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REVIEW

Unraveling the molecular mechanisms governing axillary meristem initiation in plants

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Abstract

Main conclusion **Axillary meristems (AMs) located in the leaf axils determine the number of shoots or tillers eventually formed, thus contributing significantly to the plant architecture and crop yields. The study of AM initiation is unavoidable and beneficial for crop productivity.**

Abstract Shoot branching is an undoubted determinant of plant architecture and thus greatly impacts crop yield due to the panicle-bearing traits of tillers. The emergence of the AM is essential for the incipient bud formation, and then the bud is dormant or outgrowth immediately to form a branch or tiller. While numerous reviews have focused on plant branching and tillering development networks, fewer specifically address AM initiation and its regulatory mechanisms. This review synthesizes the significant advancements in the genetic and hormonal factors governing AM initiation, with a primary focus on studies conducted in *Arabidopsis* (*Arabidopsis thaliana* L.) and rice (*Oryza sativa* L.). In particular, by elaborating on critical genes like *LATERAL SUPPRESSOR* (*LAS*), which specifically regulates AM initiation and the networks in which they are involved, we attempt to unify the cascades through which they are positioned. We concentrate on clarifying the precise mutual regulation between shoot apical meristem (SAM) and AM-related factors. Additionally, we examine challenges in elucidating AM formation mechanisms alongside opportunities provided by emerging omics approaches to identify AM-specific genes. By expanding our comprehension of the genetic and hormonal regulation of AM development, we can develop strategies to optimize crop production and address global food challenges effectively.

Keywords Axillary meristem (AM) · Shoot apical meristem (SAM) · Plant hormones · Grain yield and regulatory networks

Introduction

Plants demonstrate remarkable shoot plasticity to ensure survival and propagation, particularly in the face of challenging external and internal conditions. In seed plants, the architecture of shoots is predominantly influenced by factors such as the number, position, orientation, and size of shoot branches. The precise regulation of shoot branching represents a crucial adaptive strategy orchestrated by a complex regulatory network.

The development of the primary shoot axis originates from the activity of the shoot apical meristem (SAM), a group of mitotic cells formed during embryogenesis. Subsequently, derivatives of this meristem generate all aboveground portions of plants (Bowman and Eshed 2000). The SAM continuously produces aerial organs by adding growth units called phytomere, which typically consist of an internode, a leaf, and an axillary meristem (AM) which is initiated in the leaf axil (Wang et al. 2018). The AM, acting as a new SAM of the secondary growth axis, differentiates into, such as in rice, tiller bud, leaf sheath primordium, and leaf primordium (Yan et al. 2023; Wang 2021). Therefore, the scalable branching across multiple levels is enabled by the specification and activity of AMs, leading to the generation of diverse architectural forms. Notably, AM activity has long

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been a target of breeding selection due to its significant contribution to crop yield through effects on tiller/branch number, panicle number, and panicle branches (Springer 2010; Wang and Li 2008, 2011; Shao et al. 2019).

Numerous studies over several decades have sought to elucidate AM initiation mechanisms. The prevailing model proposes that main endogenous and developmental cues interact to regulate this process. For instance, the *LATERAL SUPPRESSOR* (*LAS*), *REVOLUTA* (*REV*), and *CUP-SHAPED-COTYLEDON* (*CUC*) genes (Aida et al. 1999; Otsuga et al. 2001; Greb et al. 2003; Hibara et al. 2006) can function together in a regulatory cascade controlling AM initiation. An Auxin minimum niche is required to sustain *SHOOT MERISTEMLESS* (*STM*) expression in the boundary zone, thereby maintaining AM identity (Guo et al. 2015). Subsequently, *WUSCHEL* (*WUS*) follows the expression of *STM* and then activates the expression of *CLAVATA3* (*CLV3*) (Shi et al. 2016; Cao and Jiao 2020; Cao et al. 2020), forming a *WUS-CLV3* loop in the center of leaf axils to maintain stem cell activity (Xin et al. 2017), indicating the completion of AM initiation and de novo formation of new stem cell niches in the leaf axils (Yang et al. 2023). Following the auxin minimum, a cytokinin pulse occurs in the leaf axil during AM formation (Wang et al. 2014b). In addition to auxin and cytokinin, many other phytohormones (e.g., strigolactones, brassinosteroids, gibberellins, and abscisic acid) participate and interplay in shoot AM formation (Napoli and Ruehle 1996; Beveridge et al. 1997; Beveridge 2000, 2006; Turnbull et al. 2002; Sorefan 2003; Foo et al. 2001; Morris et al. 2001; Zhang et al. 2020b; Chatfield et al. 2000; Reddy et al. 2013). These factors intricately interact and regulate AM initiation through shared or distinct mechanisms.

In this article, we focus on explaining, unifying, and differentiating the intertwined mechanisms of AM development in several plant species, including *Arabidopsis* (*Arabidopsis thaliana*), tomato (*Solanum lycopersicum*), and rice (*Oryza sativa*). Additionally, we also discuss the challenges of identifying more genes specially involved in AM formation and propose available methodologies suitable for resolving these problems. But outgrowth is not the focus of this present review, and we refer readers to our newly published review on this topic (Yuan et al. 2023). All the genes mentioned in this review are listed in Table 1.

Origin of AM: detached or de novo?

A distinctive characteristic of plants is their remarkable ability for reiterative growth and continuous organogenesis over their lifetimes. Analogous to the SAM, AMs play a pivotal role in initiating the development of lateral organs. This precisely regulated developmental process can result in AM formation, subsequently giving rise to the development of branches/tillers. In crops such as wheat and rice, the branches or tillers originating from AMs ultimately contribute to the formation of panicles, determining the overall grain yield (Fig. 1 and Video S1). However, grain yield loss will occur if plants have defective AM formation (Fig. 1 and Video S1).

Two alternative models for AM formation have been proposed: the 'detached meristem' model and the 'de novo induction' model (Fig. 2 and Video S2). The 'detached meristem' model posits that AMs form from pluripotent stem cells that bud off from the primary SAM and maintain meristematic potential in the leaf axils as leaf primordia develop (Steeves and Sussex 1989). This is supported by evidence showing that leaf axil cells remain undifferentiated and express the meristem marker *STM* (Grbic and Bleecker 2000; Long and Barton 2000). Laser ablation experiments have also shown that AMs originate from cells with *STM* mRNA persistence (Shi et al. 2016). In addition, studies reveal that AM progenitor cells are set aside early in SAM development (Burian et al. 2016).

Alternatively, the 'de novo induction' model proposes that a set of differentiated cells equivalent to their neighbors can form an AM given an appropriate localized signal (Long and Barton 2000). Evidence for this includes AMs arising on the underside of leaves in *Arabidopsis phabulosa-1d* (*phb-1d*) mutants. (McConnell and Barton 1998). Additionally, ectopic AM occurs in *stm* mutants that lost SAM characteristics (Endrizzi et al. 1996). Adventitious SAMs can arise from the axils of cotyledons and cultured root explants in *pinhead* mutants (McConnell and Barton 1995). Ectopic expression of the AM regulator, *Super determinant 1A* (*SDE1*), which is confined in leaf axils and regulates AM development, leads to ectopic meristem formation at the distal leaflets even in the shoot away from leaf axils (López et al. 2021). Together, these observations lend credence to the 'de novo induction' hypothesis, whereby differentiated cells can acquire meristematic identity.

The ongoing debate between the "detached meristem" and "de novo induction" theories in AM initiation is complex. Moreover, the expression of *STM* in the interprimordial regions between SAM and leaf axils (Greb et al. 2003; Shuai et al. 2002) complicates this debate, as it challenges clear differentiation between these two concepts. We propose investigating a range of mutants, specifically those with distinct AM initiation but no SAM defects as a strategy, and the second one that knocks out the AM-specific expressed genes by high-through CRISPR-Cas9 system, to bridge the understanding between these two mechanisms. This approach is promising because the genes associated with these mutants might exclusively influence AM or interact with SAM-related genes to trigger AM initiation.

Table 1. Genes regulating AM formation. This table summarizes key genes identified and characterized in plants that play a role in AM formation

Gene names	Accession numbers	Species	Functional annotation	References
MOC1	*Os06g0610350*	Rice	A GRAS protein	(Li et al. 2003)
LAS	*AT1G55580*	Arabidopsis	A GRAS protein	(Greb et al. 2003)
STM	*AT1G62360*	Arabidopsis	A class I knotted-like homeodomain protein	(Long et al. 1996)
REV	*AT5G60690*	Arabidopsis	A small homeodomain-leucine zipper family	(Otsuga et al. 2001)
SPS	*AT1G16410*	Arabidopsis	A member of CYP79F proteins	(Tantikanjana et al. 2001)
CLV3	*AT2G27250*	Arabidopsis	CLAVATA3/ESR-related	(Otsuga et al. 2001)
ATH1	*AT3G47730*	Arabidopsis	A BEL1-like homeodomain (BLH) type three-amino-acid loop extension (TALE) class homeodomain protein	(Cao et al. 2020)
WUS	*AT2G17950*	Arabidopsis	A homeodomain transcription factor	(Wang et al. 2017)
MOC3	*Os04g0663600*	Rice	A homeodomain transcription factor	(Lu et al. 2015)
PHV	*AT1G30490*	Arabidopsis	Belonging to HD-Zip family	(Shi et al. 2016)
RAX1	*AT4G23100*	Arabidopsis	Belonging to the class R2R3 MYB genes	(Wang et al. 2017)
RAX2	*AT2G36890*	Arabidopsis	Belonging to the class R2R3 MYB genes	(Guo et al. 2015)
RAX3	*AT3G49690*	Arabidopsis	Belonging to the class R2R3 MYB genes	(Guo et al. 2015)
ARR1	*AT3G16857*	Arabidopsis	An Arabidopsis response regulator (ARR) protein	(Zheng and Chen 2011)
axr1	*AT1G05180*	Arabidopsis	Encoding a subunit of the RUB1 activating enzyme	(Stirnberg et al. 1999)
PINHEAD	*AT5G43810*	Arabidopsis	A member of the EIF2C class of proteins	(Zhang et al. 2020a)
CUC1	*AT3G15170*	Arabidopsis	A transcription factor	(Hibara et al. 2006)
CUC2	*AT5G53950*	Arabidopsis	A transcription factor	(Hibara et al. 2006)
CUC3	*AT1G76420*	Arabidopsis	A transcription factor	(Hibara et al. 2006)
DA1	*AT1G19270*	Arabidopsis	A ubiquitin-activated peptidase	(Li et al. 2020)
UBP15	*AT1G17110*	Arabidopsis	A ubiquitin-specific protease	(Li et al. 2020)
DPA4	*AT5G06250*	Arabidopsis	Transcription repressor of CUC2/CUC3	(Li et al. 2020)
SOD7	*AT3G11580*	Arabidopsis	Encoding nuclear localized B3 DNA binding domain	(Li et al. 2020)
drn	*AT1G12980*	Arabidopsis	Encoding an AP2/ERF protein	(Tian et al. 2014)
drnl	*AT1G24590*	Arabidopsis	Encoding an AP2/ERF protein	(Tian et al. 2014)
miR164A	*AT2G47585*	Arabidopsis	A microRNA that targets several genes containing NAC domains	(Reinhart et al. 2002)
miR164C	*AT5G27807*	Arabidopsis	A microRNA that targets several genes containing NAC domains	(Wang et al. 2004)
miR164B	*AT5G01747*	Arabidopsis	A microRNA that targets several genes containing NAC domains	(Bonnet et al. 2004)
LFY	*AT5G61850*	Arabidopsis	A transcriptional regulator	(Chahtane et al. 2013)
LOF1	*AT1G26780*	Arabidopsis	A MYB-domain transcription factor	(Lee et al. 2009)
EXB1	*AT1G29860*	Arabidopsis	A member of WRKY Transcription Factor	(Guo et al. 2015)
ROX	*AT5G01305*	Arabidopsis	Encoding a bHLH protein	(Yang et al. 2012)
LAX1	*Os01g0831000*	Rice	Encoding a bHLH protein	(Komatsu et al. 2003)
BA1	*Zm00001d042988*	Maize	Encoding a bHLH protein	(Gallavotti et al. 2004)
PIN1	*AT1G73590*	Arabidopsis	Encoding Encodes an auxin efflux carrier	(Wang et al. 2014a)
TAA1	*AT1G70560*	Arabidopsis	The auxin biosynthesis gene	(Guo et al. 2015)
PIN5	*AT5G16530*	Arabidopsis	A functional auxin transporter	(Guo et al. 2015)
PID	*AT2G34650*	Arabidopsis	A protein serine/threonine kinase	(Michniewicz et al. 2007)
mp	*AT1G19850*	Arabidopsis	a transcription factor (IAA24)	(Guo et al. 2020)
BZR1	*AT1G75080*	Arabidopsis	a positive regulator of the (BR) signaling pathway	(Van De Velde et al. 2017)
SPL9	*AT2G42200*	Arabidopsis	A putative transcriptional regulator	(Zhang et al. 2020b)
GA2ox4	*AT1G47990*	Arabidopsis	A gibberellin 2-oxidase	(Zhang et al. 2020b)
LOB	*AT5G63090*	Arabidopsis	Involved in lateral organ development	(Gendron et al. 2012)
BAS1	*AT2G26710*	Arabidopsis	A member of the cytochrome p450 family	(Bell et al. 2012)
DSP	*Os02g0594300*	Rice	APETALA2/ethylene-responsive element binding protein	(Yu et al. 2023)
AHK2	*AT5G35750*	Arabidopsis	A histidine kinase	(Wang et al. 2014b)

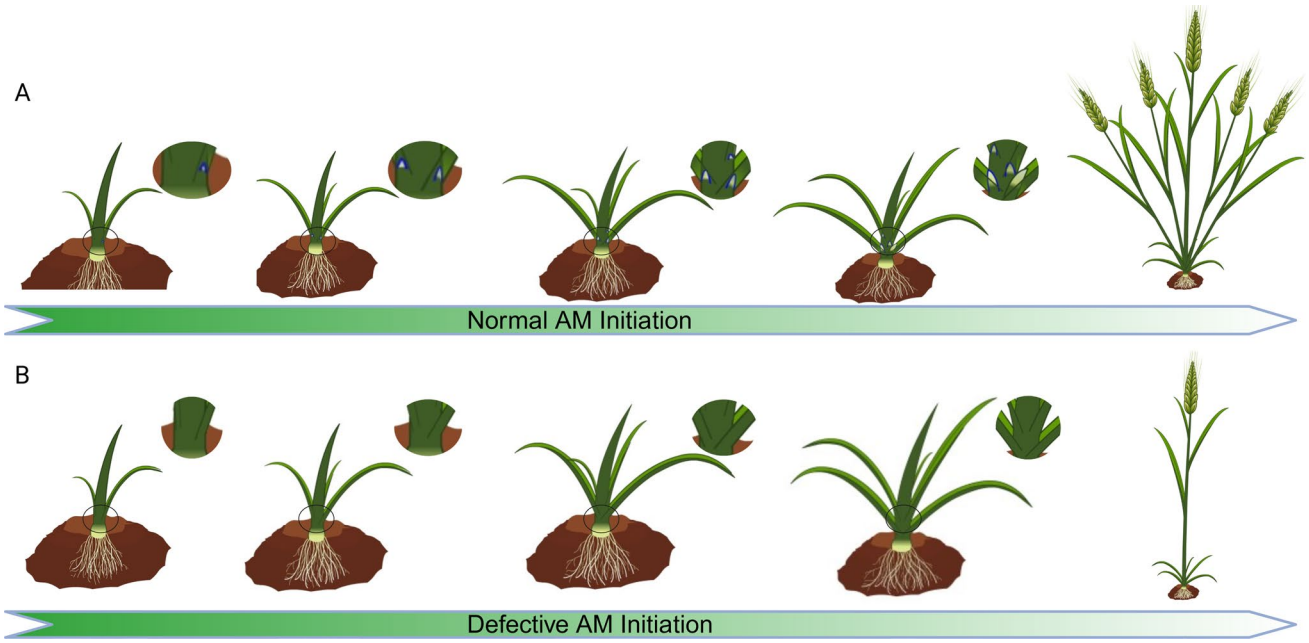

Fig. 1. IIlustrution of the dynamic developmental process of axillary meristem (AM) initiationof crops and efects comparisons between plants with normal ability to launch AM initiation or not **A** in the up line depicts the plant with the normality to generate AMs, resulting in more tillers/branches (the top right) if AMs can outgrowth subsequently. AMs arises from the leaf axil framed by black circles. The AM boxed in the shoot base are closed up in the top right. **B** in the bottom line delineates the plant with severly defect in AM initiation, which can lead to monoculm phenotype with low grain yield (the bottom right). The leaf axil where generate AM are barren. For more detail information, please refer to Video S1

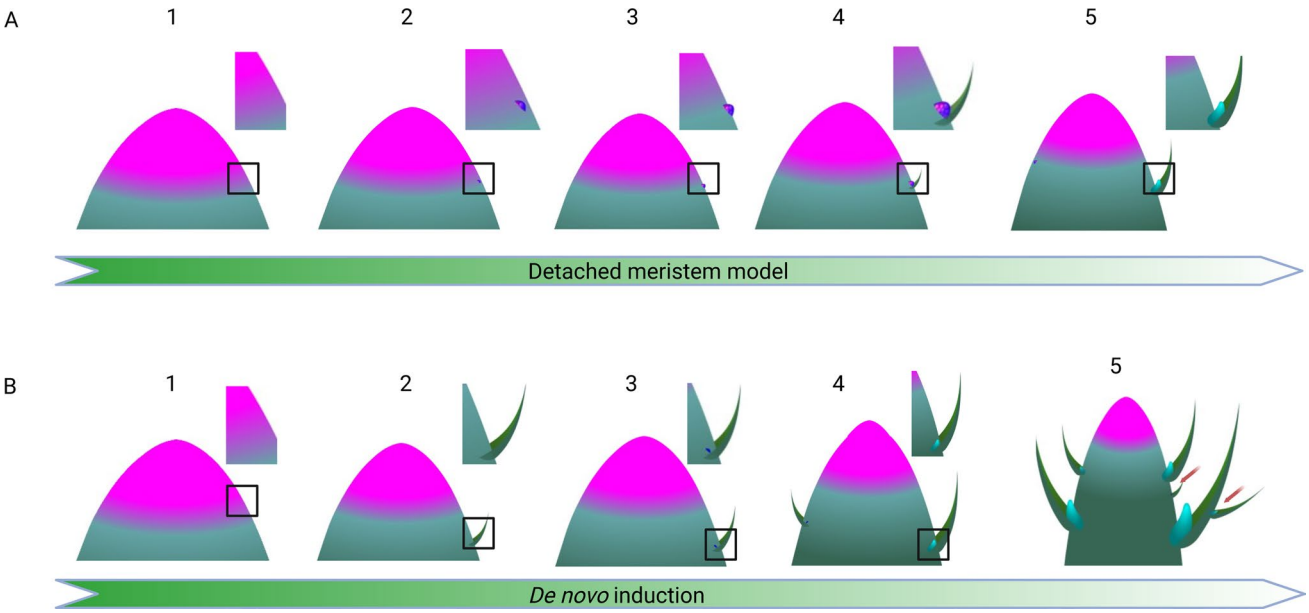

Fig. 2. Dynamic illustration of distinct origins of AM. **A** The detached meristem model: one axillary meristem emanates from the shoot apical meristem (SAM). This process is represented successively from a group of cells frst derived from the SAM,which then grow up to one tiller bud. **B** The de novo induction model: In some cases, the tiller forms directly from determinate cells, without derivation from the SAM. When essential genes are disrupted, the tiller can even form far from the leaf axil (indicated by arrow). The enlarged pictures (top right) represent the region framed by boxes. For more detail information of these processes, please refer to Video S2

The genetic and epigenetic factors regulate AM initiation

Understanding the molecular mechanisms that govern shoot branching heavily relies on characterizing genes responsible for AM initiation. To this end, multiple genes, including the key genes like rice *MONOCULM1* (*MOC1*) and its orthologues *Lateral suppressor* (*LS*) and *LAS* in tomato and *Arabidopsis*, *STM*, and *REVOLUTA* (*REV*) in *Arabidopsis* (Long et al. 1996; Schumacher et al. 1999; Otsuga et al. 2001; Greb et al. 2003; Li et al. 2003), have been identified and thoroughly studied.

Generally, current mutants related to AM development exhibit morphological defects that can be categorized into two classes. The first type comprises mutations affecting AM initiation, leading to a lack of AM formation, as the *las* mutant exemplifies. The second type enhances AM formation, resulting in a bush phenotype, as observed in *supershoot* (*sps*) mutants (Tantikanjana et al. 2001). In the following discussion, we summarize genes associated with AM initiation, accompanied by a critical analysis of their hierarchical relationships, where applicable (Fig. 3).

Given that AMs function as new SAMs, generating vital plant structures such as tillers/branches, leaves, flowers, etc., it is getting essential to explore whether the genes instrumental in SAM development also play a role in the initiation of AMs. It is hypothesized that these sorts of genes are involved in the fundamental processes of meristem initiation. This involvement is direct unless the formation of AMs is an indirect result of these mutations. To understand this relationship, we examine key genes like *STM*, *CUCs*, *REV*, *PINHEAD*, *CLV3*, and more, aiming to elucidate their functions and the potential hierarchical interactions among them. For example, the sustained expression of *STM*, a number of the *KNOTTED* class of homeodomain genes essential for SAM formation, suggests the presence of cells in

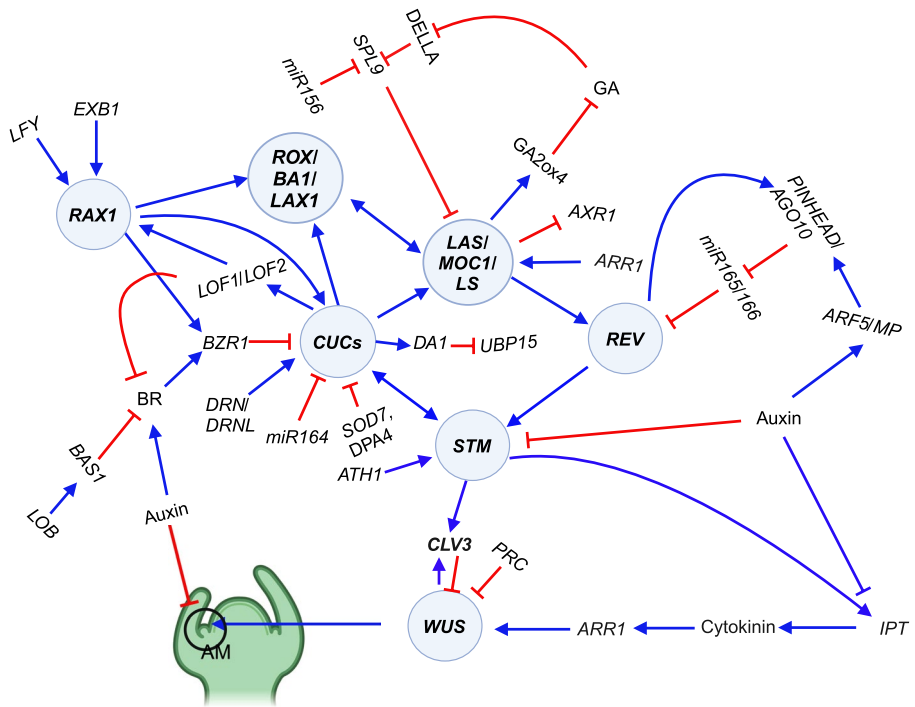

Fig. 3. Summary diagram of components and phytohormones in AM initiation. Blue arrows indicate promotion and red-fat ended lines depict inhibition. The axillary meristem (AM) is circled. The genes acting with several genes are framed framed with circles with blue background. In this model, many factors function at diferent stages, including key hub genes (e.g., CUCs and LAS genes) and phytohormones (e.g., auxin, BR, and CK, etc.). *LFY*, LEAFY; *EXB1*, EXCESSIVE BRANCHES1; *LOF1/LOF2*, LATERAL ORGAN FUSION1/2; *RAX1*, REGULATOR OF AXILLARY MERISTEM1; *BR*, Brassinosteroid; *BZR1*, BRASSINAZOLE-RESISTANT1; *BAS1*, PHYB ACTIVATION TAGGED SUPPRESSOR1; *LOB*, LATERAL ORGAN BOUNDARIES; *DRN*, DORNROSCHEN; *CUCs*, CUP-SHAPED-COTYLRDON genes; *BRC1*, BRANCHED1; *STM*, SHOOT MERISTEMLESS; *LAS*, LATERAL SUPPRESSOR; *SPL9*, SQUAMOSA PROMOTER BINDIN PROTEIN-LIKE9; *GA*, Gibberellin; *ATH*: ARABIDOPSIS THALIANA HOMEOBOX GENE1; *GA2ox4*, gibberellin 2- oxidase4; *DELLA*, aspartic acid–glutamic acid–leucine–leucine–alanine; *AXR1*, Arabidopsis auxin-resistant 1; *ROX*, REGULATOR OF AXILLARY MERISTEM FORMATION; *REV*, REVOLUTA; *PAGO10*, ARGONAUTE10; *BR*, Brassinosteroid; *MP*, monopteros; *ARF5*, monopteros; *IPT*, ADENYLATE ISOPENTENYLTRANSFERASE; *ARR1*, Arabidopsis response regulator1; *WUS*, WUSCHEL; *CLV3*: CLAVATA3; *PRC*, Polycomb repressive complex; *DPA4*, NGATHA-LIKE transcription factors DEVELOPMENT-RELATED PcG TARGET IN THE APEX4; *SOD7*, SUPPRESSOR OF DA1-1 7; *UBP15*, UBIQUITIN-SPECIFIC PROTEASE15; *B-ARR*, type-B Arabidopsis response regulator

an indeterminate state (Long et al. 1996). Overexpression of *STM* can lead to many ectopic SAMs in tobacco plants, indicating a cell fate switch from determinacy to indeterminacy in cell fate (Sinha et al. 1993). Notably, despite the role of *STM* in SAM, ablation of most cells within the *STM*-expressing region prevents AM initiation (Shi et al. 2016). These findings indicate that AM and SAM share a comparable molecular regulatory mechanism, with *STM* also playing a crucial role in AM initiation. However, it is noteworthy that ectopic *STM* expression is inadequate to activate AM formation from leaf axil cells that have lost *STM* expression (Shi et al. 2016), suggesting some cells undergo irreversible fate change or require special triggers to reverse them to indeterminate states. As *STM* has been proposed as an early marker of AM initiation (Long and Barton 2000), a small group of stem cells in the boundaries between the SAM and the emerging leaf primordium will develop into AM expressing *STM,* suggesting an involvement of *STM* in AM initiation (Keller et al. 2006). Furthermore, Wang et al. proposed a two-stage model for cell division during AM initiation, associating each stage with distinct *STM* expression levels (Wang and Jiao 2018b). In this model, maintaining low *STM* expression is required but insufficient for AM initiation. A subsequent increase in *STM* induces AM initiation and bulging (Shi et al. 2016). The early low levels of *STM* expression are presumably needed for stem cell competence, although these cells lack *CLV3* or *WUS* expression, which are also essential for AM formation (Shi et al. 2016). It has been shown that the *ARABIDOPSIS THALIANA HOMEOBOX GENE1* (*ATH1*), encoding a BEL1-like homeodomain (BLH) type three-amino-acid loop extension (TALE) class homeodomain protein, maintains *STM* expression, thus preserving the meristemic cell fate (Gómez-Mena and Sablowski 2008).

The *WUS* gene, a homeodomain transcription factor expressed in the SAM of the organing center, defines the stem cell niche (Wang et al. 2017). Despite its role in embryonic SAM formation, *WUS* in *Arabidopsis* and its rice ortholog *MONOCULM 3* (*MOC3*) are required to initiate AM (Lu et al. 2015; Tanaka et al. 2015; Wang et al. 2017; Xin et al. 2017). Moreover, *WUS* expression is repressed by a polypeptide signal encoded by *CLV3*, acting as a stem cell marker (Schoof et al. 2000). Interestingly, *WUS* and *CLV3* have a feedback relationship during AM initiation (Fig. 3). *CLV3* is undetectable in leaf axils in *wus* mutants, suggesting that *WUS* can activate *CLV3* (Xin et al. 2017). However, *WUS* expression is highly elevated in the *clv3-2* leaf axils, where the AM primordium is larger than that in the wide-type, suggesting *CLV3* signaling already restricted *WUS* expression to enable proper AM size determination in early developmental stages (Xin et al. 2017). *WUS* expression precedes AM initiation after *STM* expression in the initial phases (Guo et al. 2020) (Fig. 3). Moreover, Wang et al. 2021 considered that AM initiation concurs with the expression of *WUS* and *CLV3* between leaf primordium 11 (P11) and P13 in *Arabidopsis* (Wang 2021). This finding is underscored by a similar observation in rice, where AM initiation can be detected at the P3 stage, as evidenced by the expression of *MOC3*, a rice ortholog of *WUS* (Lu et al. 2015; Shao et al. 2019).

The *REV* gene, encoding an HD-ZIPIII transcription factor, is indispensable for forming all lateral meristems in addition to its role in SAM development (Otsuga et al. 2001). Indeed, the loss-of-function of *REV* mutants leads to the absence of AMs (Tian et al. 2014; Talbert et al. 1995). REV can bind to the *STM* promoter region, indicating the *STM* requirement of REV during AM formation (Tian et al. 2014). The ectopic expression of the *REV* homolog *PHAVULOTA* (*PHV*) maintains and further activates ectopic *STM* expression on the abaxial leaf side, leading to ectopic AM formation (Shi et al. 2016). Histological analysis revealed that *REV* expression precedes *WUS,* which indicates that *REV* activity is epistatic to *WUS* (Otsuga et al. 2001). Regarding *STM* expression, no difference exists between *wus* mutants and wild-type, consistent with *STM* acting epistatically to *WUS* (Wang et al. 2017).

The redundant *CUP-SHAPED COTYLEDON* (*CUC*) genes (*CUC1*, *CUC2*, and *CUC3*) in *Arabidopsis* encode NAC transcription factors that significantly contribute to embryonic shoot meristem formation and shoot organ boundary specification. *CUC2* and *CUC3*, but not *CUC1,* influence AM formation. Further analysis indicates that *CUC3* plays a more significant role in regulating shoot branching than *CUC2*, but the effect is most prominent when *CUC2* and *CUC3* are combined (Hibara et al. 2006). CUC2 and CUC3 can directly activate the expression of *DA1*, encoding a ubiquitin-dependent peptidase, while mutations of the DA1 substrate in UBIQUITIN-SPECIFIC PROTEASE15 (UBP15) lead to repression of AM initiation (Li et al. 2020). Two transcription factors, the NGATHA-LIKE transcription factors *DEVELOPMENT-RELATED PcG TARGET IN THE APEX4* (*DPA4*) and *SUPPRESSOR OF DA1-1 7* (*SOD7*) redundantly repress *CUC* expression in the leaf axil and *dpa4-2 sod7-2* double mutants display delayed AM initiation (Nicolas et al. 2022). *DRN* and its homolog *DRNL*, which encode AP2-type transcription factor family proteins, are required for AM initiation by directly activating *CUC2*. Large portions of the leaf axils in single or double *dornroschen* (*drn*) and *drnlike* (*drnl*) mutants are barren (Tian et al. 2014).

Recessive mutations in the *PINHEAD* locus of *Arabidopsis* disrupt the primary shoot meristem and AM formation, resulting in a single leaf or a slender pin-like organ and reduced lateral buds both in axils of cauline and rosette leaves (McConnell and Barton 1995; Ratcliffe et al. 1999; Zhang et al. 2020a). *PINHEAD* expression coincides in the

leaf axil where AMs form and its overexpression occasionally produces more than one AMs per leaf axil (Zhang et al. 2020a), indicating its role in controlling AM formation. *STM* and *REV* are both down-regulated and up-regulated in the *pinhead* and its overexpression mutants, respectively (Fig. 3), suggestive of an epistatic role of *PINHEAD* to *REV* and *STM* (Zhang et al. 2020a).

Plant microRNAs are endogenous, single-stranded, and nontranslated RNA molecules that are highly complementary to their target mRNAs, mediating post-transcriptional gene silencing through mRNA cleavage (Bartel and Bartel 2003). The *miR164* genes, comprising *miR164A*, *miR164B,* and *miR164C* (Wang et al. 2004; Bonnet et al. 2004; Reinhart et al. 2002), post-transcriptionally regulate *CUC* genes (Schwab et al. 2005; Raman et al. 2008). Constitutive overexpression of *miR164* phenocopies the branching habits of *cuc1 cuc2* double mutants by downregulating *CUC1* and *CUC2* transcripts (Nemhauser et al. 2004; Mallory et al. 2004). Conversely, the loss of function of *miR164* genes can produce more accessory buds (Raman et al. 2008). In contrast, overexpression of *miR164* in the *cuc3-2* mutant abolishes AMs, indicating that *miR164*, *CUC1*, *CUC2*, and *CUC3* play a pivotal role in AM initiation (Raman et al. 2008). Besides *miR164*, the rescue of AM defects in *Arabidopsis argonaute 10* (*ago10*, also known as *pinhead*) by sequestering *miR165/166* (Zhu et al. 2011), which targets *REV*, suggests *AGO10/PINHEAD* acts upstream of the *REV* and *STM* through the sequestration of *miR165/166*.

Collectively, these genes, such as *STM*, *CUCs* and *REV*, are integral in orchestrating the development of both the SAM and AMs. Yet, the precise timing and spatial dynamics governing the initiation of AM development remain topics for further exploration. We hypothesize that these genes depend on additional genes, specifically expressed in the leaf axils, to function as triggers for the initiation of AMs. This activation might occur either preceding or following their expression. Notably, a particular category of genes, known to manifest AM defects by specially regulating AM initiation, includes *Arabidopsis LAS* and *REGULATOR OF AXILLARY MERISTEM* (*RAX*) (Keller et al. 2006), as well as rice *MOC1* (Li et al. 2003) and *LAX PANICLE1* (*LAX1*) (Komatsu et al. 2003). Disruption of *MOC1* in rice or its orthologous genes (e.g., *LS* in tomato and *LAS* in *Arabidopsis*), transcription factors of the GRAS family, results in the shortage of AMs and, consequently, fewer branches or tillers. *MOC1* and *LAS* are expressed explicitly in the AM initiation zone (Greb et al. 2003; Li et al. 2003; Schumacher et al. 1999). These studies suggest a conserved function of these genes in both monocot and dicot. It is worth noticing that, in *Arabidopsis*, *STM* is focused on a group of small and densely cytoplasmic cells near the adaxial center of the primordium border, where it is required for AM initiation. However, these cells fail to develop into a new AM without *STM* expression in *las* mutants, suggesting that focused *STM* expressing denoting meristem organization onset relies on *LAS* function (Greb et al. 2003). This coincides with our hypothesis that SAM-regulating genes necessitate precise mediation to initiate AM development at an appropriate location. Furthermore, *LAS* has been proven to be a hub gene that integrates inputs from many upstream genes, as indicated by a leaf axil-enriched gene regulatory network analysis (Tian et al. 2014).

Despite the similarities in expression patterns between *LAS* and *MOC1*, notable differences exist. *LAS* expression regions extend to several layers of SAM beyond the AM, compared with *MOC1*, which remains undetectable in SAM (Greb et al. 2003; Li et al. 2003). Furthermore, the inflorescence meristems generated by AM were not affected in *las* mutants but otherwise in *moc1* mutants (Chun et al. 2022; Greb et al. 2003; Zhang et al. 2021b). These variations underscore the evolutionary divergence between monocotyledonous and dicotyledonous plants, highlighting distinct regulatory mechanisms in plant architecture development. Likewise, *REGULATOR OF AXILARRY MERISTEM FORMATION* (*ROX*) is an *Arabidopsis* gene encoding bHLH protein, orthologous to the branching regulators *LAX1* in rice and *BARREN STALK1* (*BA1*) in maize (Yang et al. 2012; Komatsu et al. 2003; Matthes et al. 2019). Loss-of-function of *ROX* caused compromised AM formation. Its expression extended to the SAM and the AM, unlike *LAX1*'s AM location (Oikawa and Kyozuka 2009). In contrast to *LAX1* and *BA1*, flower development was uninfluenced in *rox* mutants (Yang et al. 2012), further supporting the hypothesis of evolutionary distinctions between monocotyledons and dicotyledons. However, this inference should be pitched out with caution as the differences between *las* and *rox* mutants of *Arabidopsis* and their corresponding wild types became more pronounced when studied under short-day conditions (Yang et al. 2012; Greb et al. 2003). Because *Arabidopsis,* native to Europe and central Asia, has spread in the temperate climate zones of the five continents and, therefore, originally adapted to long-day conditions (Hsu et al. 2019).

In addition to the reliance of *STM* on *LAS*, other SAM-related genes are also affected in *las*. For example, *WUS* is undetectable in mutants *las,* indicating that *WUS* acts downstream of *LAS* (Wang et al. 2017). Enriched *REV* expression in leaf axils relies on *LAS*, a member of the GRAS family controlling AM initiation (Greb et al. 2003). Substantial upregulation of *LAS* accumulation was observed in *mir164* triple mutants, indicating that *miR164* can negatively regulate *LAS* (Raman et al. 2008). Thus, AM initiation is inhibited by *miR164* through restricting *CUC1/2* accumulation, which in turn regulates *LAS* expression (Raman et al. 2008). This is further substantiated by the observed downregulation of *LAS* in the double mutant *cuc1 cuc2,* placing *LAS* downstream of *CUC1* and *CUC2* (Hibara et al. 2006)

(Fig. 3). The regulation of the AM-specific gene *LAS* by *CUC* genes suggests that specific signals from the SAM are necessary to trigger AM initiation. This mechanism underscores the intricate interplay between SAM and AM development.

In addition to *LAS*, other genes specially expressed in or near AM also mediate SAM-related genes. For example, *REGULATOR OF AXILLARY MERISTEMS1* (*RAX1*), an MYB family gene, promotes AM initiation by specifying the location of the stem cell niche. *RAX1* functions redundantly with *RAX2* and *RAX3* to regulate AM initiation (Keller et al. 2006). *RAX1* is initially detectable in a subregion along the boundary between the meristem and leaf primordia, similar to *LAS* (Keller et al. 2006). *LEAFY* (*LFY*), a master regulator of the transition of the reproductive stage, directly activates *RAX1* to promote AM initiation (Chahtane et al. 2013). *RAX1* also directly enhances *CUC2* expression in vivo and in vitro (Tian et al. 2014). *LATERAL ORGAN FUSION1* (*LOF1*) is also an MYB domain gene, which is expressed in the boundary domain of the SAM and leaf primordia. Loss-of-function if *lof1* mutants lack AMs and *STM* expression in the corresponding boundary domain (Lee et al. 2009). Further gene expression analysis in *lof1* mutants positioned *LOF1* upstream of the *RAX1*, *STM*, *LAS*, and *CUC* genes (Shuai et al. 2002). However, Gendron et al. (2012) suggested that *CUC* genes may positively regulate *LOF1* and *LOF* genes (Gendron et al. 2012), a way similar to that of *LAS* and *CUCs*. The *EXCESSIVE BRANCHES1* (*EXB1*) gene, encoding the WRKY transcription factor WRKY71, affects AM initiation. *EXB1* disruption results in reduced branching, while overexpression of *EXB1* in *exb1-D* gain-of-function mutants leads to severe bushy and dwarf phenotypes (Guo et al. 2015). *EXB1* is shown to control AM initiation by positively regulating the transcription of *RAX1*, *RAX2*, and *RAX3* (Guo et al. 2015). Interestingly, overexpression of rice *WRKY72* in *Arabidopsis* also increases shoot branches (Song et al. 2010), implying evolutionary conservation of *EXB1/WRKY71* function in AM formation between monocots and dicots (Guo et al. 2015).

In addition to the precise regulation of SAM-related genes by genes expressed in AMs, epigenetic modulation allows nuanced expression of the same gene in distinct cell types and developmental contexts. Such epigenetic control allows for the diverse expression patterns of genes, which may be broadly expressed yet exhibit distinct functions in various cellular environments and stages of plant development. Thus, epigenetic control is intrinsically involved in all developmental processes, including AM initiation. Since many genes mediating *WUS* and *STM*, such as *REV* and *ARABIDOPSIS RESPONSE REGULATOR 1* (*ARR1*) which is a type-B ARR transcription factor, are not exclusively expressed in leaf axils. Therefore, precisely expressing the genes needed for particular stages is imperative for AM control. The Polycomb Repressive Complex 2 (PRC2) establishes the H3K27me3 mark in plants and animals, providing a docking site for *PRC1* to impose repressive chromatin (Zheng and Chen 2011). In mature leaves, where cells are fully differentiated, both *WUS* and *STM* exhibit high levels of H3K27me3, indicative of a low abundance of *WUS* and *STM* mRNA. In contrast, these two genes have a low concentration of H3K27me3 and a high concentration of H3K4me2/3, a mark associated with active chromatin, in tissues containing the leaf axil *STM*-expressing cells (Shi et al. 2016; Cao and Jiao 2020). Accordingly, *STM* and *WUS* are elevated in *prc* mutant (Shi et al. 2016; Wang et al. 2017). Moreover, applying histone deacetylation inhibitor trichostatin A induces ectopic *WUS* expression (Xin et al. 2017). Wang et al. showed that epigenetics contributes to the dynamic of *WUS*, with expression terminated in leaf axils and then reactivated de novo (Xin et al. 2017). A large abundant H3K27me3 represses the *WUS* expression in the leaf axil, while histone H3/H4 acetylation (H3/4Ac) is depleted. Before *WUS* activation, the levels of the H3K27me3 repressive mark decrease, while the levels of the active H3/4Ac mark increase (Wang et al. 2017; Cao and Jiao 2020).

In conclusion, the intricate web of genetic and epigenetic factors orchestrates the initiation of axillary meristems in plants. The collaborative action of genes such as *CUC*, *WUS*, *STM*, *REV*, *LAS*, and others, along with the regulatory influence of miRNAs, creates a finely tuned molecular symphony.

How do phytohormones precisely control AM initiation?

Phytohormones regulate diverse developmental processes throughout the plant life cycle. For example, auxin orchestrates developmental responses such as gravitropism and apical dominance, which depend on forming auxin gradients in plant tissues (Casanova-Sáez et al. 2021; Leyser 2018). Cytokinins (CKs) influence agricultural processes, including growth, nutrient responses, and biotic/abiotic stress responses (Kieber and Schaller 2018). Gibberellins (GAs) promote growth by regulating seed germination, root/shoot elongation, flowering, and fruit patterning (Binenbaum et al. 2018). Brassinosteroids (BRs) also stimulate plant growth and development by controlling cell division, elongation, and differentiation (Planas-Riverola et al. 2019). Given the multiple roles of phytohormones in plant development, regulatory mechanisms must exist to precisely control axillary meristem (AM) initiation. Based on current understanding, we synthesize the hormonal control of AM initiation as follows:

Lines of evidence that auxin is involved in AM initiation have been reported. For example, the AGC III kinase PINOID (PID) modulates polar auxin transport by regulating PIN1 localization within the cell (Michniewicz et al. 2007).

Severe homozygous *pid* mutants resemble *pin1* mutants and fail to form AMs compared to wide-type plants (Wang et al. 2014a). This highlights the importance of directional auxin transport for AM initiation. In the dominant mutant *exb-1D*, which displays excessive branching, the auxin biosynthesis gene *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1* (*TAA1*) and the auxin transport genes (e.g., *PIN5*), are repressed by *EXB1* induction (Guo et al. 2015), suggesting the importance of auxin homeostasis to control shoot branching. Additionally, as in *auxin resistance 1* (*axr*1) mutants, reduced auxin sensitivity enhances AM formation (Stirnberg et al. 1999), implying the significance of auxin in mediating AM initiation. The dynamic requirement of auxin was further investigated. Namely, the auxin minimum is one prerequisite for AM initiation, exemplified by that: PIN1 mediates auxin flow in the adaxial domain away from the leaf axil toward the tip of the leaf primordium, thus establishing the auxin minimum in the leaf axil (Wang et al. 2014b; Bayer et al. 2009). The shoot meristem marker *STM* is activated during AM formation (Long and Barton 2000; Greb et al. 2003). Conversely, ectopically expressing an auxin biosynthesis gene, indicating higher auxin levels in the leaf axil, decreases *STM* expression (Wang et al. 2014b). This auxin minimum niche sustaining *STM* expression in the boundary zone to maintain the AM identity was evidenced by Guo et al. 2015 (Guo et al. 2015). In contrast, an elevated auxin concentration in the stem cell maintenance stage, driven by a specially located leaf axil gene promoter, perturbs the AM initiation (Wang et al. 2014b). Consistent with this, *pin-formed 1* (*pin1*)-like phenotypes are observed in mutants lacking *PID* function. Because the *PID* disruption presumably maintains unidirectional auxin apical-basal transport, consequently increasing auxin to inhibit AM formation (Friml et al. 2004). These lines of evidence show that the auxin minimum is the precondition for AM initiation. However, maize auxin biosynthesis mutants display defects in vegetative AM formation (Matthes et al. 2019), implying that auxin is still required at some points during AM initiation. The requirement of auxin during AM initiation was demonstrated by the *monopteros* (*mp*) mutant. *MP*, also known as *ARF5*, is an auxin response factor that activates downstream signaling in response to auxin. *MP* expresses in the youngest leaf primordia and ceases its expression at the later AM formation stage when AM starts bulging (Guan et al. 2017; Guo et al. 2020), indicating the existence of a high concentrate auxin in the later AM bulging stage and a positive role for auxin in AM initiation (Zhang et al. 2020a). Furthermore, *MP* may activate the expression of *PINHEAD*, whose protein sequesters *miR165/166* to release *REV* and *STM*, thereby promoting AM initiation (Zhang et al. 2020a). Moreover, rescued AM defects in *mpΔ* mutants by deleting the ARF5-binding site in the *AGO10*/*PINHEAD* promoter indicated auxin signaling is required in late AM initiation stages (Zhang et al. 2020a). In summary, auxin is essential for AM initiation, playing dynamic roles during this process. In the early stages of AM formation, an auxin minimum is required for meristematic cell maintenance, while in the later stages, higher auxin levels promote their activation. Further dissection of auxin synthesis, transport and signaling dynamics will provide deeper insights into its complex regulation of AM development.

Genetic analysis has shown that CK perception and signaling are essential for AM initiation. Several instances have favored the requirement of CK. For instance, AM initiation deficiency of *rax1* mutants can be partially rescued by either CK production in leaf axil or exogenous CK treatment (Wang et al. 2014b). Additionally, CK receptors and the signaling detector in the leaf axils are upregulated prior to and during AM initiation, indicating cytokinin's role in AM initiation (Wang et al. 2014b). Mutants defective in CK receptors such as *Arabidopsis histidine kinase 2* (*ahk2*), *ahk3*, and *ahk4* with compromised CK perception and the corresponding double mutants exhibit defects in AM initiation. Moreover, B-type *ARABIDOPSIS RESPONSE REGULATOR* (*ARR*) transcription factors, which act downstream of the CK signaling pathway, are also required for AM initiation (Wang et al. 2014b). Furthermore, the SAM-related genes and CKs were mutually regulated to guarantee AM initiation. For example, during AM initiation, *STM* activates CK biosynthesis in leaf axils (Guo et al. 2015). CK signaling then activates de novo *WUS* expression in leaf axils (Wang et al. 2017; Guo et al. 2020). Type-BRRs, transcriptional activators in CK signaling, especially ARR1, directly bind to the *WUS* promoter to activate its expression (Cao and Jiao 2020). ARR1 also directly binds to the *LAS* promoter to activate its expression (Tian et al. 2014). In addition to the low auxin condition, a subsequent pulse of CK occurs prior to AM initiation (Wang et al. 2014b). No CK signal can be detectable without an auxin minimum in the leaf axil (Wang et al. 2014b), demonstrating the dependence of the leaf axil CK pulse on the auxin minimum. Supporting this, s*ps* mutants, which show a bushy phenotype due to enhanced AM formation and lateral bud release, have elevated levels of CK but decreased levels of auxin (Tantikanjana et al. 2001). Furthermore, in mutants such as *las*, *rax, and rev* with compromised AM initiation, the leaf axils lack CK signaling pulse (Wang et al. 2017), indicating that CK is required for AM initiation. Overall, the evidence demonstrates that CK signaling is a key step following the establishment of the auxin minimum niche. CK perception and downstream transcriptional activation of *WUS*, *LAS* and other AM regulators promote the activation and bulging of meristematic cells to initialize AM development.

Gibberellic acids (GAs) are growth-promoting hormones that mediate various plant developmental processes throughout the plant life cycle (Yamaguchi 2008).

However, exogenous GA application decreases AM formation. Leaf axils ectopically expressing a GA biosynthesis gene showed significantly lower AM formation (Zhang et al. 2020b). Conversely, the GA-deficient mutant *ga1-3* displays more AMs, indicating a negative role of GA in AM formation. However, how GA precisely regulates AM initiation, especially regulating AM-specific or SAM-related genes, deserves investigation due to the importance of AMs. DELLA proteins, master repressors of GA signaling, participate in various physiological processes by interacting with various transcription factors, including BRASSINAZOLE-RESISTANT1 (BZR1), ARR1, and ARF6 (Van De Velde et al. 2017). A *della* pentuple mutant shows defects in AM formation, suggesting that DELLAs play a role in this process. DELLAs interact with SPL9, thus attenuating its repression of *LAS*. This promotes AM initiation, with *LAS* then inducing GA deactivation enzyme Gibberellin 2-beta-dioxygenase 4 (GA2ox4) to form a low-GA condition in leaf axils (Zhang et al. 2020b). Thus, the crosstalk and balance between GA metabolism and *LAS* precisely modulate AM formation spatiotemporally.

In addition to auxin and Cks, BR-responsive genes are highly enriched in organ boundary cells, suggesting these sites are novel centers of BR activity (Tian et al. 2014). BR is an essential plant steroid hormone regulating cell division and expansion (Gendron et al. 2012). As low cell division rates are required in the boundary zone to maintain AM competence, BR accumulation is negatively regulated in leaf axils by *LATERAL ORGAN BOUNDARIES* (*LOB*), a key boundary-specific transcription factor. *LOB* directly upregulates *PHYB ACTIVATION TAGGED SUPPRESSOR1* (*BAS1*), a cytochrome P450 enzyme that inactivates BRs through C-26 hydroxylation, thereby reducing BR levels to decrease cell division and expansion in the boundary zone (Gendron et al. 2012; Bell et al. 2012). Furthermore, the SAM-developmental gene *CUC3* was inhibited by the BR-activated gene *BRASSINAZOLE-RESISTANT1* (*BZR1*) by directly binding to the promoter of *CUC3*, indicating low BR levels in boundary zones are required to activate AM initiation. In summary, *LOB* restricts BR accumulation in leaf axil boundary zones through *BAS1* induction. Low BR levels inhibit cell division and expansion while also alleviating *BZR1* repression of *CUC3*. Fine-tuned crosstalk between BR and key AM regulators like *LOB* and *CUC3* allows proper AM initiation.

While plant hormones, such as auxin, CKs, BRs, and GAs, participate in various development in the entire plant life, precisely where and when they act is the key point for development. Likewise, AM initiation is associated with phytohormones, which must be involved at the right time and location.

Genes regulating AM formation affect grain yield

For crop species, AMs are essential for producing tillers bearing grains, determining the number of seed spikes per plant and the number of seeds per spike—all key factors influencing overall crop yield (Wang and Jiao 2018a), such as in rice, maize, and wheat. These factors are directly determined by branching ability during vegetable and reproductive growth stages. Namely, AM essentially harbors a niche with a group of meristematic cells to influence branching in tilers and panicles. For instance, the *LAX1* and *MOC1* genes in rice are involved in the formation of both tillers and panicle branches. Mutations in either *MOC1* or *LAX1* resulted in a reduced number of both tillers and panicle branches (Wang and Li 2011). Further exploring indicated that *LAX1* is regulated indirectly by the gene *DEFECTIVE STIGMA AND PANICLE* (*DSP*), determining tiller primordium formation and synergistically regulating panicle primordium development (Yu et al. 2023). Likewise, in *Helianthus annuus*, a dicotyledonous species, the mutated *REGULATOR OF AXILLARY MERISTEM FORMATION-LIKE* (*Ha-ROXL*), akin to *LAX1*, affects both AM initiation and Floral meristems (FMs) (Basile et al. 2019), which suggested a shared role of *LAX1* and its orthologs in influencing grain yield across dicots and monocots. However, disruption of *ROX* in *Arabidopsis*, an ortholog of *LAX1*, displays compromised AM formation during the vegetative phase, particularly noticeable under short-day photoperiods (Yang et al. 2012). Again, we must be cautious about drawing definitive conclusions regarding this abnormality observed in *Arabidopsis*, since *Arabidopsis* originated in and is generally grown under long-day conditions (Hsu et al. 2019). Furthermore, in *las* and *rax* mutants, the AM initiation defects are more easily recognized under short-day conditions than that under long-day conditions (Greb et al. 2003; Keller et al. 2006), concurring with our perspective in making critical sense of AM formation in *Arabidopsis*. Collectively, further research on AM initiation genes is needed to elucidate the genetic mechanisms underlying tiller and panicle branching. Exploring conserved and specialized regulators of AMs will provide insights to improve branching and optimize crop yields.

Challenges and opportunities regarding isolation genes involved in AM formation

While essential for plant development and agriculture, the molecular mechanisms underlying AM formation stay elusive. The study of AM initiation has been hurdled, mainly due to the shortage of mutants, specially affecting AM development in plants like rice and *Arabidopsis*. This implies that many unknown AM initiation regulators demand to be identified. Notably, alternative methodologies developed recently could be employed to resolve this problem. For example,

Yang et al. suggested the utilization of genetic backgrounds with reduced apical dominance to identify more AM initiation regulators (Yang et al. 2023). Genome editing technologies drive significant advances in life sciences due to precise modifications at target genomic loci (Xing et al. 2023; Doudna and Charpentier 2014). CRISPR/Cas9 systems have been broadly adopted as a targeted genetic manipulation tool that has been applied to many species, such as rice, wheat, tomato, and more (Doudna and Charpentier 2014). This routine technology can also be used to identify and validate new genes that act specifically in AM interactions. The utility of this technology is not limited to model plants, but can further be extended to cultivated crop plants and their wild progenitors, which often have very different architectures. Utilizing this technology allows for discovering AM-specific genes across a diverse range of plant species. Emerging yet thriving omics may also help distinguish new genes involved in AM initiation regulation. For example, single-cell omics technologies reveal the intracellular dynamics of different individual cells and answer biological questions with high-dimensional catalogs of millions of cells, including transcriptomics, genomics, chromatin accessibility, epigenomics, and proteomics data across species (Mo and Jiao 2022). Initially applied in animals, single-cell RNA sequencing (sc-RNA) technologies have been embraced by the field of plants. In *Arabidopsis*, Zhang et al. carried out Sc-RNA to define the cellular taxonomy of the *Arabidopsis* vegetative shoot apex at the transcriptome level and found that the shoot apex is composed of highly heterogeneous cells (Zhang et al. 2021a); in maize, sc-RNA analyzed single cells from developing maize ears, helping to identify candidate genes associate crops yield traits (Xu et al. 2021); in rice, analysis of root tips using Sc-RNA provided insight into the transcriptomic landscape of major cell types of rice root tip at single-cell resolution (Wang et al. 2021). In addition, a gene regulatory network-based investigation of trichoblast differentiation in *Arabidopsis* revealed novel transcription factors and previously unknown feedback loops/mechanisms by harnessing trajectory inference, one algorithm used in Sc-RNA analysis (Denyer et al. 2019). Despite Sc-RNA, single-cell level chromatin has also been practiced in plants, which is essential in AM initiation. For example, scATAC-seq has been applied to profile root tip cells in *Arabidopsis*, along with sc-RNA data, suggesting a connection between chromatin accessibility and expression dynamics (Farmer et al. 2021). Together, since the efficiency of the omics-based approaches, these techniques are more commonly used to investigate cell identity and fate changes, which also occur in AM initiation. As cell identity and fate changes occur during AM initiation, applying single-cell omics techniques could reveal new genes and networks controlling this process. These emerging approaches may expedite research on AM initiation mechanisms. Combined with clever genetics, single-cell omics technologies provide promising avenues to elucidate the molecular control of AM development.

Concluding remarks

This review has covered significant recent advances in elucidating the intricate molecular mechanisms governing AM initiation. Research over decades has revealed that AM development relies on coordinated regulation by transcriptional, hormonal, and epigenetic factors. Key regulators such as *LAS*, *RAX1*, *STM*, *REV, WUS*, and *CUC2* converge to control gene expression programs activating meristematic fate in leaf axil cells. Intricate crosstalk between auxin, CKs, GAs, and other hormones establishes a niche conducive to AM formation. Moreover, dynamic changes in chromatin modifications facilitate spatiotemporal patterns of AM gene expression. Despite progress, questions remain regarding the developmental origin of AM progenitor cells, limitations for identifying more AM-specific, and integration of the various pathways regulating AM initiation. Key next steps include: (1) Elucidating the developmental relationship between the shoot apical meristem and AM progenitor cells and reconciling detached versus de novo origins during AM initiation; (2) Identifying additional novel regulators and networks of AM formation, combing omics- sequencing and cellular resolution imaging techniques; (3) Exploring divergence and specialization of AM developmental programs between plant species; (4) Leveraging knowledge of AM formation mechanisms to improve crop architecture and yield. Collectively, unraveling the AM initiation process remains an exciting frontier in plant development biology. Translation of these fundamental findings to crop species holds immense promise for agricultural enhancement. We anticipate the next decades would witness transformative discoveries illuminating how plants elaborate their axillary meristems to elaborately branch out their forms.

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