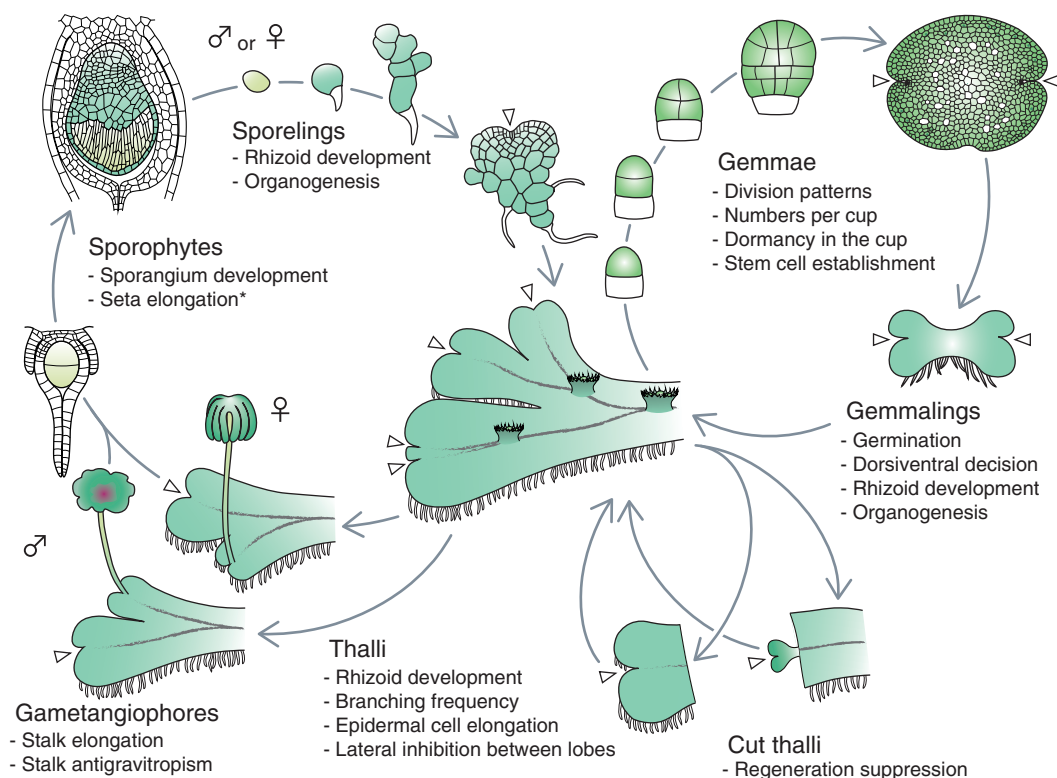


Figure 1. Auxin-regulated processes in thalloid liverwort throughout the life cycle. *Marchantia polymorpha* is depicted as a representative thalloid liverwort. Sporelings of *M. polymorpha* quickly establish 3D thalli, which have meristematic stem cell regions at the apical notches (arrowheads) and outgrow through repetitive bifurcations of the notches (Inoue 1960; Shimamura 2016; Solly et al. 2017). Thalli produce single-celled rhizoids on the ventral surface for substrate anchoring and develop the assimilatory organ air chamber with a single air pore and the asexual reproductive organ gemma cup on the dorsal surface, in which multicellular elliptic discoid propagules, gemmae, with notches at both longitudinal tips are produced and kept dormant (Barnes and Land 1908; Apostolakos et al. 1982; Shimamura 2016; Kato et al. 2020b; Suzuki et al. 2020). Outside the cup, gemmae germinate and develop into thalli via gemmalings. Upon thallus excision, new thalli regenerate only from the pieces without apical notches. Male and female gametangiophores, consisting of stalks and heads, emerge from notches of thalli, which in turn develop sporophytes through fertilization (Shimamura 2016). Auxin-regulated processes are listed below each developmental stage. Illustrations of the largest, the second-largest sporelings, and the mature gemma are modified from Kny (1890). See also Thelander et al. (2018) for auxin responses in the moss *Physcomitrella patens*. * Auxin responses not examined in *M. polymorpha*.

germination (Fig. 1; Rousseau 1954; Otto and Halbsguth 1976).

Auxin is also involved in gemma dormancy within gemma cups. In the thalloid liverwort *Lunularia cruciata*, surgical excision of apical notches caused gemma germination within gemma cups on the remaining thalli, which was suppressed by IAA applied to the cut faces (LaRue and Narayanaswami 1957), suggesting

that mobile signals from apical notches keep gemmae dormant within gemma cups and that IAA is a potential candidate. *M. polymorpha* grown in the presence of L-Kyn showed an elevated ratio of gemma cups containing nondormant gemmae, supporting the concept that IAA facilitates gemma dormancy within gemma cups (Fig. 1; Eklund et al. 2015). Ectopic regeneration is also suppressed by apical notch-

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derived mobile signals, probably IAA, because excision of apical notches caused regeneration from the cutting faces of the remaining thalli, which was suppressed by IAA application in *L. cruciata* and *M. polymorpha* (Fig. 1; Vöchting 1885; LaRue and Narayanaswami 1957; Rota and Maravolo 1975; Nishihama et al. 2015). Suppression of regeneration by auxin has also been reported in several phylogenetically distant liverworts (Allsopp and Ilahi 1970c; Ilahi and Allsopp 1970b). Thus, in general, asexual propagation is prevented by auxin.

Inhibitory effects of IAA on apical notches are also observed in intact thalli. Davidonis and Munroe (1972) showed that in *M. polymorpha*, notch bifurcations often resulted in two lobes with different growth rates and that surgical separation at an early stage after bifurcation released the smaller lobe from inhibition, which was suppressed by IAA application. Lateral inhibition seems to occur between the bifurcated lobes mediated by IAA (Fig. 1).

Moss gametophores also show inhibition by the apex: lateral branch primordia and basal stem regeneration were reactivated by removing apices, both of which were suppressed by IAA application to the decapitated stem tips (MacQuarrie and von Maltzahn 1959; Coudert et al. 2015). Auxin is also involved in regeneration in the moss *Splachnum ampullaceum* because regeneration of isolated leaves was promoted or inhibited by IAA application at low or high concentrations, respectively (MacQuarrie and von Maltzahn 1959).

Thallus shapes are also affected by auxin as L-Kyn caused a dose-dependent reduction of thallus growth in *M. polymorpha* (Solly et al. 2017). In general, exogenous auxin strongly inhibits growth at high concentrations, but slightly enhances growth at low concentrations in liverworts (Ilahi and Allsopp 1969, 1970a; Allsopp and Ilahi 1970b; Ishizaki et al. 2012), although the detailed effects on thallus growth vary according to plant species and auxin types. In *Riccardia multifida*, various auxins promoted and inhibited thallus elongation and branching at low and high concentrations, respectively. In *Moerckia flotowiana*, *Pellia epiphylla*, and *Matkinoia crispata*, high auxin suppressed wing de-

velopment and causes cylindrical thalli (Ilahi and Allsopp 1969). These results suggest that auxin greatly contributes to overall plant shapes.

In vascular plants, auxin is known to be a major player in tropic responses to suit environmental cues (Retzer et al. 2014). In *M. polymorpha*, the dorsiventrality of gemmalings is determined by sensing gravity soon after gemma germination, and exogenous auxin disturbs proper dorsiventral determination (Fig. 1; for review, see Bowman 2016). Auxin was also reported to elongate and bend stalks of gametangiophores, although the correlation with gravitropic or phototropic responses was not discussed (Fig. 1; Rousseau 1953). In *P. patens*, auxin-mediated negative gravitropism in the dark and positive phototropism were observed in gametophores (Cove et al. 1978; Bennett et al. 2014a; Bao et al. 2015). In the moss *Ceratodon purpureus*, the auxin efflux inhibitors NPA and pyrenoylbenzoic acid (PBA) inhibited the gravitropic curvature of protonemal cells (Schwuchow et al. 2001).

Auxin functions in sporophyte generation. Auxin promoted seta elongation in the liverwort *P. epiphylla* and the moss *Polytrichum ohioense* (Fig. 1; Schnepf et al. 1979; Thomas 1980; Poli et al. 2003). In the hornworts *Anthoceros laevis* and *Anaxyrus punctatus*, 2,4-dichlorophenoxyacetic acid (2,4-D) inhibited the elongation of sporophytes but increased their diameter (Rousseau 1958). In the hornwort *Phaeoceros personii*, although the application of IAA failed to promote or impede sporophyte elongation, an auxin antagonist, *p*-chlorophenoxyisobutyric acid (PCIB), reduced sporophyte elongation (Poli et al. 2003). In *P. patens*, NPA has been reported to cause branching of sporangia (Fujita et al. 2008).

AUXIN HOMEOSTASIS: BIOSYNTHESIS, METABOLISM, AND TRANSPORT

Auxin levels are controlled by biosynthesis, temporary inactivation, permanent degradation, intercellular transport, and isolation into several intracellular compartments (not mentioned here). In vascular plants, IAA is a major intrinsic auxin, which is also detected in bryophyte spe-

cies and more basal algal sisters (Löbenberg 1959; Fries 1964; Schneider et al. 1967; Ashton et al. 1985; Atzorn et al. 1990; Cooke et al. 2002). In *Arabidopsis thaliana*, the indole-3-pyruvic acid (IPyA) pathway is known to be a major IAA biosynthesis route, where tryptophan is converted to IPyA and then to IAA. Each reaction is catalyzed by TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1/TRYP- TOPHAN AMINOTRANSFERASE-RELATED (TAA1/TAR) and YUCCA (YUC), respectively (for review, see Kasahara 2016), both of which are conserved in all three taxa of Bryophyta (Rensing et al. 2008; Eklund et al. 2015; Bowman et al. 2017; Li et al. 2020; Zhang et al. 2020). TAA1/TAR is inactivated by phosphorylation at an evolutionarily conserved Thr residue in an auxin-dependent manner. MpTAA, the sole TAA1 homolog in *M. polymorpha*, loses its enzymatic activity by a phosphorylation-mimicking mutation at the corresponding residue (Wang et al. 2020). The knockout of MpTAA at the sporeling stage results in a cell mass phenotype without any organ differentiation, which is partially rescued by exogenous IAA (Eklund et al. 2015). Likewise, the IPyA pathway mediated by PpTARs and PpYUCs is the main IAA biosynthesis route in *P. patens* (Landberg et al. 2020), which is consistent with competitive effects on caulonema differentiation between a YUC inhibitor and exogenous auxin (Tsugafune et al. 2017). These results suggest that the IPyA pathway and its regulatory systems exist in the common ancestor of land plants and that the IPyA pathway is a major IAA source and plays pivotal roles in the development and environmental responses of bryophytes. Indeed, MpTAA was reported to be regulated under the control of the circadian clock to regulate the nyctinastic movement of *M. polymorpha* thalli (Lagercrantz et al. 2020). However, other IAA biosynthetic pathways have not yet been the focus in bryophytes, although their existence is implied by the finding that *Mptaa* plants still contain detectable levels of IAA (Eklund et al. 2015).

In addition to IAA, phenylacetic acid (PAA) is detected in a broad range of plants, including angiosperms, *P. patens*, and *M. polymorpha* (Sugawara et al. 2015). In *Arabidopsis*, IAA and

PAA show different biochemical features, such as affinity to coreceptors and transport profiles. Although PAA shows auxin activity in *P. patens*, physiological differences between IAA and PAA remain unclear in bryophytes (Sugawara et al. 2015).

In *Arabidopsis*, IAA is temporarily inactivated by GRETCHEN HAGEN 3 (GH3)-mediated conjugation with amino acids (Staswick et al. 2005) and by UDP-glycosyltransferase-mediated ester linking with glucose (Jackson et al. 2001), or permanently inactivated by DIOXYGENASE FOR AUXIN OXIDATION 1 (DAO1)-mediated conversion into 2-oxindole-3-acetic acid (ox[IAA]; Porco et al. 2016; Zhang et al. 2016). Both amide- and ester-linked IAA conjugates were detected in the three Bryophyta taxa. oxIAA has been detected in some liverwort and moss species (Sztein et al. 1995, 1999, 2000; Ludwig-Müller et al. 2009b; Závěská Drábková et al. 2015; Landberg et al. 2020). Sztein et al. proposed that free-IAA levels are primarily regulated via the balance between biosynthesis and degradation in liverworts, whereas they are regulated by competitive conjugation versus hydrolysis of conjugates in hornworts and mosses, based on their observation that hornwort and moss species exhibited higher conjugation rates than liverwort species (Sztein et al. 1995, 1999, 2000; for review, see Cooke et al. 2002). However, Závěská Drábková et al. (2015) reached the opposite conclusion that liverworts preferred conjugation, whereas mosses favored degradation to maintain IAA homeostasis because liverwort species exhibited higher amino acid conjugation rates than moss species in their analysis. Although these studies could not be simply compared because of the differences in plant species and growth conditions, the conservation of both conjugation and degradation strategies to sustain IAA homeostasis in Bryophyta could be determined. Indeed, two GH3 homologs in *P. patens* have been shown to mediate IAA conjugation to amino acids (Ludwig-Müller et al. 2009a,b) and are thought to regulate free IAA levels under unfavorable environmental conditions (Mittag et al. 2015). Further molecular studies of GH3 and other IAA metabolic enzymes, which are conserved in the genomes of

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bryophytes (Rensing et al. 2008; Bowman et al. 2017; Li et al. 2020; Zhang et al. 2020), are needed to clarify the strategies for IAA homeostasis.

In *Arabidopsis*, in addition to several active auxin transporters, including PIN-FORMED (PIN) (for review, see Grønes and Friml 2015), a plasmodesmatal auxin transport controlled by callose deposition has been suggested, which occurs in *P. patens* (Han et al. 2014; Coudert et al. 2015). *P. patens* encodes polarly membrane-localized long-PIN homologs (PpPINA/B/C) and an endoplasmic reticulum-localized short-PIN homolog ([PpPIND]; Viaene et al. 2013, 2014; Bennett et al. 2014a,b). Generally, mutations of the former result in an auxin-excessive phenotype, whereas their overexpression results in auxin-repressive phenotypes, which are probably caused by trapping and overdrainage of IAA, respectively (Bennett et al. 2014b; Viaene et al. 2014). These facts suggest that the long PpPINs act as efflux carriers. Indeed, basipetal polar transport could be detected by tracer assays of radiolabeled IAA within multicellular rhizoids of the moss *Funaria hygrometrica* (Fig. 2A; Rose and Bopp 1983). Tracer assays did not demonstrate polar transport in stalks of gametophores along the longitudinal axis but did show basipetal polar transport in setae of several mosses (Fig. 2B; Poli et al. 2003; Fujita et al. 2008). Meanwhile, based on the velocity and direction of transport, IAA is thought to be carried by nonpolar active transport or simple diffusion in setae of liverworts or young sporophytes of hornworts, respectively (Fig. 2C), suggesting that auxin transport strategies in sporophytes were independently evolved in bryophytes. Radiolabeled IAA was transported basipetally in thalli of *M. polymorpha* (Binns and Maravolo 1972; Maravolo 1976; Gaal et al. 1982), which was inhibited by 2,3,5-triiodobenzoic acid, a transport inhibitor, suggesting that an active transporter(s) basipetally carries IAA (Fig. 2D; Maravolo 1976).

PRINCIPLE OF AUXIN SIGNALING

Auxin regulates gene expression through the NAS pathway, comprising TRANSPORT INHIBITOR RESPONSE 1/AUXIN SIGNALING

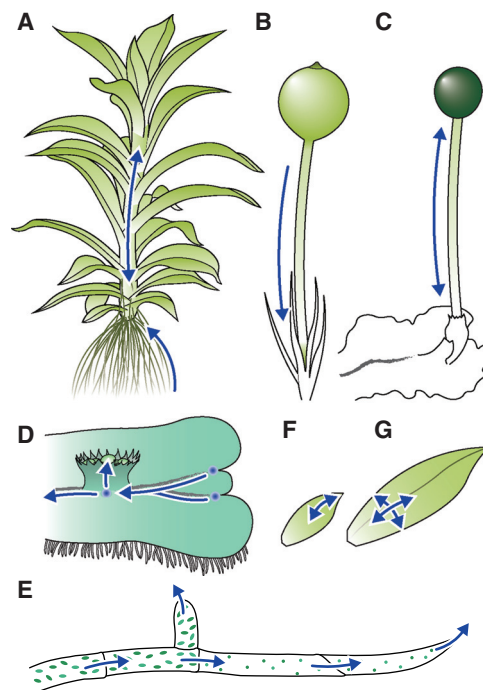


Figure 2. Auxin transport in bryophytes. Estimated auxin transport (blue arrows) in stalks (A), rhizoids (A), setae (B), protonemata (E), young leaves (F), and larger leaves (G) in mosses, and setae (C) and thalli (D) of liverworts.

F-BOX (TIR1/AFB) receptors, AUXIN/INDOLE-3-ACETIC ACID (AUX/IAA) transcriptional repressors, and AUXIN RESPONSE FACTOR (ARF) transcription factors (Fig. 3A; Table 1). TIR1/AFBs, an F-box-containing subunit of an SCF-type E3 ubiquitin ligase complex, form coreceptor complexes with AUX/IAAs via auxin, which in turn facilitate ubiquitination and subsequent degradation of AUX/IAAs (Gray et al. 1999, 2001; Dharmasiri et al. 2005; Kepinski and Leyser 2005; Tan et al. 2007). Canonical AUX/IAAs have three conserved domains: domain I (DI), domain II (DII), and the Phox and Bem1 domain ([PB1], also called domain III/IV). Each of these interacts with TOPLESS (TPL)-type corepressors, TIR1/AFBs, and PB1 domains of ARFs and AUX/IAAs, respectively (Kim et al. 1997; Dharmasiri et al. 2005; Kepinski and Leyser 2005; Szemenyei et al. 2008). DII is known as the degron region. ARFs consist of a DNA-binding

domain (DBD), a middle region (MR), and a PB1 domain. Each of these binds to *cis*-elements, regulates transcriptional activity, and homo- and heterotypically interacts with ARFs and AUX/IAAs, respectively (Kim et al. 1997; Ulmasov et al. 1997, 1999a,b; Tiwari et al. 2003). Auxin releases ARFs from AUX/IAAs via TIR1/AFB-mediated degradation and hence facilitates ARF-mediated transcriptional regulation. Phylogenetic analyses demonstrated NAS establishment in the common ancestor of land plants (Bowman et al. 2017; Flores-Sandoval et al. 2018a; Mutte et al. 2018).

In *P. patens*, four TIR1/AFB and three AUX/IAA homologs interact with each other in an auxin-dependent manner (Prigge et al. 2010). *P. patens* expressing stabilized mutants of PpIAA, which had been originally forward-genetically isolated as auxin-insensitive mutants, and PpAFB knockdown plants showed auxin depletion phenotypes, confirming that PpAFB–PpIAA pairs act as auxin coreceptors (Ashton et al. 1979; Prigge et al. 2010). *M. polymorpha* encodes a single canonical AUX/IAA homolog, MpIAA. Overexpression of a DII-mutated MpIAA protein conferred

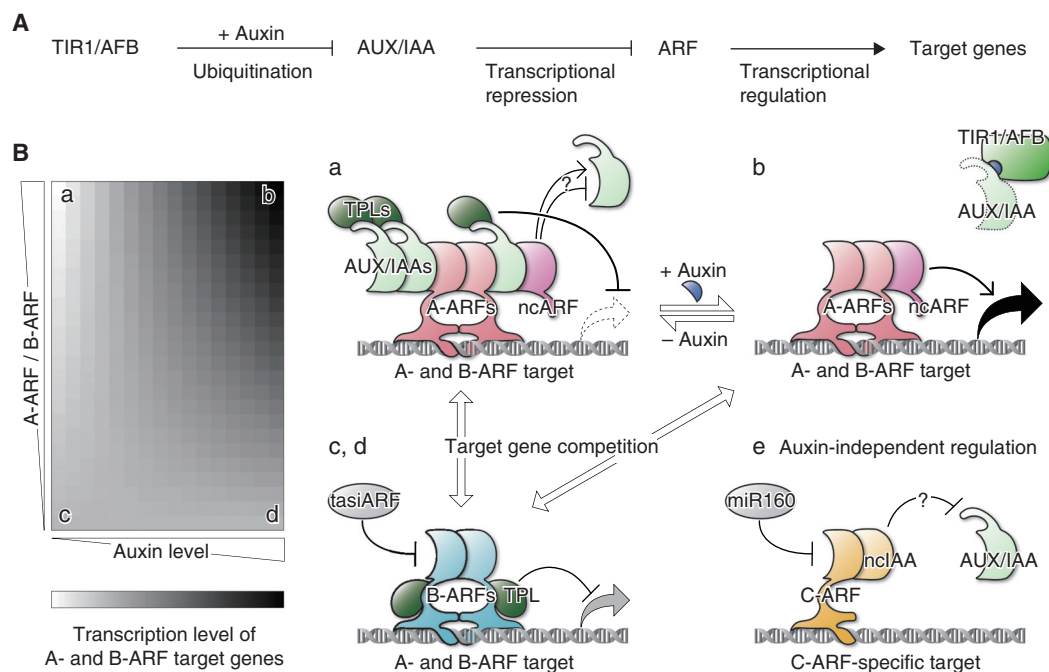


Figure 3. Deduced design principles of nuclear auxin signaling (NAS) from studies with bryophytes. (A) Components and basic scheme of the NAS pathway. (B) Heat map showing putative expression levels of A- and B-ARFs' common target genes (left) and functional models of the NAS components (right) at different A-ARF/B-ARF ratios and auxin levels (a–d). A-ARFs (and noncanonical ARFs [ncARFs]) recruit AUX/IAAs and thus repress target gene transcription at low auxin levels (a). A-ARFs (and ncARFs) activate target gene transcription at high auxin levels as TIR1/AFBs interact with AUX/IAAs via auxin and promote AUX/IAA degradation (b). B-ARFs buffer AUX/IAA-mediated intense repression at low auxin levels (c) and A-ARF-mediated activation at high auxin levels (d) by competing with A-ARFs for binding to their target DNA sites, which fine-tunes auxin responses. C-ARFs neither share target DNA sites with A- or B-ARFs, nor interact with AUX/IAAs, and thus regulate distinct target genes in an auxin-independent manner (e). ncIAA may prevent canonical AUX/IAAs from binding to C-ARFs. Note that the functions of ncARFs and ncIAAs are hypothetical. These models are based on the following papers and our deduction, in addition to the well-known NAS model established in angiosperms (A): Flores-Sandoval et al. (2018a,b), Kato et al. (2017, 2020a), Lavy et al. (2016), Lv et al. (2020), Mutte et al. (2018), and Tao and Estelle (2018).

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Table 1. Copy numbers and IDs of the nuclear auxin signaling (NAS) component homologs in bryophytes

	<i>Marchantia polymorpha</i>	<i>Physcomitrium patens</i>	<i>Anthoceros angustus</i>	<i>Anthoceros agrestis</i> (Bonn)	<i>Anthoceros agrestis</i> (Oxford)	<i>Anthoceros punctatus</i>	<i>Arabidopsis thaliana</i>
TIRI/AFB	1 (MpTIR1; Mp6g02750)	4 (PpAFB1; Pp3c23_5870, PpAFB2; Pp3c20_17150, PpAFB3; Pp3c23_11300, PpAFB4; Pp3c24_9800/ Pp3c24_9814)	1 (AANG003364)	1 (AagrBONN344.691)	1 (AagrOXF000012L.465)	1 (Apun000117L.230)	6
AUX/IAA	1 (MpIAA; Mp6g05000)	3 (PpIAA1A; BAB71765.1, PpIAA1B; BAB71766.1/ Pp3c8_14720, PpIAA2; EDQ70046/ Pp3c24_6610)	1 (AANG009773)	1 (AagrBONN117.2250)	1 (AagrOXF000103L.142)	1 (Apun000023L.749)	28
ncIAA	1 (MpNCIAA/ MpAXL2; Mp8g07850)	0	1 (AANG012370)	1 (AagrBONN117.1187)	1 (AagrOXF000083L.132)	1 (Apun000058L.129)	1
TPL/TPL-RELATED	1 (MpTPL; Mp7g17410)	2 (PpTPL1; Pp3c15_9880, PpTPL2; Pp3c9_21250)	1 (AANG014214)	1 (AagrBONN340.1726)	1 (AagrOXF000065L.67)	1 (Apun000149L.87)	4

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Table 1. Continued

	<i>Marchantia polymorpha</i>	<i>Physcomitrium patens</i>	<i>Anthoceros angustus</i>	<i>Anthoceros agrestis</i> (Bonn)	<i>Anthoceros agrestis</i> (Oxford)	<i>Anthoceros punctatus</i>	<i>Arabidopsis thaliana</i>
A-ARF	1 (MpARF1; Mp1g12750)	8 (PpARFa1; Pp3c1_14480, PpARFa2; Pp3c1_14440, PpARFa3; Pp3c2_25890, PpARFa4; Pp3c13_4720, PpARFa5; Pp3c26_11550, PpARFa6; Pp3c17_19900, PpARFa7; Pp3c14_16990, PpARFa8; Pp3c1_40270)	1 (AANG012813)	1 (AagrBONN340.233)	1 (AagrOXF0001261.206)	1 (Apun0000451.202)	5
B-ARF	1 (MpARF2; Mp4g11820)	4 (PpARFb1; Pp3c27_60, PpARFb2; Pp3c16_6100, PpARFb3; Pp3c5_9420, PpARFb4; Pp3c6_21370)	0	0	0	0	15
C-ARF	1 (MpARF3; Mp1g07070)	3 (PpARFc1A; Pp3c4_12970, PpARFc1B; Pp3c4_13010, PpARFc2; Pp3c6_26890)	3 (AANG000742, AANG000743, AANG012642)	4 (AagrBONN340.199, AagrBONN344.1784, AagrBONN369.337, AagrBONN369.338)	4 (AagrOXF0000061.365, AagrOXF0000691.94, AagrOXF0000691.95, AagrOXF0001261.173)	4 (Apun0000451.242, Apun0000891.87, Apun0000891.88, Apun0001441.74)	3

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Table 1. Continued

	<i>Marchantia polymorpha</i>	<i>Physcomitrium patens</i>	<i>Anthoceros angustus</i>	<i>Anthoceros agrestis</i> (Bonn)	<i>Anthoceros agrestis</i> (Oxford)	<i>Anthoceros punctatus</i>	<i>Arabidopsis thaliana</i>
Noncanonical (nc)ARF	1 (MpNCARF/ MpAXII; Mp2g02890)	2 (PpARFd1; Pp3c6_26890, PpARFd2; Pp3c15_9710)	0	0	0	0	0

Homologs were obtained from MarpollBase (www.marchantia.info) for the liverwort *M. polymorpha*, and from Phytozome v13 (www.phytozome-next.jgi.doe.gov) and NCBI (www.ncbi.nlm.nih.gov) for the moss *P. patens* from DRYAD (www.datadryad.org/stash/dataset/doi:10.5061/dryad.msbcc2ftv) for hornwort *A. angustus*, from the University of Zurich (www.hornworts.uzh.ch/en.html) for the hornwort *A. agrestis* (Bonn), *A. agrestis* (Oxford), and *A. punctatus*. AgROXF_evm.model.utg0000081.194.1 and Apun_evm.model.utg0000391.220.1 are omitted from TIR1/AFB candidates here, since they are closer to PpXFBs (Pp3c22_6020 and Pp3c18_16630), which are F-box/Leucine Rich Repeat proteins absent in angiosperms (Prigge et al. 2010) and are more homologous to jasmonate/oxo-phytodienoic acid receptors than TIR1/AFBs (see a phylogenetic tree in Mutte et al. 2018). Names: “AggrBONN_evm.model.Sc2ySwM_XXX.XXXX,” “AgROXF_evm.model.utgXXXXX1.XX,” and “Apun_evm.model.utgXXXXX1.XXX” are modified as “AggrBONNXXX.XXXX,” “AgROFXXXXX1.XX,” and “ApunXXXXX1.XXX,” respectively, to save space.

auxin tolerance, suggesting a conserved role of MpIAA in auxin perception (Kato et al. 2015).

Although several studies have used stabilized, dominant mutants of AUX/IAAs for functional analyses, reports of their loss-of-function phenotypes were limited. Lavy et al. (2016) performed a prominent mutational study with AUX/IAA-null mutants in *P. patens* in which all three *PpIAA* homologs were knocked out. The *Ppiaa* triple mutants showed no responses to exogenous auxin or L-Kyn, leading to an excessive auxin-response phenotype. Expression of a PpIAA1a mutant incapable of binding to TPL, which lacked an ethylene-responsive element binding factor-associated amphiphilic repression (EAR) motif (Causier et al. 2012) within DI and an EAR-like motif between DI and DII, still caused an excessive auxin phenotype, but slightly restored growth and auxin responsiveness, suggesting EAR-independent repressive functions of AUX/IAAs (Tao and Estelle 2018). Considering that *Arabidopsis* ARF5/MONOPTEROS interacts with chromatin remodeling ATPases to exert transcriptional regulation (Wu et al. 2015), simple binding of AUX/IAAs to ARFs could inhibit recruitment of other required factors, thus repressing ARF functions (Tao and Estelle 2018). A PB1 mutant of PpIAA1a, incapable of binding to itself nor PpARFs, failed to complement the null *Ppiaa* mutant. However, another PB1 mutant of PpIAA1a, incapable of self-binding but capable of binding to PpARFs, slightly rescued the mutant. Taken together, the PB1-mediated heterotypic interaction with ARFs is critical for the function of AUX/IAA, and their homotypic interactions and subsequent oligomerization contribute to, but are not essential for, their repressive functions (Tao and Estelle 2018).

ARF transcription factors are phylogenetically classified as A, B, and C (Finet et al. 2013). Comprehensive phylogenetic analysis in green plants showed that ancestral C-ARFs diverged from ancestral A/B-ARFs before the TIR1/AFB–AUX/IAA auxin coreceptor system (Flores-Sandoval et al. 2018a; Mutte et al. 2018). Based on sequence traits in the MR and results of transactivation assays, A- and B-ARFs are generally thought to be transcriptional activa-

tors and repressors, respectively (Ulmasov et al. 1999a; Tiwari et al. 2003; Kato et al. 2015). Overexpression of A- and/or B-ARF homologs in the *Ppiaa* triple mutant revealed that A- and B-ARFs share common target genes and that B-ARFs exert repressive functions in an AUX/IAA-independent manner (Lavy et al. 2016). Counterintuitively, knockout of B-ARFs reduced auxin sensitivity, and overexpression of A-ARFs down-regulated the transcription of some auxin-responsive genes. Based on these conflicting results, Lavy et al. (2016) proposed a model in which B-ARFs compete for target gene sites with A-ARFs, and affect the occupancy of A-ARFs and AUX/IAAs, providing a buffering capacity for fine-scale regulation of target genes.

M. polymorpha encodes three canonical ARFs, each belonging to class A, B, or C (Flores-Sandoval et al. 2015; Kato et al. 2015; Bowman et al. 2017). Knockout of MpARF1, the sole A-ARF, conferred auxin insensitivity and caused various developmental defects, including narrow twisted thalli, small tubular gemma cups, decreased gemmae per cups, nondormant gemmae, and disrupted patterning in gemma development (Kato et al. 2017). Interestingly, inhibition of NAS via DII-mutated nondegradable MpIAA resulted in very severe growth defects, such as formation of cell masses, ectopic air pores, ectopic serrated structures, and/or adventitious buds, and failure to develop thalli (Kato et al. 2015). Furthermore, the aforementioned *Mptaa* auxin biosynthesis mutant or overexpressors of a bacterial auxin-conjugating enzyme formed cell masses (Eklund et al. 2015). *Mparf1* phenotypes are much milder than those in that they develop thaloid organs. One possible explanation is that AUX/IAA proteins are not recruited to target sites in the absence of the A-ARF, allowing its target genes to be transcribed at basal levels (Kato et al. 2017, 2018).

Kato et al. (2020a) investigated whether the conserved domains of MpARFs could be interchangeable by expressing various domain-swapped constructs in the *Mparf1* mutant background. Chimeric MpARF1, which exchanged the DBD with MpARF2 but not MpARF3, restored development and auxin responsiveness,

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suggesting that MpARF1 and MpARF2 share target genes, while MpARF3 targets another group of genes. The MR of MpARF1 was interchangeable with that of neither MpARF2 nor MpARF3. Interestingly, MpARF2 was shown to interact directly with MpTPL through a conserved “LFG motif,” and small fragments containing the LFG motif but not a mutated motif from all MpARFs exhibited repressor activities upon coexpression with MpTPL. Thus, the MR of MpARF2 (and probably that of MpARF3) exerts auxin-independent transcriptional repression via direct recruitment of MpTPL (Kato et al. 2020a).

All combinations of homo- and heterotypic interactions among the PB1 domains of MpARFs and MpIAA were demonstrated except for MpARF3–MpARF3 homotypic interactions (Kato et al. 2015). In the domain-swapping assay, the PB1 domain of MpARF1 was not interchangeable with that of MpARF3. Chimeric MpARF1 with its PB1 domain swapped with that of MpARF2 partially rescued developmental defects of *Mparf1* but did not rescue responsiveness to exogenous auxin. In general, PB1 domains have positive and negative faces and oligomerize through consecutive interactions between the two faces (Korasick et al. 2014). Docking of structural models, however, predicted that both the positive and negative faces of MpARF2 and MpARF3 PB1 domains interact with one face of the MpIAA PB1 domain, while one face of the MpARF1 PB1 domain does so, allowing further oligomeric interactions, which seems critical for establishing sufficient auxin responsiveness. Chimeric MpARF1, which has a distinct oligomerization domain in place of the PB1 domain, rescued *Mparf1* growth defects but not auxin insensitivity, suggesting that MpARF1–MpARF1 homotypic interactions through the PB1 domain are critical for transcriptional activation by MpARF1 and that auxin response requires MpARF1–MpIAA oligomerization (Kato et al. 2020).

These results offer design principles of the auxin response system: in the absence of auxin, A-ARFs oligomerize with AUX/IAAs causing transcriptional repression, while in the presence of auxin, A-ARFs interact with each other and

promote target transcription. B-ARFs compete for target sites with A-ARFs and A-ARF–AUX/IAA complexes to fine-tune auxin responsiveness (Kato et al. 2020a). Even though B-ARFs recruit TPLs, B-ARFs are speculated to exert weaker repression than A-ARF–AUX/IAA complexes, as the latter is capable of recruiting many more TPLs via oligomerized AUX/IAAs. Thus, B-ARFs could buffer both transcriptional activation and repression at high and low levels of auxin, respectively (Fig. 3B).

B-ARFs are known to be posttranscriptionally regulated by *trans*-acting small interfering RNA (tasiRNA) generated by miR390 from *TAS3* gene products in angiosperms (Allen et al. 2005; Williams et al. 2005). *P. patens* and *M. polymorpha* also have conserved miR390, suggesting the existence of a similar regulatory system (Axtell et al. 2007; Arif et al. 2012; Lin et al. 2016; Plavskin et al. 2016; Xia et al. 2017). In *P. patens*, it is demonstrated that *TAS3*-derived tasiRNAs target transcripts of B-ARF genes to restrict their expression at a random subset of protonemal tip cells, which increases sensitivity and robustness of auxin responses. The tasiARFs thus allow stochastic modulation of cell differentiation and developmental plasticity in response to environmental cues (Plavskin et al. 2016). In *M. polymorpha*, conditional knockout of MpARF2 in gemmalings or thalli caused severe growth arrest, suggesting that B-ARF plays critical roles in development (Kato et al. 2020a). Interestingly, hornworts do not encode a B-ARF in their genome, which was probably lost after divergence from a liverwort/moss common ancestor (Flores-Sandoval et al. 2018a; Mutte et al. 2018; Li et al. 2020; Zhang et al. 2020), suggesting the acquisition of a distinct buffering mechanism for A-ARFs.

C-ARFs are posttranscriptionally regulated by miR160 (Mallory et al. 2005; Wang et al. 2005), whose target sites are conserved among land plants (Axtell et al. 2007; Lin et al. 2016; Tsuzuki et al. 2016; Flores-Sandoval et al. 2018a). In contrast to MpARF1 and MpARF2, neither knockout nor overexpression of MpARF3 affected auxin sensitivity, unless its miR160-resistant form is overexpressed, leading to auxin-insensitive cell masses (Flores-



Sandoval et al. 2018a). Additionally, MpARF3 is not a part of the auxin-related coexpression module (Flores-Sandoval et al. 2018b). Together with the phylogenetic position, these studies suggest that C-ARFs are not under the control of auxin; rather, it is proposed that the TIR1/AFB–AUX/IAA- and miR160-mediated pathways evolved independently to regulate pre-existing A/B- and C-ARF transcriptional networks, respectively (Flores-Sandoval et al. 2018a,b). Indeed, as the DBDs of MpARF1 and MpARF3 were unexchangeable, many, if not all, A- and B-ARFs seem to regulate gene sets distinct from target genes of C-ARFs (Kato et al. 2020a).

Phylogenetic analysis indicated the existence of conserved noncanonical AUX/IAAs (ncIAAs) lacking DI and DII (Flores-Sandoval et al. 2018a; Mutte et al. 2018), although ncIAA homologs are absent from *P. patens*. Liverworts, mosses, and lycophytes, but not hornworts and other vascular plants, have noncanonical ARFs (ncARFs) lacking DBD, which diverged from A-ARFs (Paponov et al. 2009; Mutte et al. 2018). *M. polymorpha* has one ncIAA, MpNCIAA/MpAXI2 and one ncARF, MpNCARF/MpAXI1 (Bowman et al. 2017). Although Mpncarf mutants developed relatively normal thalli, they showed tolerance to exogenous auxin and reduced fold changes of some auxin-up-regulated genes (Mutte et al. 2018). Based on these results, Mutte et al. (2018) proposed two hypothetical roles of ncARFs: ncARFs protect canonical ARFs from AUX/IAAs and/or recruit cofactors to ARF-target loci via interaction with canonical ARFs. In contrast to the first model, given that oligomeric interactions of AUX/IAAs to A-ARFs are required for sufficient auxin responsiveness (Kato et al. 2020a; see above), ncARFs may assist the A-ARF–AUX/IAA interaction as spacers (Fig. 3B). Meanwhile, the roles of MpNCIAA in NAS are unclear, as Mpnciaa mutants showed comparable auxin responses to wild-type (Mutte et al. 2018). AtIAA33, the sole ncIAA ortholog in *Arabidopsis*, has recently been demonstrated to interact with C-ARFs, AtARF10/16, which protects AtARF10/16 from canonical AUX/IAA-mediated repression (Lv et al. 2020). Additionally, like MpARF3,

MpNCIAA is expressed independently of the auxin coexpression group (Flores-Sandoval et al. 2018b). Although further experiments are needed, ncIAAs may play a role in sequestering C-ARFs from NAS (Fig. 3B).

MOLECULAR MECHANISMS UNDERLYING DEVELOPMENTAL REGULATION

As seen in the previous sections, auxins and their inhibitors cause multiple responses in bryophytes. For these context-dependent, diverse auxin responses, its localization and responsiveness should be spatiotemporally controlled. Accordingly, reporters of the soybean *GmGH3* promoter (Hagen et al. 1991), which operate under the control of NAS also in a bryophyte (Kato et al. 2015, 2017), exhibit high activities in multiple tissues or organs throughout the life cycle in both *P. patens* (Bierfreund et al. 2003; Fujita et al. 2008; Landberg et al. 2013) and *M. polymorpha* (Ishizaki et al. 2012). Additionally, the synthetic auxin response reporter *DR5revV2* shows correlated expression patterns with those of *GmGH3* (Thelander et al. 2019). As an ARF-independent auxin-sensing reporter, a moss-optimized “ratiometric version of two D2s,” PpR2D2, was recently developed. PpR2D2 semiquantitatively visualizes auxin sensitivities as AUX/IAA degradation rates and enables further fine monitoring in *P. patens* (Liao et al. 2015; Thelander et al. 2019).

In protonema colonies of *P. patens*, PpR2D2 shows a gradient along filaments where auxin-sensing levels are high in proximal cells and low in tip cells (Thelander et al. 2019). Both the *GmGH3* and *DR5revV2* reporters show stronger activity in proximal cells than in tip cells (Thelander et al. 2019). Thus, apical stem cells show relatively low auxin sensing and response levels. Polar localization of long PINs on the plasma membrane toward tips suggests acropetal auxin transport and its accumulation in and drainage from tip cells, which is needed for proper caulonema differentiation (Fig. 2E; Viaene et al. 2014). Although the auxin source is still unclear, PpSHI, a positive regulator of auxin biosynthesis homologous to *Arabidopsis* SHORT INTER-NODES/STYLISH ([SHI/STY]; Sohlberg et al.

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2006; Ståldal et al. 2008; Eklund et al. 2010a), is specifically expressed in caulonema (Eklund et al. 2010b). The above-mentioned B-ARF gradient generated by tasiARFs adds another tier of auxin response regulation for caulonema differentiation and bud formation (Plavskin et al. 2016). Under the control of auxin, homologs of ROOT HAIR DEFECTIVE 6-LIKEs and *Lotus japonicus* Roothairless Like (PpRSL1/2 and PpLRL1/2, respectively) function in caulonema differentiation (Jang and Dolan 2011; Tam et al. 2015). For gametophore development, auxin-responsive transcription factors, AINTEGUMENTA, PLETHORA, and BABY BOOM homologs (PpAPBs), act as master regulators of bud formation (Aoyama et al. 2012).

Consistent with the undetectable polar transport with radiolabeled IAA in gametophore stems (Fujita et al. 2008), *Pppina/b* double mutants, and NPA treatment have only a minor effect on branching patterns; rather, plasmodesmata are supposed to be a major transport route (Coudert et al. 2015). Nevertheless, since long *PpPIN* mutants exhibit a multifaceted phenotype in gametophores, such as stem elongation, leaf abnormalities, and disruption of tropic responses, PIN-mediated transport also plays pleiotropic roles in gametophores (Bennett et al. 2014b). During leaf development, PpPINA/B are expressed in apical regions and show bipolar localization on both apical and basal plasma membranes at the early stages. The expression zones move toward the basal region as leaves develop, where PpPINA/B localize on all sides. Such a shift in expression zones correlates with a similar shift in cell elongation (Viaene et al. 2014). The PpR2D2 reporter indicated that high auxin-sensing zones also expand from apex to base during leaf development (Fig. 2F,G; Thelander et al. 2019). Auxin application at a certain concentration transforms leaves into narrow, elongated shapes (Barker 2011; Bennett et al. 2014b). Knockout of long PINs also caused long narrow leaves because of enhanced longitudinal cell elongation and reduced mediolateral cell division, probably caused by auxin trapping (Bennett et al. 2014b). Taken together, long PINs affect auxin responsiveness via transport, which regulates the transition from cell division to expansion

in leaf development (Bennett et al. 2014b; Viaene et al. 2014; Thelander et al. 2019).

Auxin promotes rhizoid development in both liverworts and mosses. In *P. patens*, the PpR2D2 reporter revealed that auxin-sensing levels are low in rhizoid precursors and show a gradient from proximal to tip after the three-cell stage (Thelander et al. 2019). Consistently, *GmGH3* and *DR5revV2* reporters show gradient patterns in multicelled stages, although they are also active in precursors (Thelander et al. 2019). On the other hand, *PpSHI* is highly expressed in tip cells (Eklund et al. 2010b). Considering the basipetal transport of radiolabeled IAA in *F. hygrometrica* rhizoids (Rose and Bopp 1983), it is suggested that IAA is biosynthesized in tip cells, is transported toward proximal regions, and then evokes signaling in rhizoids of mosses (Fig. 2A). In *P. patens*, mutations in the auxin-inducible *PpRSL1/2* or *PpLRL1/2* genes (see above) result in loss of rhizoid development regardless of auxin treatment, suggesting that these basic helix-loop-helix (bHLH) transcription factors regulate rhizoid development downstream of auxin (Menand et al. 2007; Jang et al. 2011; Tam et al. 2015). Their angiosperm homologs regulate root hair development (Mascucci and Schiefelbein 1994; Menand et al. 2007; Ding et al. 2009; Karas et al. 2009). In particular, the class II RSL genes are regulated by auxin in *Arabidopsis* (Yi et al. 2010; Pires et al. 2013). It is known that their *M. polymorpha* homologs, MpRSL1 and MpLRL, also regulate rhizoid development, but their association with auxin is unclear (Breuninger et al. 2016; Proust et al. 2016).

In the thalli of *M. polymorpha*, both MpTAA and MpYUC2 promoters show strong activity at apical notches and the bottom of gemma cups (Eklund et al. 2015). In line with the observations that removal of apical notches broke gemma dormancy in the cup (LaRue and Narayanaswami 1957; Eklund et al. 2015), IAA biosynthesized at the bottom of gemma cups but also that supplied from apical notches (Maravolo 1976; Gaal et al. 1982) are suggested to play roles in maintaining gemmae dormancy. An increase in nondormant gemmae caused by MpYUC2 knockdown, overexpression of



iaaL, and *MpARF1* knockout also supports the notion that auxin promotes gemma dormancy (Eklund et al. 2015; Kato et al. 2017). Regulation of dormancy, however, seems to be a complex process as direct IAA application broke gemma dormancy within gemma cups (Tarén 1958). Additionally, the question remains as to how auxin affects mature gemmae detached from mother plants but still in the cup. Recently, abscisic acid (ABA) was shown to function autonomously in gemmae to initiate and/or maintain dormancy within gemma cups (Eklund et al. 2018), but the underlying mechanisms bridging the two signaling pathways remain unclear.

Auxin also mediates cell elongation in *M. polymorpha*. Gametangiophore stem elongation was suppressed by high doses of nondegradable *MpIAA* and dominant-negative *TPL* mutants (Flores-Sandoval et al. 2015). The former also caused defects in the antigravitropic bending of stalks, supporting that the *NAS* pathway regulates tropic responses (Kato et al. 2015). Additionally, it suppressed auxin-induced protrusion of air pores and elongation of gemma cup cells, suggesting that the *NAS* pathway regulates cell elongation. Several *EXPANSIN* genes shown to be up-regulated in response to auxin treatment might be potential targets (Kato et al. 2017; Mutte et al. 2018).

Analysis of the *NAS* components revealed that auxin also plays a significant role in apical stem cell functions. Consistent with the fact that auxins promote branching in *R. multifida* (Ilahi and Allsopp 1969), overexpression of *MpARF1* and the dominant-negative *MpTPL* mutant enhanced thallus branching (Flores-Sandoval et al. 2015). Conditional knockout of *MpARF2* terminated thallus growth, suggesting that coordinated regulation between A- and B-ARFs is essential for stem cell maintenance (Kato et al. 2020a). *Mparf1* mutation causes abnormalities in division patterns and deviation of position and numbers of stem cell regions during gemma formation, suggesting that the establishment of developmental axes through formative divisions is a common trait of A-ARFs in land plants (Yoshida et al. 2014; Kato et al. 2017, 2018). A bHLH transcription factor, *M. polymorpha* TARGET OF MONOPTEROS 5 (*MpTMO5*),

is also needed for proper patterning in gemma development (Lu et al. 2020). Given that *Arabidopsis TMO5* is a direct target of A-ARF (Schlreth et al. 2010), it would be worthwhile to test whether *MpARF1* targets *MpTMO5* in gemma development.

In sporophytes, strong *GmGH3* activities were observed in the whole or apical part of *M. polymorpha* and *P. patens*, respectively, at the immature stage, but limited to the basal side as they mature (Fujita et al. 2008; Ishizaki et al. 2012). In *M. polymorpha*, nondegradable *AUX/IAA* induction halts sporophyte development (Kato et al. 2015). In *P. patens*, transport disruption by *NPA* application or knockout of *PpPINB* occasionally caused the branching of sporangia, and knockout of both *PpPINA* and *PpPINB* caused severe or lethal growth arrest (Fujita et al. 2008; Bennett et al. 2014b). These studies highlight the importance of spatiotemporal auxin regulation in the sporophyte bodies of bryophytes. Although auxin response in sporophytic tissues plays an essential role in angiosperms, as shown by the embryonic lethal phenotype of *AtTIR1/AFB* sextuple mutants (Prigge et al. 2020), little is known about the molecular mechanisms underlying sporophyte development of bryophytes. Future analyses are needed to determine whether conserved regulatory systems are used for sporophytes.

CONCLUDING REMARKS

Studies with bryophytes have contributed to our understanding of how the auxin system evolved in green plants. As described above, bryophytes share with vascular plants, not only the core mechanisms of auxin biosynthesis, polar transport, inactivation, and signaling, but also various auxin-regulated physiological processes, such as cell division, cell elongation, cell differentiation, tissue and organ development, and tropic responses. Thus, most of the auxin system was established early in land plant lineages, which reemphasizes the utility of bryophytes as models for auxin biology. In particular, the design principle of the functions of the *NAS* components has been determined effectively using simple platforms.

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Auxin plays a role in reproductive organ differentiation in bryophytes. In *P. patens*, *PpTARs* regulate gamete precursor development by local auxin biosynthesis (Landberg et al. 2020); *PpSHI* regulates egg cell differentiation, sexual organ opening, and programmed canal cell death (Landberg et al. 2013). In *M. polymorpha*, *MpIAA* is expressed in gametangia (Kato et al. 2015). In bryophytes, germline cells are formed directly on vegetative somatic tissues, distinct from seed plants where they are specified in meiosis-derived cells within several rounds of divisions (Hisanaga et al. 2019). Elucidation of the roles of auxin in reproductive development should provide insights into the reduction of gametophyte generation during land plant evolution. Further analyses are needed on how auxin biosynthesis, transport, signaling, and inactivation are used in various contexts in bryophytes.

Auxin is regarded as a signal that fuels programs predetermined by other mechanisms (Bennett and Leyser 2014) most likely via epigenetic regulation. However, its underlying biochemical basis has not been fully elucidated. For example, little is known regarding how A-ARFs reverse the deacetylated histone status. Additionally, there are still many unanswered questions on the functions of the NAS components, such as whether all or only a subset of A-ARF-target genes are coregulated by B-ARFs and whether and how the ratio of A- and B-ARFs varies for individual target genes. Simple auxin systems in bryophytes can make significant contributions to these central issues in auxin biology. So far, the majority of molecular studies on bryophytes have been reverse genetically performed. Given the low genetic redundancy, forward genetics would provide clues to explore novel molecular mechanisms for auxin biology.

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