REVIEW ARTICLE

Axillary Meristem Initiation and Bud Growth in Rice

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Abstract

The morphology of a plant is not predestined during embryonic development and its myriad branching architecture is post-embryonically determined by axillary meristem (AM) activity as the plant adapts to a varying environment. The shoot apical meristem produced during embryonic development repetitively gives rise to a phytomer that comprises a leaf, an AM, and an internode. The final aerial architecture is regulated by shoot branching patterns derived from AM activity. These branching patterns are modulated by two processes: AM formation and axillary bud outgrowth. Several transcription factors (TFs) that regulate these processes have been identified. Various plant hormones, including strigolactones and auxin, have major roles in controlling these TFs. In this review we focus on molecular mechanisms that guide AM initiation and axillary bud growth, using rice as our main species of emphasis.

Keywords: Axillary meristem, Hormones, Rice, Shoot branching, Tillering

Introduction

Shoot branching determines plant architecture as well as biomass and yield (Tian and Jiao 2015). Branching is one strategy by which plants adapt to environmental changes (Graham et al. 2000). The shoot apical meristem (SAM) and axillary meristem (AM) cooperate in the aerial branching system and all post-embryonic organs originate from these meristematic tissues (Yang and Jiao 2016). During the embryonic stage, SAM establishes the primary axis that will eventually develop into shoot structures (Pautler et al. 2013). During post-embryonic development, SAM gives rise to phytomers consisting of a leaf, AM, and an internode (Yang and Jiao 2016). After AM has been established in or near the leaf axil, it functions as a new SAM for the secondary growth axis, producing the overall above-ground architecture (Pautler et al. 2013).

Axillary Meristem Formation

The first step in axillary shoot branching is formation of AM (Stirnberg et al. 2002), which usually develops post-embryonically only on the adaxial side of organs where meristematic genes are preferentially expressed (McConnell and Barton 1998, Long and Barton 2000). In Arabidopsis, cells that develop into AM in the axils of leaf primordia are indistinguishable from their neighboring cells. Two tunica layers (L1 and L2) are uninterrupted between SAM and those axils. Afterward, a group of cells in the tunica layers divides anticlinally while cells in the corpus exhibit a mixture of periclinal, anticlinal, and oblique divisions. The boundary of the future AM then becomes prominent as the surrounding cells enlarge and are vacuolated. Finally, dome-shaped AM forms. Leaf primordia and floral meristems develop from AM and then become new shoot branches via axillary bud outgrowth (Grbic and Bleecker 2000).

For rice (*Oryza sativa*), formation of AM occurs in four stages. In Stage 1, a bulge of cytoplasm-dense cells forms in the axil of a leaf primordium before protruding as a cone-like AM structure with incipient prophyll in Stage 2. In Stage 3, AM becomes prominent. Finally, axillary buds and leaf primordia develop from AM in Stage 4 (Tanaka et al. 2015). The AM shares many genetic requirements in common with primary SAM formed in the embryo (Evans and Barton 1997).

Two hypotheses have been proposed for AM initiation: 1) de novo from partially or fully differentiated cells of the leaf axil separate from SAM (McConnell and Barton 1998); or 2) development from cell groups originated from SAM (Steeves and Sussex 1989). In Arabidopsis, the latter hypothesis is supported by an overlap in expression for the SAM marker gene *SHOOT MERISTEMLESS* (*STM*) and the AM identity gene *LATERAL SUPPRESSOR* (*LAS*). However, the molecular mechanism for AM formation may differ among species.

Molecular Mechanisms Controlling AM Formation

During AM formation in rice, *O. sativa homeobox1* (*OSH1*), a homolog of Arabidopsis *STM* and *Knotted1* from maize (*Zea mays*), is preferentially expressed in AM (Komatsu et al. 2003; Tabuchi et al. 2011; Tanaka et al. 2015). Significantly reduced expression of *OSH1* and less tiller production in mutants imply that this gene is essential for the initiation or maintenance of undifferentiated cell fate at a very early stage of AM formation (Oikawa and Kyozuka 2009; Tabuchi et al. 2011; Tanaka et al. 2015). In Arabidopsis, *STM* is strongly expressed in a cluster of cells that are morphologically distinct to form a bump during AM development. Because this timing of *STM* expression is similar to that of *OSH1*, the mechanism controlling AM initiation may be conserved in part between the dicot Arabidopsis and the monocot rice (Long and Barton 2000).

LAX PANICLE1 (*LAX1*), which encodes a bHLH transcription factor (TF), plays a critical role in the establishment and maintenance of AM in rice (Komatsu et al. 2001, 2003; Oikawa and Kyozuka 2009). Its expression is confined to the intersection between AM and SAM, and does not occur in developing AM. The LAX1 protein accumulates transiently at the Plastochron 4 (P4) stage of leaf development and moves to AM for full functioning. Mutations in *LAX* affect the initiation of AM and cause a reduction in tiller numbers (Oikawa and Kyozuka 2009). In fact, *LAX1* modulates aerial branching by functioning together with *LAX2* and *MONOCULM 1* (*MOC1*), as evidenced by the more severe branching phenotypes displayed in double *lax1 moc1* and *lax1* and *lax2* mutants (Tabuchi et al. 2011).

Maize *BARREN STALK1* (*BA1*), a homolog of rice *LAX1*, is crucial for the formation of AM during vegetative and inflorescence development. Expression of *BA1* is detected on the adaxial surface of early tiller buds (Gallavotti et al. 2004). Maize *ba1* mutants do not develop tillers or female inflorescence branches (ears), and male inflorescences (tassels) are unbranched (Ritter et al. 2002; Gallavotti et al. 2004). This suggests that the roles of *LAX1* and *BA1* in AM development are conserved in members of the grass family (Oikawa and Kyozuka 2009; Woods et al. 2011).

REGULATOR OF AXILLARY MERISTEM FORMATION (*ROX*), which is the *LAX1*/*BA1* ortholog in Arabidopsis, controls AM formation. Overexpression of *ROX* generates accessory buds in the axils of late rosette leaves and cauline leaves, which are not found in wild type (WT) plants. This gene is expressed in the adaxial boundary of leaf and flower primordia. Its expression relies on *REGULATOR OF AXILLARY MERISTEMS1* (*RAX1*) and *LAS* activity, with all functioning together to control AM formation. That type of formation is inhibited in *rox* mutants at the early vegetative phase under short-day (SD) conditions. Like *las rax* double mutants, *rox las* double mutants do not have any axillary buds in their rosette leaf axils, and *rox rax las* triple mutant plants form very few

axillary buds in their rosette and cauline leaves. This demonstrates, therefore, that *ROX*, *RAX1*, and *LAS* modulate AM formation redundantly in Arabidopsis (Yang et al. 2012).

The *LAX2* gene encodes a nuclear protein with a plant-specific conserved domain, and it functions together with *LAX1* in the process of AM formation in rice. Transcripts of the former are detected in the leaf axil, where AM develops, as well as in all AMs during the reproductive stage. Both genes act synergistically, but independently, to regulate AM development. The *lax2* mutant shows altered phenotypes of AM formation and a reduction in axillary branches during both vegetative and reproductive phases, similar to those of the *lax1* mutants. *LAX2* also cooperates with *MOC1* to modulate AM formation. In the *lax2 moc1* double mutant, AM does not proliferate at the P4 leaf axil and is absent at the P5 leaf axil. Consequently, the double mutant lacks tiller branches. This indicates that *LAX2* functions together with *LAX1* and *MOC1* in different pathways of maintenance and AM formation to control aerial shoot branching at both the vegetative and reproductive phases (Tabuchi et al. 2011).

MONOCULM 1, which encodes a transcriptional regulator of the GRAS family, is also a key regulator involved in AM formation in rice (Li et al. 2003). It is expressed predominantly in the leaf axil where AM develops and in the axillary buds (Li et al. 2003; Tanaka et al. 2015). In *moc1* mutants that lack axillary buds and tillers, *OSH1* expression is completely abolished in the leaf axils but unaffected in SAM (Li et al. 2003). During AM formation, *MOC1* is expressed earlier than *LAX1*, *LAX2*, and *TILLERS ABSENT1* (*TAB1*), which suggests that sequential or independent action by these genes is responsible for axillary bud formation, and that multiple pathways contribute to AM development (Li et al. 2003; Oikawa and Kyozuka 2009; Tabuchi et al. 2011).

Screening MOC1 interacting proteins has revealed MOC1 interacting protein 1 (MIP1), a member of the Bre1 family that contains a C3HC4 RING finger domain (Sun et al. 2010).

Overexpression (OE) of *MIP1* leads to phenotypes of increased tiller numbers, semi-dwarfism, and delayed heading, which are similar to those of *MOC1*-OE plants.

Arabidopsis *MOC1* analog *LAS* and its analog *Lateral suppressor* (*Ls*) in tomato (*Lycopersicon esculentum*) also play crucial roles in the formation of AM (Schumacher et al. 1999; Greb et al. 2003; Yang et al. 2012). *LAS* is expressed in the axils of leaves from which new AM develops but *las* mutants are unable to generate axillary shoots. This gene functions upstream of other regulators of AM development, such as *REVOLUTA* and *AUXIN RESISTANT 1* (Greb et al. 2003). Initiation of AM is inhibited in *ls* mutants of tomato, and cells in the axils of leaf primordia cannot maintain their meristematic abilities, resulting in the absence of side shoots during the vegetative phase. This mutant phenotype arises due to substantial alterations in hormone levels, i.e., more auxin and gibberellic acid but reduced amounts of cytokinin (Schumacher et al. 1999). The similarity in functions among homologous genes in rice, tomato, and Arabidopsis implies that the roles of MOC1 and orthologous genes are conserved in eudicots and monocots.

TILLERS ABSENT1 (*TAB1*; also termed *OsWUS*), is also necessary for initiation of AM in rice. This gene encodes a protein containing a homeodomain, WUS box, and EAR motif. *TAB1* is generally expressed only in the pre-meristem zone, and usually not in SAM or established AM (Tanaka et al. 2015) although it has been detected in SAM as well (Nardmann and Werr 2006). Highly reduced expression of *OSH1* and a lack of meristem dome formation in *tab1* mutants suggest that *TAB1* plays a vital role in the initiation of AM by prompting the expression of *OSH1* to maintain the stem cells in the pre-meristem zone. Expression of *MOC1* and *LAX1* is not changed in *tab1* mutants, implying that *TAB1* functions either in an independent pathway or downstream of *MOC1* and *LAX1* (Tanaka et al. 2015). Because the *wus* mutation does not affect axillary meristem development in Arabidopsis (Laux et al. 1996), the rice *TAB1* appears to control AM formation by a mechanism that differs from that of Arabidopsis WUS (Tanaka et al. 2015).

Another *WUS* gene, *WUSCHEL-RELATED HOMEOBOX4* (*WOX4*), is a close paralog of *OsTAB1* (Nardmann and Werr 2006) and has a role in AM development by functioning alternatively with *OsTAB1* (Tanaka et al. 2015). Unlike *OsTAB1*, however, *WOX4* is not expressed in the pre-meristem but is detected in SAM and established AM, indicating that *WOX4* helps maintain AM after it is almost entirely established (Ohmori et al. 2013; Tanaka et al. 2015). *OsTAB1* acts to form AM by enhancing the expression of *OSH1* and *WOX4*. Thus, two WOX genes function alternatively during the formation of AM in rice (Tanaka et al. 2015).

Another regulator of AM development is *RICE FLORICULA/LEAFY* (*RFL*), or *ABERRANT PANICLE ORGANIZATION 2* (*APO2*) (Deshpande et al. 2015). *RFL* is expressed in the axils of leaves and young AM (Rao et al. 2008). During AM development, *RFL* promotes AM specification by regulating *LAX1* and *CUP SHAPED COTYLEDON1* (*CUC1*). In *rfl* mutants, AM development is greatly reduced due to decreased expression of *LAX1* and CUC, which suggests that *LAX1* and *CUC*s act downstream of *RFL* (Deshpande et al. 2015). Arabidopsis *LFY* plays a vital role in floral meristem identity rather than being active in AM (Weigel and Nilsson 1995; Moyroud et al. 2010). As a variant of *LFY*, the *LFYHARA* allele promotes growth of vegetative AM through its direct target *RAX1*, which encodes an R2R3 MYB domain factor (Chahtane et al. 2013).

FRIZZLE PANICLE (*FZP*) is important in the control of floral identity genes during panicle and tiller development. Its overexpression leads to marked decreases in tiller numbers through the suppression of *RFL*/*APO2* (Bai et al. 2016). In SAM, the expression patterns are similar between *FZP* and *RFL*, implying that they interact at the transcriptional level.

TILLER ENHANCER (*TE*), a rice homolog of *Cdh1* that functions as an activator of the anaphase promoting complex/cyclosome (APC/C) complex in Arabidopsis, plays a role in AM formation. It is expressed in AM in the root meristems and the axils of young leaves at the P3 stage. However, AM initiation and formation is suppressed in *TE*-OE plants. This is accomplished by

repressing the expression of *OSH1* and degrading *MOC1* by the ubiquitin–26Sproteasome pathway. The *te* mutant plants have excess tillers because additional ones emerge from both lower and higher nodes. *TE* is involved in ubiquitination of *MOC1*, activating APC/CCdh1 E3 ubiquitin ligase by directly coupling to the CDC27 subunit of the APC/C complex (Lin et al. 2012).

Tillering and Dwarf 1 (*TAD1*) encodes a co-activator of APC/C. It is expressed ubiquitously in roots, shoot apices, internodes, vascular bundles, and panicles, but more predominantly in leaf primordia, young leaves, axillary buds, inflorescence primordia, crown root primordia, and nodes. This gene appears to function upstream of *MOC1* based on evidence that the latter accumulates at higher levels in the *tad1* mutant but at reduced levels in *TAD1*-OE plants (Xu et al. 2012). *TAD1* interacts with *MOC1* for degradation in a cell-cycle-dependent manner.

Blind, a tomato gene that encodes an R2R3 Myb TF, regulates the very early step in AM initiation (Schmitz et al. 2002; Muller et al. 2006). Mutants defective in this gene produce fewer axillary buds due to a flaw in AM initiation (Schmitz et al. 2002). *REGULATORS OF AXILLARY MERISTEMS* (*RAX*) genes in Arabidopsis, which are homologous to *Blind*, also control the initiation of AM. These genes are expressed in leaf axils where AM develops. For example, *RAX1* regulates the formation of AM in the early vegetative phase while *RAX2* and *RAX3* function redundantly in later phases (Keller et al. 2006; Muller et al. 2006). Homologs of *Blind* and *RAX* do not function in AM formation in rice, suggesting that the *Blind*/*RAX* pathway is specific to eudicots.

In Arabidopsis, the NAC domain TFs *CUC1*, *CUC2*, and *CUC3* regulate redundantly the initiation of AM in addition to their role in initiating SAM and establishing organ boundaries. The *cuc3* mutants are severely impaired in AM initiation during vegetative development and display dramatic reductions in the number of axillary buds produced in the axils of rosette leaves under SD conditions (Raman et al. 2008). *STM* expression is completely absent at the adaxial boundary of leaf primordia in *cuc3* mutant plants (Greb et al. 2003). Those plants are compromised in AM initiation

rather than in axillary bud outgrowth. *CUC1* and *CUC2*, which are targets of *miR164*, also have an overlapping function in the establishment of AM. Enhanced expression of *miR164* in the *cuc3* mutant background is linked with significant downregulation of *CUC1* and *CUC2*, consequently resulting in almost total suppression of AM formation. In contrast, more axillary buds are formed in *mir164* mutants and in plants possessing *miR164*-resistant alleles of *CUC1* or *CUC2*. Because *LAS* transcript accumulates in *mir164 a b c* triple mutants and *cuc3* mutant plants, it is suggested that *miR164* modulates axillary meristem formation via *CUC1* and *CUC2*, which further control the expression of *LAS* (Raman et al. 2008). *CUC2* transcription is constantly down-regulated in *RAX1* mutants, indicating that *RAX1* influences AM initiation by modulating the expression of *CUC2* (Keller et al. 2006). Expression of *miR 164*, *CUC1*, *CUC2*, *CUC3*, *RAX1*, and *LAS* is found in overlapping domains at the axils of developing leaf primordia (Greb et al. 2003; Vroemen et al. 2003; Keller et al. 2006; Mueller et al. 2006). In addition, their mutant phenotypes resemble each other (Vroemen et al. 2003; Mueller et al. 2006). These findings suggest that those genes function together to regulate AM formation (Raman et al. 2008). However, no genetic evidence has been revealed that confirms the role of *CUC*s in AM formation in rice.

All of these observations demonstrate that, although some regulatory pathways in the formation of AM are conserved in eudicots and monocots, other pathways are specific to only one of those plant groups.

Molecular Mechanism Controlling Axillary Shoot Growth

After AM is formed, its outgrowth as a shoot branch reiterates the primary shoot developmental pattern, conferring the plant branching architecture (Deshpande et al. 2015). Generally, AM immediately develops into axillary buds that either grow out instantly or remain dormant in order to

modulate axillary shoot branching (Bennett et al. 2006). Several factors and genes are involved in controlling such outgrowth.

Strigolactones (SLs), previously known as carotenoid-derived signals that are exuded from roots and move acropetally, have an inhibitory role in axillary shoot branching (Gomez-Roldan et al. 2008; Umehara et al. 2008). They are thought to function downstream of auxin (Bainbridge et al. 2005; Foo et al. 2005; Brewer et al. 2009; Hayward et al. 2009). Alternatively, SLs can negatively regulate auxin transport in the main stem and constrain its movement from the axillary bud into the stem (Bennett et al. 2006; Crawford et al. 2010; Domagalska and Leyser 2011).

Genes involved in SL biosynthesis or signalling pathways play a significant role in controlling the outgrowth of axillary buds and SL-mediated shoot branching. They include *DWARF10* (*D10*) in rice, *MORE AXILLARY GROWTH* (*MAX*) *4* in Arabidopsis, *RMS1* in pea (*Pisum sativum*), and *DAD1* in Petunia, which encode a carotenoid cleavage dioxygenase 8 (*CCD8*) that is involved in SL biosynthesis (Arite et al. 2007). *D10*-RNA interference (RNAi) transgenic plants have reduced capacity for auxin transport due to the decreased expression of most *OsPINs* in shoot nodes. Auxin levels are higher in shoot apices with few tiller buds than in the WT (Arite et al. 2007). Application of exogenous auxin markedly increases *D10* expression in shoot nodes while exogenous cytokinin suppresses its expression (Zhang et al. 2010). These observations indicate that *D10* has a critical role in controlling axillary bud outgrowth and tillering by reducing auxin levels and promoting cytokinin biosynthesis (Zhang et al. 2010).

DWARF3 (*D3*), an Arabidopsis *MAX2* homolog that encodes a nuclear-localized F-box protein, is expressed in roots, nodes, leaf blades, leaf sheaths, tiller buds, and panicles (Ishikawa et al. 2005). DWARF3 functions in the formation of SCF complex and interacts with DWARF14 and DWARF53 to inhibit the emergence of axillary buds and tillering in rice. The *d3* mutant is defective

in SL signalling and does not respond to GR24, a synthetic analog of SL. Consequently, those mutant plants show much earlier emergence of axillary buds and produce more tillers (Zhao et al. 2014).

HIGH-TILLERING DWARF1 (*HTD1*), which encodes *CCD7*, is an ortholog of Arabidopsis *MAX*3. It also suppresses the outgrowth of axillary buds and subsequent tillering of rice (Zou et al. 2005). Axillary buds emerge much earlier from *htd1* (or *d17*) mutants and plants have excess tillers. This accelerated capacity for tillering is caused when axillary buds are released from dormancy rather than because the mutant produces more axillary buds. Because auxin induces *HDT1* expression, it can also regulate rice tillering by up-regulating *HDT1* transcription. Introduction of *HDT1* into the *max*3 mutant of Arabidopsis rescues the branched phenotype, indicating that the functional role of *HDT1/MAX*3 in axillary bud outgrowth is conserved during evolution (Zou et al. 2006).

Rice *OsMAX1a* and *OsMAX1e* – two *AtMAX1* orthologs -- inhibit tillering through synergistic regulation of SL biosynthesis (Wang et al. 2015). Their expression is induced by P-deficiency, which promotes SL biosynthesis. These genes are also expressed in response to treatment with auxin and cytokinin. *OsMAX1a*-RNAi and *OsMAX1e*-RNAi plants have 40% more tillers than the untransformed WT. The branched phenotype of Arabidopsis *max1* is rescued by overexpression of *OsMAX1a* and *OsMAX1e*.

DWARF 27 (*D27*), which encodes a novel iron-containing chloroplast protein, is predominantly expressed in young leaves, axillary buds, inflorescence primordia, lateral roots, and crown roots. *d27* mutant plants display a defect in SL biosynthesis and exhibit extensive outgrowth of tiller buds. When treated with naphthylphthalamic acid, a synthetic auxin inhibitor, such outgrowth and tiller numbers are enhanced in the WT but suppressed in the mutants. Basipetal polar auxin transport is also much higher in those mutants than in the WT plants. Exogenous treatment with the synthetic GR24 represses the emergence of axillary bud outgrowth and tillering in the *d27*

mutant. All of these data provide evidence that *D27* regulates axillary bud outgrowth and tillering of rice through the *MAX*/*RMS* pathway and also participates in SL biosynthesis (Lin et al. 2009).

DWARF14 (*D14*), also known as *HTD2* and *D88*, is a novel putative esterase gene that controls axillary bud outgrowth and tillering in rice. Mutant plants have more tillers than the WT. Furthermore, the axillary buds emerge from *d14* plants at the three-leaf stage but not from the WT. Although *d14* possesses only one axillary bud in each leaf axil, similar to WT plants, the former has more tillers because those buds are released from dormancy rather than increasing in absolute numbers (Gao et al. 2009).

DWARF53 (*D53*), which represses SL signalling and interacts with *DWARF3* (*D3*) and *DWARF14* (*D14*), also plays a crucial role in controlling axillary bud outgrowth and tillering. A gain-of-function mutation of *D53* is insensitive to exogenous SL treatment and, compared with the WT, exhibits more tillers because of an increase in higher-order nodes as well as more produced from upper-most nodes. Exogenous application of GR24 results in upregulation of *D53* expression in the WT and more rapid degradation of the D53 protein. However, the outgrowth of axillary buds in *d53* is not responsive to GR24 treatment. Thus, *D53* functions as a negative regulator in the SL-mediated branching-inhibition pathway (Zhou et al. 2014).

OsMADS57 encodes a MADS-box TF and is expressed predominantly in SAM and axillary buds. Its enhanced expression accelerates the emergence of those buds (Guo et al. 2013). Plants over-expressing this gene have more tillers while *OsMADS57* RNAi plants have fewer when compared with the WT. *D14* is markedly down-regulated in the OE plants but up-regulated in the RNAi plants. The expression patterns overlap between *D14* and *OsMADS57* in SAM and axillary buds, and various researchers have suggested that the former is a direct target of the latter. For example, OsMADS57 interacts with *TEOSINTE BRANCHED1* (OsTB1) and this interaction eases the inhibitory effect of *OsMADS57* on *D14* transcription (Guo et al. 2013). Furthermore, *OsMADS57* RNAi plants are

insensitive to the synthetic GR24. Those findings imply that *OsMADS57* enhances the outgrowth of axillary buds and subsequent tillering via SL signalling by directly suppressing the expression of *D14* (Guo et al. 2013).

MicroRNA *miR444a* is involved in the growth of axillary buds and tillering by negatively regulating *OsMADS57*. The MADS box gene is down-regulated and tillering is reduced in *miR444a*-OE transgenic plants (Guo et al. 2013). Because *D14* expression is up-regulated in the OE plants, downregulation of *OsMADS57* expression, as mediated by *miR444a*, might control the outgrowth of axillary buds.

In Arabidopsis, four MAX genes regulate axillary bud outgrowth via the SL pathway. *MAX1*, *MAX3*, and *MAX4* play roles in SL synthesis while *MAX2* is involved in the SL signal perception. *MAX1*, an ortholog of rice *MAX1*, represses axillary shoot branching, resulting in a bushy phenotype in *max1* mutant plants. *MAX1* acts downstream of *MAX3* and *MAX4* in SL biosynthesis (Stirnberg et al. 2002; Booker et al. 2005). *MAX3* is orthologous to rice *D17*, which encodes CCD7 involved in SL synthesis. *max3* mutant plants display increased axillary branching from the rosette leaves (Booker et al. 2004). *MAX4* is orthologous to rice *D10* and is involved in the synthesis of a mobile branch-inhibiting substance in SL biosynthesis (Sorefan et al. 2003). Grafting studies have shown that MAX genes are critical for a novel graft-transmissible signal that moves acropetally to repress axillary bud outgrowth (Beveridge et al. 1996; Beveridge 2000; Booker et al. 2004, 2005).

*MAX*2, encoding an F-box leucine-rich repeat protein, is orthologous to rice *D3*. *max2* mutant plants promote shoot branching that leads to bushy phenotypes (Stirnberg et al. 2002). *MAX2* interacts with *BES1*, which induces axillary shoot branching by positively regulating the brassinosteroid signalling pathway, resulting in BES1 ubiquitination and degradation by the 26S proteasome (Yin et al. 2002; Wang et al. 2013).

Two *MAX4*/*D10* orthologs -- pea *RAMOSUS1* (*RMS1*) and Petunia *DECREASED APICAL DOMINANCE1* (*DAD1*) -- control shoot branching (Sorefan et al. 2003; Snowden et al. 2005). Mutations in *RMS1* lead to enhanced axillary branching at the basal and aerial nodes (Apisitwanich et al. 1992; Arumingtyas et al. 1992; Beveridge et al. 1997; Sorefan et al. 2003; Foo et al. 2005), while disruption of *RMS6* and *RMS7* results in increased branching only at the basal nodes (Morris et al. 2003). In *Petunia hybrida*, *DAD1*, *DAD2*, and *DAD3* regulate axillary branch development, and their mutations enhance the growth of AM into branches during vegetative development (Napoli 1996; Snowden et al. 2005; Simons et al. 2007; Hamiaux et al. 2012). Those genes influence axillary branching by modulating SL biosynthesis or its signalling pathway. These findings suggest that shoot branching is controlled by a mechanism in the SL pathway that is common to both eudicots and monocots.

Rice *OsTB1* functions as a negative regulator, repressing the subsequent outgrowth of axillary buds (Takeda et al. 2003). The gene encodes a TCP TF and is expressed throughout those buds, the basal portion of SAM, vascular tissues in the pith, and the lamina joint. Its overexpression causes a significant reduction in tiller numbers, whereas the *tb1* mutant, also known as *fc1* mutant, shows an increased number. Application of GR24, a synthetic SL analog, that suppresses the outgrowth of tiller buds in WT does not affect tiller growth in *fc1* mutants, suggesting that *OsTB1* may function downstream of SL (Minakuchi et al. 2010).

Mize *TB1* is preferentially expressed in AM and axillary shoots (Hubbard et al. 2002). Homozygous *tb1* mutant plants display many tillers, resulting in a bushy phenotype due to the presence of secondary and tertiary axillary branching (Doebley et al. 1997; Wang et al. 1999; Hubbard et al. 2002).

BRANCHED1 (*BRC1*), an ortholog of *TB1*, regulates axillary bud outgrowth in Arabidopsis. Expression of *BRC1* in developing axillary buds affects their outgrowth. *brc1* mutant plants produce

more axillary shoots from the rosette leaves when compared with the WT. Whereas *BRC1* inhibits vegetative AM initiation under long-day conditions, no role for *OsTB1* in AM initiation has been reported. During that process, *LAS* and *REV*/*IFL1* function before *BRC1*. Because expression of *BRC1* is significantly down-regulated in *max* mutants, the *MAX*-mediated pathway appears to control *BRC1* activity, which is imperative for auxin-induced apical dominance to suppress the outgrowth of axillary buds during their development (Aguílar-Martínez et al. 2007). *BRC2*, paralogous to *BRC1*, is also expressed in axillary buds and acts redundantly in the regulation of their outgrowth. However, *brc2* mutant plants show only a mild-branching phenotype when compared with *brc1* mutant plants (Aguílar-Martínez et al. 2007; Finlayson 2007; Finlayson et al. 2010).

The *APO1* gene, which encodes an F-box protein, is an ortholog of Arabidopsis *UNUSUAL FLORAL ORGANS* (*UFO*). The former regulates the outgrowth of axillary buds and tillering of rice. When compared with the WT, *apo1* mutant plants generate more tillers (Ikeda et al. 2007) while *APO1*-OE transgenic plants show inhibited outgrowth of tiller buds and have few or no tillers (Ikeda-Kawakatsu et al. 2009).

Heading date 3a (*Hd3a*), a *Flowering Locus T* (*FT*) homolog, controls axillary bud outgrowth and tillering while also functioning as a florigen in rice (Tsuji et al. 2015). *Hd3a* promotes tillering by activating lateral bud outgrowth. This is evidenced by the accelerated outgrowth of dormant buds in transgenic plants expressing *Hd3* where axillary bud formation does not differ from the WT. Hd3a protein generated in the phloem is transported to axillary buds where it interacts with 14–3–3 proteins and the TF OsFD1 to enhance bud growth. Expression of tiller marker genes (e.g., *MOC1* and *OsTB1*), SL biosynthesis genes, and auxin signalling genes is not affected in those *Hd3a*-OE transgenics. This suggests that the regulatory mechanism of *Hd3a*-mediated axillary bud outgrowth is independent from previously known networks.

High tillering, reduced height, and infertile spikelets (*THIS1*), which encodes a Class III lipase, plays a role in the release of axillary bud outgrowth and subsequent tillering in rice (Liu et al. 2013). Up until the stem elongation stage, the number of tiller buds does not differ between *this1* mutant plants and the WT, and the emergence of tillers is also similar between those genotypes. However, after the stems elongate, axillary bud outgrowth in the axils of the elongated culm is accelerated in the mutants, and they produce twice as many tillers. Results from expression analyses have suggested that *THIS1* is involved in signalling pathways for phytohormones such as SL and auxin.

DOMAINS REARRANGED METHYLASE 2 (*OsDRM2*) which encodes DNA methyltransferases responsible for de novo CG and non-CG methylation in the rice genome sequence, also regulates tillering (Moritoh et al. 2012). *Osdrm2* mutant plants display significant reductions in tiller numbers and plant growth as well as a delayed-flowering phenotype. This suggests that DNA methylation regulated by OsDRM2 plays an important role in controlling tillering.

Hormones Affecting Axillary Shoot Branching

In addition to SL, other hormones govern axillary shoot branching. One well-known example involves ‘apical dominance’, i.e., the inhibitory effect that the shoot apex has on the outgrowth of axillary buds (Thimann and Skoog 1933; Thimann 1937; Cline 1996; Napoli et al. 1999). Auxin is synthesized from the terminal bud and transported basipetally to prohibit this outgrowth. Although decapitating a plant and applying the auxin-transport inhibitor 2,3,5-triiodobenzoic acid to its intact stem can accelerate outgrowth, that response is suppressed when the buds are exposed to exogenous auxin (Snyder 1949; Panigrahi and Audus 1966).

As a polar auxin transporter, *OsPIN1* plays a critical role in auxin-dependent tillering of rice. *OsPIN1* RNAi transgenic plants produce more tillers due to the disturbance of auxin-mediated axillary bud inhibition (Xu et al. 2005). The same phenotype is found with transgenic plants that over-express *OsPIN2*. Because *OsLazy1* is markedly down-regulated in transgenic rice, *OsPIN2* appears to regulate tillering by repressing *OsLazy1* (Chen et al. 2012).

OsIAA6, which is preferentially expressed in AM of the basal stem where tillers emerge, suppresses axillary bud outgrowth and subsequent tillering of rice. When compared with the WT, tiller numbers increase by approximately two-fold in *osiaa6* plants because axillary buds are released at elongated nodes that are usually degenerated in WT plants. Therefore, *OsIAA6* appears to have an effect on axillary bud outgrowth rather than AM formation because only one axillary bud develops at each node. Likewise, because *OsPIN1* and *OsTB1* are down-regulated in *osiaa6* plants, *OsIAA6* regulates axillary bud outgrowth and tillering by modulating auxin signalling (Jung et al. 2015).

In Arabidopsis, *AUXIN-RESISTANCE1* (*AXR1*) represses further growth by the axillary buds after they form (Stirnberg et al. 1999). Mutation of that gene leads to enhanced axillary shoot branching, giving rise to a bushy phenotype. Because *axr1* is insensitive to treatment with 2,4-dichloro phenoxyacetic acid (2,4-D), the mutation appears to regulate axillary bud outgrowth by influencing auxin signalling (Estelle and Somerville 1987; Lincoln et al. 1990; Stirnberg et al. 1999).

YUCCA, which encodes a flavin monooxygenase-like enzyme, inhibits axillary bud outgrowth in Arabidopsis by enhancing the level of endogenous auxin (Zhao et al. 2001). In contrast, that outgrowth for most buds in the axils of rosette leaves is suppressed in *yucca1* mutant plants. However, overexpression of the bacterial *iaaL* gene, encoding an enzyme that conjugates free IAA to lysine, in the *yucca* mutant restores the WT phenotype, thereby demonstrating that auxin has a role in controlling shoot branching.

OsmiR393 is dramatically expressed in AM, and OE transgenic plants show greater outgrowth of axillary buds and emergence of more tillers (Xia et al. 2012; Li et al. 2016). In contrast, the *osmiR393* mutant plants produce fewer tillers. The OE plants are hyposensitive to 2,4-D and auxin transport is reduced to their axillary buds. In those plants, expression is down-regulated for the auxin transporter gene *OsAUX1* and auxin receptor genes *OsTIR1* and *OsAFB2*, which in turn inhibits the expression of *OsTB1*. *OsmiR393* is partly implicated in nitrogen-enhanced rice tillering. This gene modulates auxin signal transduction in the axillary buds when its expression is up-regulated by nitrogen treatment (Li et al. 2016).

BUSHY AND DWARF1 (*BUD1*), which encodes MAPKINASE KINASE7 (MKK7), controls axillary bud outgrowth in Arabidopsis by modulating polar auxin transport. In contrast to the WT, *bud1* plants display a more severe bushy phenotype due to the emergence of higher-order branches that result when dormant buds are released from the axils of rosette and cauline leaves (Dai et al. 2006).

Sugar, which also acts as a signalling molecule in many physiological processes in plants (Smeekens et al. 2010; Granot et al. 2013), promotes axillary bud outgrowth (Rameau et al. 2015). Its role in regulating this outgrowth is compounded because the mobilization of starch reserves in stem tissues enhances the activity of sugar-metabolizing enzymes (Maurel et al. 2004; Girault et al. 2008; Rabot et al. 2012) and also improves sugar absorption in the buds (Marquat et al. 1999; Maurel et al. 2004; Decourteix et al. 2008). During the outgrowth process, soluble sugar contents are increased in the buds (Marquat et al. 1999; Girault et al. 2008) and in xylem sap (Maurel et al. 2004; Decourteix et al. 2008). Furthermore, sugar accelerates the outgrowth of axillary buds when plants are decapitated. In pea, sucrose treatment also reduces the expression of *BRC1*, a gene that suppresses this outgrowth (Mason et al. 2014). Expression of *RhSUC2*, a gene encoding a sucrose transporter in *Rosa* sp., is generally up-regulated in outgrowing buds but is inhibited by auxin treatment, which

suggests that the effect of sugar in this outgrowth is negatively modulated by auxin (Henry et al. 2011).

Elevated levels of endogenous cytokinin in the axillary buds are closely linked with their accelerated outgrowth (Shimizu-Sato and Mori 2002). An increase in cytokinin concentrations in the xylem exudate and axillary buds after decapitation suggests that cytokinins are pivotal for the initiation of this outgrowth (Bangerth 1994; Turnbull et al. 1997).

Rice Cytokinin Oxidase2 (OsCKX2), an enzyme that degrades cytokinin, controls the outgrowth of axillary buds and tillering by suppressing the accumulation of cytokinin in the shoots. In comparison with the WT, *OsCKX2*-RNAi plants generate more tillers while *OsCKX2*-OE transgenics have fewer tillers. Because expression levels of *D53* and *TB1* – two negative regulators of axillary bud outgrowth -- are not changed in those OE plants, it is thought that *OsCKX2* contributes to AM initiation (Yeh et al. 2015).

Arabidopsis *SPS*, also called *CYP79F1*, encodes a cytochrome P450 and is strongly expressed in the leaf axils. Mutants such as *bushy* (*bus*) show phenotypes of increased AM numbers and subsequent outgrowth of axillary buds in the axils of rosette and cauline leaves, which leads to an extreme-branching phenotype. Because endogenous cytokinin levels are enhanced in the mutant, researchers have proposed that *SPS* modulates those levels in the location where axillary buds are initiated (Reintanz et al. 2001; Tantikanjana et al. 2001).

Whereas gibberellin (GA) normally inhibits shoot branching, plants that over-express GA catabolism genes and mutants that are GA-deficient exhibit greater shoot branching (Silverstone et al. 1997; Agharkar et al. 2007; Lo et al. 2008; Qi et al. 2011).

GA1 encodes ent-kaurene synthase A, which catalyzes the first step in the GA biosynthetic pathway. Arabidopsis mutants defective in this gene display excessive axillary branches that emerge from the rosette stem (Silverstone et al. 1997). Genes for Gibberellin 2-oxidases, such as *GA2ox5*,

GA2ox6, and *GA2ox9*, cause GAs to be inactivated through 2β-hydroxylation. However, rice plants that over-express those genes form more tillers and at an earlier date when compared with the WT (Lo et al. 2008). Overexpression of *AtGA2ox1* in *Paspalum notatum* also leads to an extremely low level of endogenous bioactive GA that then enhances the number of vegetative tillers produced (Agharkar et al. 2007).

Overexpression of *OsEATB* significantly reduces the endogenous level of GA in rice by down-regulating ent-kaurene synthase A. As a consequence, OE plants have more tillers and panicle branches (Qi et al. 2011).

OsNAC2, encoding a NAC TF, is important in the outgrowth of axillary buds and tillering of rice, but not in AM initiation. Transgenic plants over-expressing this gene do not form additional tiller buds or AM but do display accelerated tillering due to weak suppression of tiller bud activity (Chen et al. 2015). Buds at the first and second nodes from the top that are normally dormant in the WT develop into tillers in the OE plants. Expression of genes involved in GA biosynthesis and the signalling pathway, e.g., *OsKO2*, *OsEATB*, *OsKAO*, and *OsSLRL*, is altered in the transgenics, which suggests that *OsNAC2* regulates axillary bud outgrowth by mediating the GA pathway (Chen et al. 2015). Similar to *CUC2* and *CUC3*, *OsNAC2* protein plays a role in AM formation in Arabidopsis (Mao et al. 2007).

Brassinosteroid (BR) is also involved in controlling tiller numbers. *DWARF AND LOW-TILLERING* (*DLT*), which encodes a GRAS TF, participates in feedback inhibition of BR biosynthesis. Rice *dlt* mutant plants exhibit a defect in the brassinosteroid pathway and fewer tillers (Tong et al. 2009). *GSK3/SHAGGY-like kinase* (*GSK2*), the ortholog of Arabidopsis *GSK3/SHAGGY-like kinase BRASSINOSTEROID-INSENSITIVE2* (*BIN2*), phosphorylates and inhibits the activity of DLT. Over-expression of *GSK2* alters BR signalling and reduces tiller numbers, as observed from *dlt* mutant plants (Tong et al. 2012). *bri1-EMS-suppressor 1* (*BES1*), a positive regulator in the BR

signalling pathway, is implicated in rosette branching of Arabidopsis. A gain-of-function mutation of *BES1* generates excess rosette branches while *BES1*-RNAi plants have only one bolt (Yin et al. 2002; Wang et al. 2013).

Polyamines, i.e., aliphatic nitrogen compounds, also have a role in the outgrowth of axillary buds and shoot branching. The *bushy and dwarf* (*bud2*) mutant in Arabidopsis, which results from the disruption of a gene that encodes an S-adenosylmethionine decarboxylase (SAMDC), also displays higher levels of putrescine and but less spermidine and spermine, as well as consequent production of excess axillary branches due to altered polyamine homeostasis that is responsible for breaking the dormancy of axillary buds (Ge et al. 2006).

Finally, *BUSHY AND DWARF 2* (*BUD2*), a gene encoding SAMDC4, also regulates shoot branching in Arabidopsis by modulating the levels of endogenous auxin and cytokinin. The *bud2* mutant shows accelerated outgrowth of axillary buds (Ge et al. 2006). Those mutants are very sensitive to exogenous cytokinins but insensitive to auxin (Cui et al. 2010). In addition, expression of cytokinin-responsive genes is dramatically increased. These results suggest that polyamines control branching by altering the homeostasis of endogenous auxin and cytokinin. *BUD2* is likely to regulate axillary bud outgrowth via an IAA/cytokinin-regulated shoot branching pathway that is independent of *AXR1*. This conclusion is based on the higher-branching phenotype exhibited by the *bud2-2 axr1-3* double mutant when compared with either parental mutant plant (Cui et al. 2010).

We summarized the regulatory networks controlling AM formation and axillary bud growth in Figure 1.

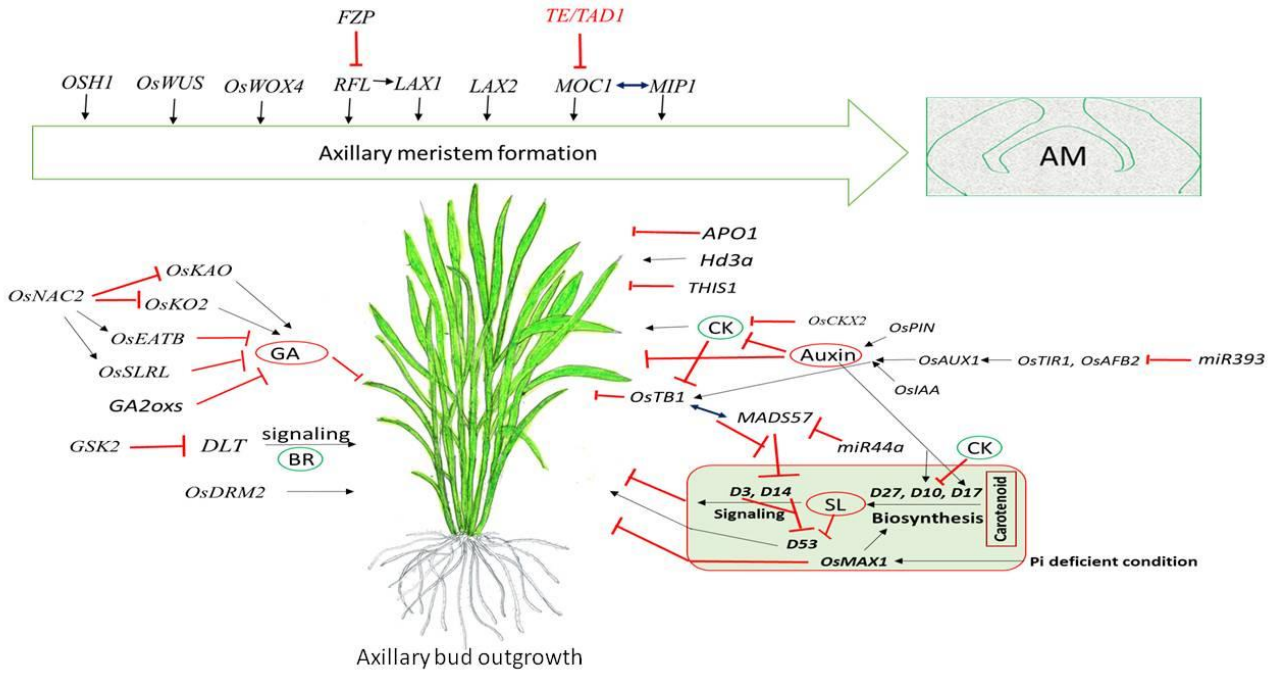

Fig. 1. Regulatory network controlling AM formation and axillary bud growth in rice.

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Author's contributions

AHW and GA collected information and wrote the manuscript.

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