

Review

Molecular Mechanisms Regulating Lamina Joint Development in Rice

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Abstract: Leaf angle (LA) is a major agronomic trait of rice plant architecture, which is determined by the development of the leaf lamina joint (LJ) and closely related to plant yield. The LJ is formed by the leaf collar belt (ligule band), leaf tongue (ligule) and leaf ear (auricle). Parenchyma cells (PCs), sclerenchyma cells (SCs), vascular bundles (VBs), and aerenchyma tissue (Ac) are present in the cross sections of LJ. The genetic and molecular regulation of rice leaf angle and LJ development has been well investigated in recent years. However, the underlying complex regulatory network still requires further elucidation and comprehensive discussion. In this review, we briefly describe the cellular characteristics of four typical stages of LJ development, and systematically summarize the genes regulating rice leaf inclination based on their roles in PC proliferation and elongation, as well as SC proliferation and differentiation. We also introduce the emerging regulatory pathways of phytohormones and transcription factors (TFs), environmental cues that are involved in rice LJ development, discussing the relevant intricate signal network that is relevant to provide further insights into the genetic improvement of leaf angle traits in rice breeding.

Keywords: rice; leaf angle; lamina joint; phytohormone; transcription factor; environmental cues



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1. Introduction

Rice is the main staple crop for about 4 billion people in the world, and rice yield must be further increased due to an increasing population. Leaf angle (LA) refers to the inclination between the stem and leaf blade, serving as an important agronomic trait of rice architecture that indicates the degree of leaf erectness. Moderately upright leaves contribute to the reasonable dense planting of rice, which can improve the leaf index per unit area, significantly improve the photosynthetic efficiency, and increase the yield per unit area [1–4].

LA size is closely related to leaf lamina joint (LJ) development in rice. A comprehensive understanding of the developmental and regulatory mechanism of rice leaf LJ contributes to further improve the plant architecture and improve the rice yield. The leaf LJ development process in rice plants is finely regulated by a multitude of factors, including plant hormones, environmental factors, and biological and abiotic factors, all of which subsequently impact the overall architecture of the plant. At present, numerous studies have extensively investigated the plant hormone pathway and transcription factor pathways, such as brassinosteroid (BR), auxin (AUX), and gibberellin (GA) [5,6]. However, the regulatory network of leaf LJ development is extremely complex and still harbors a lot of unexplored components.

In this review, we summarize the progress of different pathways and the genetic network among key genes, with the objective of facilitating the development of novel approaches for enhancing both yield and quality in rice breeding.

2. Structure and Development Process of Rice LJ

The LJ is a white ring that is located at the leaf blade base in rice, serving as a connection between the blade and sheath, and consisting of the leaf collar belt (ligule band), leaf

tongue (ligule), and leaf ear (auricle) (Figure 1a). It cannot photosynthesize because of no mesophyll cells in the LJ tissue, so photosynthesis does not take place. In the cross sections of the LJ, parenchyma cells (PCs) dominate in terms of both number and area, while the vascular bundles (VBs) are arranged regularly on the abaxial side (Ab) and the adaxial side (Ad), serving as the channels of water and nutrient transport. The aerenchyma tissue (Ac) occupies the central area, acting as the gas exchange channel. The sclerenchyma cells (SCs) are positioned at the edge of the vascular bundle to provide structural reinforcement for the LJ [6]. The LJ structure varies significantly among rice varieties with different LA (Figure 1b).

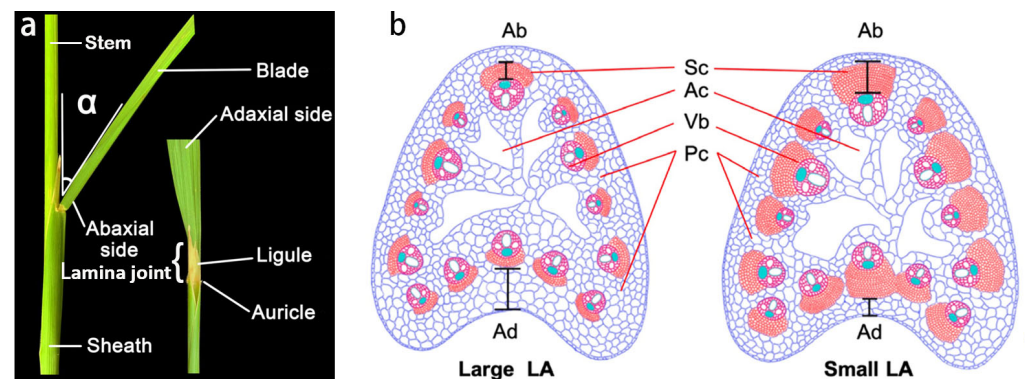


Figure 1. Leaf angle in rice and schematic models of the cross sections for the lamina joints in rice [2,4]. (a) Morphology of the lamina joints of rice leaf with leaf angle (LA). Rice leaf angle is marked with α , and leaf lamina joint with brackets. (b) Schematic models of the cross sections for the lamina joints of rice leaf with large (left) and small (right) LA. The schematic models are drawn based on the cross sections of the rice lamina joints stained with Fasga [7], in which the sclerenchyma cells are shown in red, while parenchyma cells are shown in blue, and the vascular bundles are shown in purple xylem and green phloem. Ad, adaxial side; Ab, abaxial side; Sc, sclerenchyma cell; Ac, aerenchyma; Vb, vascular bundle; Pc, parenchyma cell.

The development of rice LJ originates from the initiation region of the shoot apical meristem (SAM) according to existing studies, and can be roughly divided into four sequential stages. The development of rice LJ originates from the initiation region of the SAM, and can be broadly categorized into four sequential stages [4–6]:

Stage I: LJ begins to develop after leaf tip cells start to grow at the top of the leaf. A line of cells at the base of the leaf undergoes rapidly divided longitudinally and lateral to form a pre-ligule band (PLB) structure that is perpendicular to the base of the leaf tip and the top of the leaf sheath. Meanwhile, the development of both ligule and auricle is initiated (Figure 2a). It was found that the PCs proliferated and differentiated prior to the SCs during this period [4–6].

Stage II: The epidermal cells on the adaxial side of the PLB base undergo peripheral division, resulting in the formation of lingual and auricular cells. New leaves grow, but the LJ is still wrapped in the old leaves (Figure 2b). As the PCs continue to proliferate and expand, and the vasculature develops, the volume and number of PLB cells increase significantly [4–6].

Stage III: With the appearance of the leaf ligule and auricle, the LJ of the new leaves extends out of the old leaf sheath, and the development of the new leaf LJ gradually matures (Figure 2c), and the cell wall in SCs becomes progressively thicker [4–6].

Stage IV: The adaxial side of the LJ extends and bends towards the abaxial side due to the expansion and longitudinal elongation of PCs, resulting in a gradual increase in leaf angle until reaching its maximum [4–6] (Figure 2d).

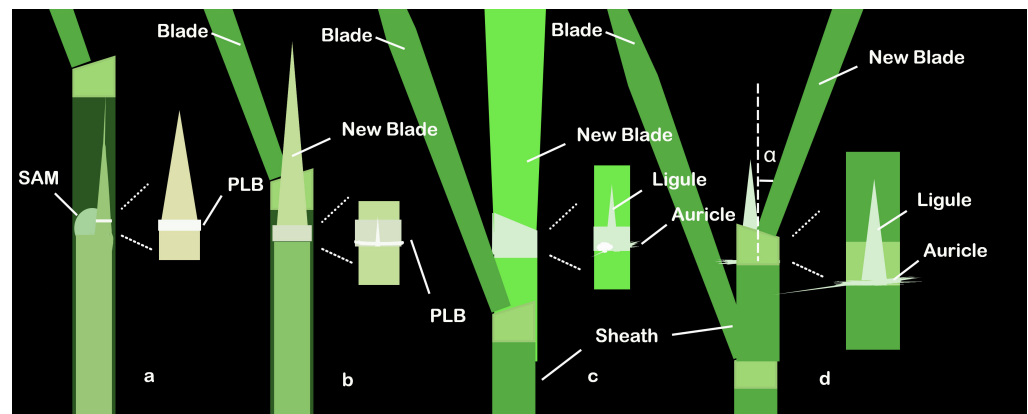


Figure 2. Schematic models of the lamina joint development in rice. (a) The lamina joint development starts in the peripheral area of the shoot apical meristem; (b) the pre-ligule band expands, and the flat division produces the primary ligule and primary auricle cells; (c) the ligule and auricle appear, and the new lamina joint extends from the old leaf sheath; (d) lamina joint matures to form leaf angle. On the left side of (a–d) is the lamina joint front; on the right side is the adaxial front; and the light green on the lower part of (a,b) indicates the wrapping in the old leaf sheath. PLB, pre-ligule band; SAM, shoot apical meristem; leaf angle labeled with α .

Based on the morphological and cytological characteristics during the development of the LJ, it is generally believed that the final LA may be determined by the balance between the thrust generated by the expansion of the adaxial PCs and the mechanical support generated by the SCs and VBs of LJ [4,5].

3. Regulation of PC Development in LJ

The shape of the LJ is determined by the quantity and dimensions of PCs on the adaxial side [3,4]. Over the past 30 years, various plant hormones and TFs in the development and regulation of PCs have been identified [5,6].

3.1. Regulation by Brassinosteroids (BRs)

BRs are widely distributed steroid phytohormones that play important crucial roles in regulating plant growth, development, and stress response. The role of BR in rice encompasses the regulation of crucial agronomic traits, including LA, plant height, and grain shape. Moreover, BR positively regulates LA by controlling LJ development [2].

Up to now, many genes related to BR synthesis have been shown to influence PC development on the adaxial side of the LJ [8], and these genes play different roles in BR synthesis pathway (Figure 3). Among them, *OsCYP51G3* and *SLG* are both involved in the synthesis of cycloartenol. *OsCYP51G3* encodes CYP51 obtusifolios 14 α -demethylase, which belongs to the cytochrome P450 superfamily and is involved in the demethylation of creosol to produce cycloartenol. *SLG* encodes a potential BAHD acyltransferase-like protein, which is also implicated in the generation of cycloartenol. *OsCYP51G3* and *SLG* both positively regulate the synthesis of endogenous BR, promote the expansion of PCs on the adaxial side of the LJ, and positively regulate the LA [9,10].

Some BR synthesis genes are responsible for the production of cycloartenol to campesterol (CR), such as *BRD2/DWF1* catalyzes 24-methylenecholesterol (24-MC) to CR [11–13]. Moreover, several cytochrome P450 protein family genes, including *OsDWARF11 (D11)* [14], *OsDWARF4* [1], *CYP90D2/D2* [15], and *BRD1 (BR-deficient dwarf 1)/OsDWARF* [16], are involved in the catalysis from CR to brassinolide (BL) [17,18]. Those genes are all part of the BR synthesis pathway and contribute to increasing LA by accelerating the proliferation and elongation of PCs on the adaxial side of the LJ. The mutants of those genes exhibit an upright leaf phenotype due to the inhibition of endogenous BR synthesis. However, some certain members of the family have opposite LA phenotypes in mutants. For example, the

function-deficient mutant *bla2* showed increased LA [19], which may be due to altered endogenous active BR content [20].

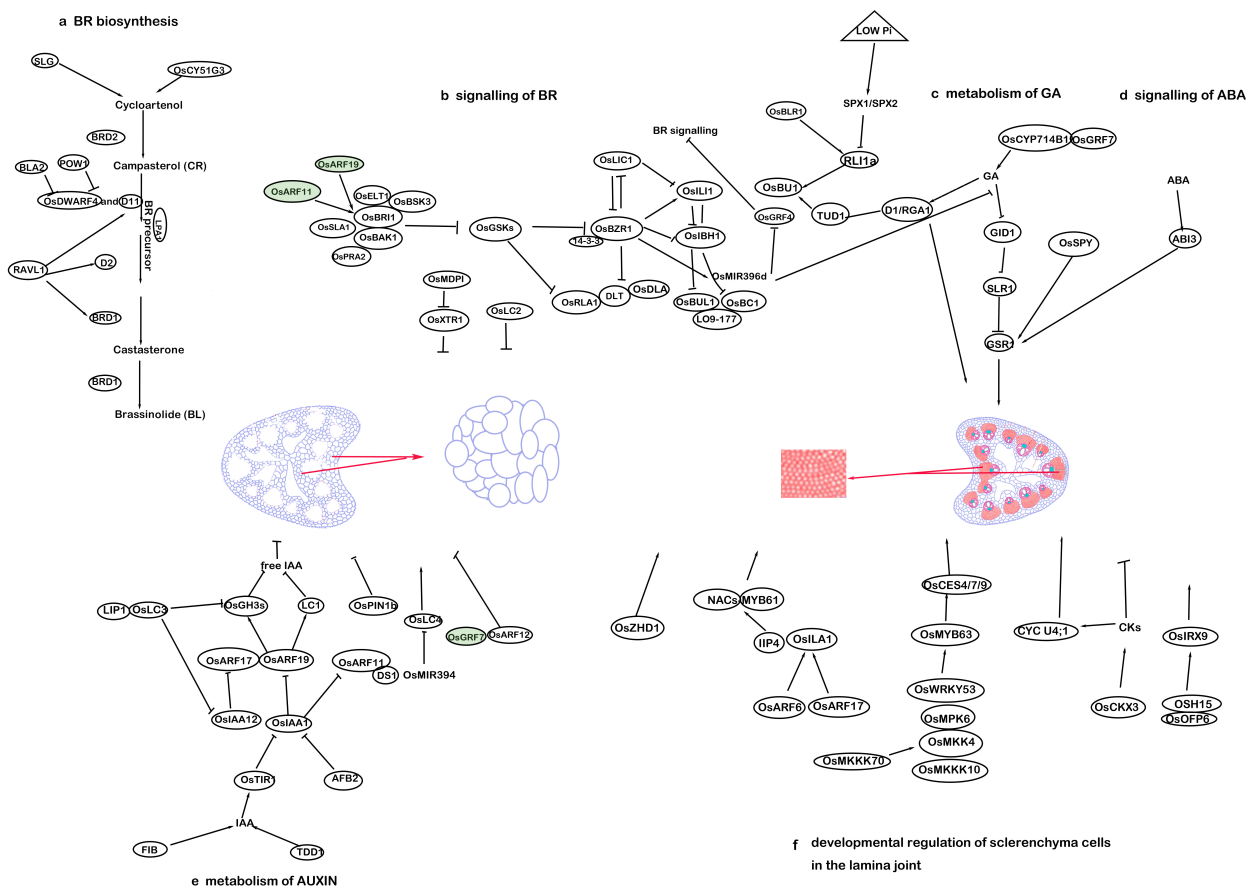


Figure 3. Genetic regulatory network for the lamina joint in rice. Developmental regulation of the lamina joint that is controlled by phytohormones and other factors. (a) BR biosynthesis; (b) signaling of BR; (c) metabolism of GA; (d) signaling of ABA; (e) metabolism of AUXIN; (f) developmental regulation of sclerenchyma cells in the lamina joint and other factors. Positive or negative regulation of leaf angle were indicated by black arrows, or black lines ending in a perpendicular bar, respectively. Certain proteins within the green-labeled circle exhibit recurrent participation across distinct biosynthetic and metabolic processes.

In addition to the identified enzymes in the BR synthesis pathway mentioned above, there are also some TFs that regulate LA indirectly. The *RAVL1* (*Related to ABI3/VP1-Like 1*) activates the transcription of some BR synthesis related genes *D2*, *D11* and *BRD1*, thereby elevating BR levels and thereby increasing LA [21,22]. But there are some genes involved in LA control that still need further study: for example, *POW1* (*Put on weight 1*), which contains a putative helix-turn-helix DNA binding domain, is implicated in regulation of leaf angle by affecting BR homeostasis in rice. The *pow1* mutant exhibits increased LA and shows inhibited expression of BR biosynthesis genes [23]. However, its transcriptional activation or repressive activity remains to be identified.

Abnormal signaling of BR also impacts LA. Previous studies have clarified that BR signaling mainly includes the plasma membrane receptor *OsBRI1* and co-receptor *OsBAK1*, as well as negative regulatory kinase *OsGSKs*, positive regulatory protein *OsBZR1*, and various downstream TFs [24,25]. Studies on conducted BR receptors revealed that both BR receptor mutants *d61-1* and *OsBAK1-AS* exhibit an upright leaf phenotype due to reduced elongation of PCs on the adaxial side of the LJ. It was identified that the interaction between

OsBRI1 and OsBAK1 is regulated by multiple proteins [26,27]. Some of these proteins act as positive regulators to facilitate the interaction between OsBRI1 and OsBAK1, such as OsSLA1. In the *sla1* mutant, the weakened BR signal transduction inhibits the elongation of PCs in the LJ, resulting in an upright leaf phenotype [28]. Meanwhile, the interaction between OsBRI1 and OsBAK1 is inhibited by certain proteins, such as OsPRA2, a rice small G protein, which binds and inhibits the kinase activities of OsBRI1, thereby exerting a negative regulatory effect on BR signaling and resulting in reduced LA in *OsPRA2* overexpressed rice [29].

In addition, there are regulators inhibit the endocytosis and degradation of OsBRI1 by interacting with OsBRI1 alone, resulting in the increased accumulation of OsBRI1 and enhanced BR signaling. For example, ELT1 (Enhanced leaf inclination and tiller number 1) was tissue-specifically expressed in the LJ and tiller buds; its gain-of-function mutant *elt1-D* exhibits the increased LA phenotype due to the proliferation of adaxial cells in the LJ [30].

After activation of the BR receptor, the BR signal was relayed downstream by regulating the kinase activities of OsGSKs (OsGSK1 to OsGSK4) negatively. The presence of mutations in OsGSKs, including simultaneous mutations in three or four genes in *OsGSK1* to *OsGSK4*, were observed to result in enhanced BR signal and increased LA [31], and the loss of function of the *OsGSK3* mutant *osgsk3* also resulted in increased LA [32]. When OsGSKs are activated, it further negatively regulates the downstream target proteins through phosphorylation [31,33]. But the mutants of the target genes exhibit an upright leaf phenotype. For example, OsGSK2 phosphorylates RLA1 (Reduced leaf angle1) regulate BR signaling through DLT (DWARF AND LOW-TILLERING) or OsDLA (Decreased leaf angles). The mutants *rla1*, *dlt*, and *dla* exhibit an upright leaf phenotype [34–37].

OsBZR1, as an important downstream component of BR signaling [25], positively regulates the elongation of paraxial PCs in the LJ, and its activity is inhibited by OsGSK1 [31,33,35,38]. In the absence of BR, OsBZR1 was phosphorylated by the activated kinases OsGSKs leading to its internalization and degradation by ubiquitin-26S proteasome in cytoplasm. The presence of BR inhibits the kinase activities of OsGSKs, thereby inhibition on OsBZR1 activity. Then dephosphorylated OsBZR1 enters into nucleus and regulates the transcription of BR responsive genes [31,33,35,39]. The positive regulation of BR signaling by OsBZR1 has been validated through transgenic experiments, the *OsBZR1-RNAi* transgenic lines exhibit the phenotype of upright leaves [38].

The nucleus entry of OsBZR1 is not only regulated by its phosphorylation state, but also influenced by its interacting proteins. For example, rice 14-3-3 (GF14c) bind to OsBZR1 in the cytoplasm, inhibiting the transfer of OsBZR1 to nucleus, and inhibiting BR signaling through OsBZR1 to some extent. The mutant *gf14c* also exhibit an upright leaf phenotype [38,40]. At high BR levels, the transcription factor OsLIC1 (Leaf and tiller angle increased controller) competitively binds to the *OsBZR1* promoter, weakening BR signaling and act as a negative regulatory factor in the BR signaling pathway. Correspondingly, gain-of-function *lic-1* and *OsLIC1* overexpression mutants exhibit upright leaf phenotype [41].

In addition to the previously identified components in the BR signaling pathway mentioned above, some genes indirectly regulate LA through the BR signaling pathway. For example, *OsMDP1* encodes a transcription factor containing the MADS-box domain, which responds to the BR signal and inhibits the expression of the target genes, such as *OsXTR1* (*xyloglucan endotransglycosylase-related*), which negatively regulates the LA by controlling the expansion of adaxial PCs [42]. Another BR response gene, *OsLC2*, encodes a VIN3-like protein, also involved in LA control. In the *lc2* mutant, the LA becomes larger due to the enhanced proliferation of the paraxial PCs in the LJ [43].

3.2. Regulation by Auxin

Auxin plays a crucial role in the regulation of diverse growth and development processes, including cell proliferation, differentiation, expansion, and elongation. Existing studies have demonstrated the mainly involvement of auxin anabolism, polar transport, and signal transduction in regulation of rice LJ [44,45]. Studies have shown that in the early

development of the LJ, the synthesis and polar transport of auxin lead to its local accumulation, stimulating cell proliferation. Along with LJ development, the local concentration of indoleacetic acid (IAA) decreases, and promotes the elongation of adaxial PCs of the LJ [45].

The biosynthesis of auxin involves two pathways: the tryptophan-dependent pathway and the tryptophan-independent pathway. At present, only the tryptophan-dependent pathway has been reported in rice. For example, the β -subunit of anthranilate synthase encoded gene *OsTDD1* participates in the tryptophan-dependent pathway. In the *tdd1* mutant, the contents of tryptophan and IAA are lower than those in the WT, and the LA is increased [46]. For *FIB* (*FISH BONE*) gene encoding tryptophan aminotransferase, the concentration of endogenous IAA was reduced and auxin polar transport was blocked, leading to increased LA in the *fib* mutant [47].

Some genes are associated with regulating the homeostasis of auxin, such as *OsGH3s*, encoding indole-3-acetic acid amino synthase, which catalyze excessive IAA to bind with a variety of amino acids, maintaining the balance of auxin in vivo. The transcriptions of *OsGH3-1/LC1*, *OsGH3-2*, *OsGH3-5* and *OsGH3-13/TLD1* are regulated by the auxin response factor OsARF19; overexpression of these genes leads to a reduction in free auxin content of the LJ, enhanced elongation of adaxial PCs, and increased LA [48–51].

The PIN family of auxin transporters are key regulatory factors that determine the direction of auxin polar transport [52]. For example, the auxin transporter OsPIN1b controls the asymmetric distribution of IAA on the adaxial and abaxial sides of the LJ, affects adaxial cell proliferation, and regulates the LA. Recently, it was found that the concentration of free auxin in the LJ of the mutant *ospin1b* increased, exhibiting decreased LA, while *OsPIN1b* overexpressed rice exhibited the opposite characteristics [45]. As mentioned above, *FIB* not only plays a crucial role in the auxin synthesis process, but also affects the polar transport of auxin. In the *FIB* functional deficient mutants, auxin polar transport was blocked, affecting adaxial cell proliferation, resulted the enlargement of LA [47].

The nuclear auxin pathway consists of three essential components, including the auxin/indole-3-acetic acid (Aux/IAA) repressors, the TRANSPORT INHIBITOR RESPONSE 1 (TIR1)/AUXIN-SIGNALING F-BOX (AFB) receptors and the auxin response factors (ARFs) [53]. The most comprehensive model of the auxin signaling pathway, which accounts for a majority of observation, relies on a mechanism involving de-repression. The interaction between Aux/IAAs and ARFs in the presence of low levels of auxin leads to the recruitment of co-repressor complexes, thereby inhibiting the activity of ARFs. When the level of auxin increases, the release of ARFs from this repression following auxin-triggered Aux/IAA degradation through the conserved ubiquitin-protosome pathway [53]. It was reported that interfering with the expression of *OsTIR1* or *OsAFB2* through RNAi technology leads to increased LA, and correspondingly, overexpression of *OsIAA1* also exhibits a large LA phenotype [53–56]. *LEAF INCLINATION 4* (*LC4*) encodes an F-box protein and has been identified as the target gene of miR394, both of which are regulated by auxin. The miR394-LC4 module facilitates auxin-mediated regulation of rice leaf inclination and architecture. In the *lc 4* mutant, the elongation and expansion of adaxial PCs in the LJ are inhibited, leading to reduced LA [57].

3.3. Crosstalk between the Phytohormones

The development of leaf LJ is a complex process regulated by multiple hormones. The consensus has been reached that BR plays a leading role in determining LA, while the other phytohormones participate in regulating LA, either positively or negatively [58,59].

3.3.1. BR and Auxin

LJ development is cooperatively regulated by the crosstalk between BR and auxin: auxin acts as a negative regulator of LA by interfering with both BR metabolism and signaling in rice. Two pathways have been discovered, namely the OsBRI1-dependent pathway and the OsBRI1-independent pathway [58–60].

In the OsBRI1-dependent pathway, auxin is involved in OsBRI1 mediated BR signal transduction [60]. For example, OsARF19, an auxin response factor, binds to auxin-responsive element AuxRE of OsBRI1 and activate the its expression, thereby participating in the regulation of BR signal on LJ development [49]. In addition, the plant-specific EMF1-like protein (OsEMF1)/DS1 interacts with auxin-responsive factor OsARF11, jointly binding to the AuxRE element of the *OsBRI1* promoter, playing a similar role to OsARF19 in regulating the development of leaf LJ. Correspondingly, both mutants *osarf11* and *ds1* have smaller LA [59,61].

The OsBRI1-independent pathway is mediated by the BR precursor C-22-hydroxylated and 6-deoxo BRs, in which the BR precursor and auxin regulate early development of the leaf LJ. Existing research has found that the crosstalk between BR and auxin is established through a transcription factor *LPA1* (*Loose Plant Architecture1*). *LPA1* is a negative regulator of leaf LJ development; its mutant *lpa1* has loose plant architecture with increased leaf angle, while *LPA1* overexpression results in reduced LA [58,60–62]. Genetic and biochemical analyses indicate that *LPA1* acts as a suppressor of auxin signaling, specifically in its interaction with C-22-hydroxylated and 6-deoxo BRs. This regulatory mechanism independently controls lamina inclination, separate from the role played by *OsBRI1* [58].

3.3.2. BR and GA

Current research suggests that gibberellin (GA) inhibits the synthesis and signaling of BR, while BR promotes GA synthesis and response through a feedback mechanism [63,64]. The BR loss of function mutants generally have an upright leaf phenotype, while GA functional deficient mutants rarely have this phenotype, and the increased LA phenotype caused by enhanced BR signaling can be inhibited by exogenous GA. Some studies suggest that the inhibitory effect of GA on BR signaling may be attributed to the key negative regulator of the GA signaling pathway DELLA (OsSLR1), which binds to the positive regulator OsBZR1 of the BR pathway, thereby inhibiting the transcription of the target genes activated by OsBZR1 [63]. The association between GA and BR signals in LA regulation is also confirmed by the study on miRNA. For example, it was found that *OsMIR396d* is activated by OsBZR1. On the one hand, OsMIR396d regulates LA by inhibiting the expression of its target gene, OsGRF4. On the other hand, it downregulates the expression of the genes related to GA biosynthesis and signal transduction by inhibiting the expression of *OsGRF6* [65]. In *OsMIR396d* overexpressed rice, the response to BR signal was enhanced in LJ, resulting in larger LA [65].

In addition to inhibiting BR signaling, GA also inhibits BR synthesis [63,64]. For example, *OsSPY* encoding acetylglucosamine transferase functions as a suppressor in GA signaling. In *OsSPY* antisense and RNAi plants, the endogenous BR levels increase, leading to increased LA [66,67]. *OsGSR1*, a member of the GAST (GA-stimulated transcript) family gene, is up- and downregulated by GA and by BR, respectively. *OsGSR1* mediates the interaction between BR and GA signaling pathways through its interaction with DIM/DWF1, an enzyme in BR biosynthesis. In its *OsGSR1-RNAi* transgenic plant, the level of endogenous BR reduces, leading to an erect leaf phenotype [68]. Evidence also implicates that *D1/RGA1*, the heterotrimeric G protein α subunit, is involved in a GA and BR signaling pathway [61,69]. The mutant of *d1/rga1*, which displays erect leaves, is identified as a GA signaling mutant originally. Subsequently, it was found that *D1/RGA1* interacts with the E3 ubiquitin ligase TUD 1 to collectively mediate BR signaling. The *d1* mutant displays reduced leaf angle, insensitivity to GA, and diminished sensitivity to BR [61,69].

3.3.3. Synergism between the Other Phytohormones

GA and auxin signal may act together in LJ development. Recent studies have found that the transcription factor OsGRF7 regulates GA synthesis and auxin signaling [70]. The overexpression of OsGRF7 in rice with compacting plant architecture and increasing the proliferation of PC layers in the culm and adaxial side of the LJ are enhanced, leading to decreased leaf angle. Conversely, the knockout and knockdown lines of OsGRF7 exhibit

contrasting phenotypes with increased leaf angle. Further research has shown that OsGRF7 not only interacts with the promoter of the cytochrome P450 gene *OsCYP714B1* to regulate GA synthesis, but also binds to the promoter of the auxin response factor *OsARF12* to control auxin signaling [70]. Moreover, the synergistic interaction between low concentrations of ABA and BR partially depends on the ABI3-OsGSR1 module. It was found that exogenous ABA treatment activates ABI3 to induce the expression of *OsGSR1*, then *OsGSR1* binds with BR biosynthesis enzymes DIM/DWF1/BRD2, inducing BR biosynthesis, leading to an LA increase [71]. Transcriptome analyses validate that approximately 60% of low-concentration ABA early response genes exhibit consistent regulation by BR [71].

3.4. Regulation by TFs

In the Plant Transcription Factor Database (<https://planttfdb.gao-lab.org/>, accessed on 13 July 2024), 2408 TFs (1862 loci) in japonica and 1891 TFs (1891 loci) in indica are identified, classified into 56 families in rice. TFs play crucial roles in plant signaling pathways, defining plant responses to both biotic and abiotic stimuli. Additionally, they are involved in responding to internal signals, which help coordinate various interacting partners throughout developmental processes [72]. Current research indicates that the transcription factor families involved in regulating rice LA include bHLH (basic helix-loop-helix), ZF-HD (zinc finger homeodomain), LBD (lateral organ boundaries domain), WRKY (WRKY domain), and so on (as shown in Table 1).

Table 1. The genes involved in the rice lamina joint development.

Gene	MSU Locus	The Encoded Protein	Leaf Inclination Regulation	Regulatory Mechanism	References
<i>CYP51G3</i>	LOC_Os05g12040	A obtusifolios 14 α -demethylase	Promotes the expansion of parenchyma cells on the adaxial side of the lamina joint; positive	BR biosynthesis	[9]
<i>SLG</i>	LOC_Os08g44840	a BAHD acyltransferase-like protein	Adaxial parenchyma cell length; positive	BR biosynthesis	[10]
<i>DWARF2</i> <i>BRD2</i> <i>DIM/DWF1</i>	LOC_Os10g25780	A δ (24)-sterolreductase	Positive	BR biosynthesis	[11–13]
<i>D11</i>	LOC_Os04g39430	A cytochrome P450 protein, CYP724B1	Adaxial parenchyma cell length; positive	BR biosynthesis	[14]
<i>DWARF4</i>	LOC_Os03g12660	A cytochrome P450 protein, CYP90B2	Positive	BR biosynthesis	[1]
<i>CYP90D2/D2</i>	LOC_Os01g10040	A cytochrome P450 protein, CYP90D2	Positive	BR biosynthesis	[15]
<i>BRD1</i> <i>OsDWARF</i>	LOC_Os03g40540	BR C-6 oxidase	Positive	BR biosynthesis	[16]
<i>BLA2</i>	LOC_Os01g29150	A cytochrome P450 monooxygenase, CYP734As	Positive	BR biosynthesis	[19,20]
<i>RAVL1</i>	LOC_Os04g49230	Related to ABI3/VP1-Like 1	Positive	BR biosynthesis and BR signaling	[21,22]
<i>POW1</i>	LOC_Os07g07880	Contains a putative helix-turn-helix DNA binding domain	Negative	Affects BR homeostasis	[23]

Table 1. Cont.

	Gene	MSU Locus	The Encoded Protein	Leaf Inclination Regulation	Regulatory Mechanism	References
BR	<i>OsBRI1</i>	LOC_Os01g52050	Brassinolide receptor kinase	Positive	BR signaling	[26]
	<i>OsBAK1</i>	LOC_Os08g07760	BRI1-ASSOCIATED RECEPTOR KINASE	Adaxial parenchyma cell elongation; positive	BR signaling	[27]
	<i>OsBZR1</i>	LOC_Os07g39220	BR-signaling factor	Adaxial parenchyma cell elongation; positive	BR signaling	[38,73]
	<i>OsSLA1</i>	LOC_Os04g41030	Immunity-associated leucine-rich repeat receptor-like protein kinase	Positive	BR signaling	[28]
	<i>OsPRA2</i>	LOC_Os06g50060	Small GTP-binding protein; small G protein	Negative	BR signaling	[29]
	<i>ELT1</i>	LOC_Os02g58390	Receptor-like protein that has enhanced leaf inclination and tiller number	Positive	BR signaling	[30]
	<i>OsGSK1</i>	LOC_Os01g10840	Glycogen synthase kinase3-like	Negative	BR signaling	[33]
	<i>OsGSK2</i>	LOC_Os05g11730	GSK3/SHAGGY-like kinase	Negative	BR signaling	[35,36]
	<i>OsGSK3</i>	LOC_Os02g14130	GSK3/SHAGGY-like kinase	Negative	BR signaling	[32]
	<i>OsGSK4</i>	LOC_Os06g35530	GSK3/SHAGGY-like kinase	Negative	BR signaling	[31]
	<i>DLT</i>	LOC_Os06g03710	GRAS protein	Positive	BR signaling	[36]
	<i>OsDLA</i>	LOC_Os01g71970	GRAS protein	Positive	BR signaling	[37]
	<i>RLA1/SMOS1</i>	LOC_Os05g32270	GRAS protein	Positive	BR signaling	[34,35]
	<i>14-3-3 (GF14c)</i>	LOC_Os08g33370	G-box factor 14-3-3 homologs	—	BR signaling	[38,40]
	<i>OsLIC1</i> <i>OsLIC</i>	LOC_Os06g49080	CCCH-Type Zinc Finger Protein	Negative	BR signaling	[41]
	<i>OsMDP1</i>	LOC_Os03g08754	Rice MADS-box transcription factor	Adaxial parenchyma cell elongation; negative	BR signaling	[42]
AUXIN	<i>OsXTR1</i>	LOC_Os11g33270	Xyloglucan endotransglycosylase-related gene 1	Adaxial parenchyma cell elongation; positive	BR signaling	[42]
	<i>OsLC2</i>	LOC_Os02g05840	Vernalization insensitive 3-like protein	Adaxial parenchyma cell division; negative	BR signaling	[43]
	<i>OsTDD1</i>	LOC_Os04g38950	Rice Anthranilate Synthase β -Subunit	Negative	IAA biosynthesis	[46]
	<i>FIB/OsTAR2</i> <i>OsTAA1</i>	LOC_Os01g07500	Tryptophan aminotransferase	Negative	IAA biosynthesis	[47]
	<i>LC1/OsGH3-1</i>	LOC_Os01g57610	Indole-3-acetic acid-amido synthetase	Adaxial parenchyma cell length and division; positive	Auxin signaling	[50]
	<i>OsGH3-2</i>	LOC_Os01g55940	Indole-3-acetic acid-amido synthetase gene	Negative	Auxin signaling	[48]
	<i>OsGH3-5</i>	LOC_Os05g50890	JA-amino acid synthetase	Negative	Auxin signaling	[49]

Table 1. Cont.

	Gene	MSU Locus	The Encoded Protein	Leaf Inclination Regulation	Regulatory Mechanism	References
AUXIN	<i>OsGH3-13/TLD1</i>	LOC_Os11g32510	Auxin-responsive GH3 gene family member	Positive	Auxin signaling	[51]
	<i>OsARF19</i>	LOC_Os06g48950	Auxin response factor	Adaxial parenchyma cell division; positive	BR and IAA interactions	[49]
	<i>OsPIN1b</i>	LOC_Os02g50960	Auxin efflux transporter	The increase in the adaxial cell division; positive	Auxin signaling	[45]
	<i>OsTIR1</i>	LOC_Os05g05800	Auxin receptor	Negative	Auxin signaling and interaction with N	[53,54]
	<i>OsAFB2</i>	LOC_Os04g32460	Auxin receptor	Negative	Auxin signaling interaction with N	[53]
	<i>OsIAA1</i>	LOC_Os01g08320	Aux/IAA protein	Positive	Auxin signaling	[55]
	<i>OsLC4</i>	LOC_Os01g69940	F-box protein	Adaxial parenchyma cell length; positive	Auxin signaling	[57]
	<i>OsMIR394</i>	miRBase Library Accession Number:MI0001027	MicroRNA	Adaxial parenchyma cell length; positive	Auxin signaling	[57]
Crosstalk between the phytohormones	<i>OsIAA12</i>	LOC_Os03g43410	Aux/IAA protein	Negative	Auxin signaling	[74]
	<i>DS1</i>	LOC_Os01g12890	EMF1-like protein	Positive	BR and IAA interactions	[59]
	<i>OsARF11</i>	LOC_Os04g56850	Auxin response factor	Positive	BR and IAA interactions	[59]
	<i>LPA1</i>	LOC_Os03g13400	NDETERMINATE DOMAIN Protein	Adaxial parenchyma cell length; positive	BR and IAA interactions	[59]
	<i>OsSLR1</i>	LOC_Os03g49990	DELLA protein	Negative	BR and GA interactions	[63]
	<i>OsMIR396</i>	LOC_Os04g57830	MicroRNA	Adaxial cell size; positive	BR and GA interactions	[65]
	<i>OsGRF4/GS2 GL2/LGS1 GLW2</i>	LOC_Os02g47280	Growth-regulating factor	Negative	BR and GA interactions	[65]
	<i>OsSPY</i>	LOC_Os08g44510	O-linked N-acetylglucosamine transferase	Negative	BR and GA interactions	[66,67]
	<i>OsGSR1</i>	LOC_Os06g15620	GA-stimulated transcript gene	Positive	BR and GA interactions	[68]
	<i>D1/RGA1</i>	LOC_Os05g26890	G protein α subunit	Positive	BR and GA interactions	[61,69]
	<i>TUD1</i>	LOC_Os03g13010	U-box E3 ubiquitin ligase	Positive	BR signaling	[69]
	<i>OsGRF7</i>	LOC_Os12g29980	Growth-regulating factor	Increased parenchymal cell layers in the adaxial side of the lamina joints	IAA and GA signaling	[70]
	<i>ARF12</i>	LOC_Os04g57610	Auxin response factor	Negative	Auxin signaling	[70]
	<i>ABI3</i>	LOC_Os01g68370	B3 domain transcription factor	Positive	IAA and ABA signaling	[71]

Table 1. Cont.

	Gene	MSU Locus	The Encoded Protein	Leaf Inclination Regulation	Regulatory Mechanism	References
Transcription factors	<i>OsBUL1</i>	LOC_Os02g51320	Atypical bHLH protein	Adaxial parenchyma cell length; positive	BR signaling	[75]
	<i>OsBC1</i>	LOC_Os09g33580	bHLH protein	Adaxial parenchyma cell length; positive	BR signaling	[75]
	<i>LO9-177</i>	LOC_Os03g43910	OsBUL1-interacting protein; kxDL motif-containing protein	Adaxial parenchyma cell length; positive	BR signaling	[75]
	<i>OsILI1</i>	LOC_Os04g54900	bHLH transcription factor	Adaxial parenchyma cell elongation; positive	BR signaling	[76]
	<i>OsIBH1</i>	LOC_Os04g56500	bHLH protein	Adaxial parenchyma cell elongation; negative	BR signaling	[76]
	<i>OsBLR1</i> <i>OsbHLH079</i>	LOC_Os02g47660	bHLH transcription factor	The expansion of cell size in the adaxial side; positive	BR signaling	[77,78]
	<i>OsZHD1</i>	LOC_Os09g29130	Zn-finger transcription factor SPOC	Positive	Other	[79]
	<i>OsLC3</i>	LOC_Os06g39480	domain-containing transcription suppressor	Adaxial parenchyma cell length; negative	Auxin signaling	[74]
	<i>LIP1</i>	LOC_Os10g37640	HIT zinc finger domain-containing protein	Adaxial parenchyma cell length; negative	Auxin signaling	[74]
	<i>OsWRKY53</i>	LOC_Os05g27730	WRKY transcription factor	Adaxial parenchyma cell elongation; positive	BR homeostasis	[80,81]
	<i>OsBU1</i>	LOC_Os06g12210	bHLH protein	Adaxial parenchyma cell length; positive	BR signaling	[82]
Sclerenchyma cells development	<i>CYC U4;1</i>	LOC_Os10g41430	Cyclin-like gene	Abaxial sclerenchyma cell proliferation; negative	BR signaling	[83]
	<i>OsCKX3</i>	LOC_Os10g34230	Cytokinin oxidase/dehydrogenase	Asymmetric proliferation of the cells and vascular bundles; negative	CK signaling	[84]
	<i>OsILA1</i>	LOC_Os06g50920	Mitogen-activated protein kinase kinase	Abnormal vascular bundle formation and cell wall composition; negative	Other	[85]
	<i>IIP4</i>	LOC_Os04g38520	ILA1-interacting protein 4	The secondary cell wall biosynthesis; negative	The secondary cell wall biosynthesis	[85,86]
	<i>NAC29</i>	LOC_Os08g02300	NAC Transcription Factor	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>NAC31</i>	LOC_Os08g01330	NAC Transcription Factor	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]

Table 1. Cont.

	Gene	MSU Locus	The Encoded Protein	Leaf Inclination Regulation	Regulatory Mechanism	References
Sclerenchyma cells development	<i>MYB61</i>	LOC_Os01g18240	MYB family transcription factor	the cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>CesA4</i>	LOC_Os01g54620	Cellulose synthase catalytic subunit 4	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>CesA7</i>	LOC_Os10g32980	Cellulose synthase catalytic subunit 7	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>CesA9</i>	LOC_Os09g25490	Cellulose synthase catalytic subunit 9	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>CESA6</i>	LOC_Os07g14850	Cellulose synthase catalytic subunit 6	The cellulose biosynthesis; positive	The secondary cell wall biosynthesis	[86]
	<i>OsARF6</i>	LOC_Os02g06910	Auxin response factor	negative	Auxin signaling	[87]
	<i>OsARF17</i>	LOC_Os06g46410	Auxin response factor	negative	Auxin signaling	[87]
	<i>OSH15 (KNOX)</i>	LOC_Os07g03770	KNOX family class 1 homeobox gene of rice	The secondary cell wall biosynthesis; negative	The secondary cell wall biosynthesis	[88]
	<i>OsIRX9</i>	LOC_Os07g49370	glycosyltransferase family 43 protein	Xylan; negative	Xylan	[88]
	<i>OsOFP6</i>	LOC_Os01g60810	OVATE family protein 6	The secondary cell wall biosynthesis; negative	The secondary cell wall biosynthesis	[88]
	<i>OsMYB63</i>	LOC_Os04g50770	R2R3-type MYB family transcription factor	Positive	The cell wall biosynthesis	[81]
	<i>MKKK10 SMG2</i>	LOC_Os04g47240	Mitogen activated protein Kinase Kinase 10	Positive	BR homeostasis	[89]
	<i>MAPKK4</i>	LOC_Os02g54600	Mitogen activated protein Kinase Kinase 4	Positive	BR homeostasis	[80,89]
Other	<i>MAPK6</i>	LOC_Os06g06090	Mitogen activated protein Kinase 6	Positive	BR homeostasis	[80,89]
	<i>MKKK70</i>	LOC_Os01g50410	Mitogen activated protein Kinase Kinase 70	Positive	BR homeostasis	[90]
	<i>SPX1</i>	LOC_Os06g40120	SPX family protein	Cell elongation of Lamin joint; negative	Involved in Pi and BR interactions	[91]
	<i>SPX2</i>	LOC_Os02g10780	SPX family protein	Cell elongation of Lamin joint; negative	Involved in Pi and BR interactions	[91]
	<i>RLI1</i>	LOC_Os04g56990	GARP family transcription factor	Cell elongation of Lamin joint; positive	Involved in Pi and BR interactions	[91,92]
	<i>OsWAK11</i>	LOC_Os02g02120	Wall-associated receptor kinase 11	Negative	The cell wall biosynthesis	[93]

Currently, the number of HLH family members are the most known TFs regulating LA. For example, the bHLH transcriptional activator OsBC1 binds with OsBUL1 in the presence of LO9-177 protein, forming a possible trimeric complex, regulating the elongation of PCs in LJ [75]. The positive regulator of LA ILI1 may deactivate inhibitory bHLH transcription factors IBH1 (ILI1 binding bHLH) through heterodimerization. The mutant of *ili1-D* shows an increased lamina inclination phenotype, and similar with the overexpression of *IBH1*. Additionally, the transcription of *OsIBH1* is regulated by *OsBZR1* in the BR signaling

pathway [76,94]. A typical bHLH transcription factor, OsBLR1 (BRASSINOSTEROID-RESPONSIVE LEAF ANGLE REGULATOR 1)/OsbHLH079, was identified as a positive regulator of LA; overexpression of the gene increased the LA, whereas *OsBLR1*-knockout mutants are the opposite [77]. *OsbHLH079* was isolated from the enhancer-trap T-DNA insertion mutant, which showed a wide LA phenotype, acting as a positive regulator of BR signaling, and is involved in LJ development by promoting the expansion of cell size on the adaxial side [78].

Some zinc finger proteins have been reported to regulate the development of leaf LJ; overexpression of *OsZHD1* resulted in abaxially curled and drooping leaf blades because of the increased number and the abnormal arrangement of bulliform cells in leaves, suggesting that *OsZHD1* acts as a positive regulator of LA [79].

Although most of the known TFs related to LJ development are involved in BR signaling, in recent years, it has been found that some TFs are involved in auxin signaling, such as transcription suppressor LC3 and HIT zinc finger domain-containing protein LIP1 (LC3-interacting protein 1), involved in LJ development regulation by interaction, synergistically suppressing auxin signaling by inhibiting the transcription of target genes *OsIAA12* and *OsGH3.2* [74]. The mutant *osl3* has excessive elongation of the paraxial PCs of the pulvinar, and increased LA [74].

4. Regulation of SC Development in LJ

Sclerenchyma tissue provides the mechanical support for leaf LJ. The area of sclerenchyma tissue and the thickness and composition of the secondary wall influence the change in mechanical support strength and LA.

4.1. Regulation of SC Proliferation

Up to now, compared with the progress on PC proliferation and differentiation in LJ development, studies on the proliferation regulation of SCs are limited. Up to now, only *CYC U4; 1* and *OsCKX3* have been cloned in rice. *CYC U4; 1*, a cyclin coding gene, is accompanied by the development of lamina joint and functions as a positive regulator in abaxial SC proliferation. It is also related to LA control, and its expression and protein activity of it are both regulated by BR signal [83]. Cytokinin accumulation mediated by rice oxidase/gehydrogenase 3 (*OsCKX3*) controls LJ development [95]; *OsCKX3* functions to maintain cytokinin homeostasis and is related to the reversible oxidative lysis of cytokinin. The *osckx3* mutants exhibited reduced LA, whereas the overexpression lines displayed increased LA, which arose from asymmetric proliferation of the cells and VBs in the LJ, and the expression of *CYC U4; 1* is upregulated in the mutant *osckx3*, resulting in asymmetric proliferation of cells in the LJ and smaller LA [84].

4.2. Regulation of Secondary Wall Synthesis

It was found that changing the structure of VBs and secondary wall synthesis had a great impact on LA. For example, *ILA1*, a Raf-like MAPKKK encoded gene, controls the development of mechanical tissue and cell wall composition in the LJ [85]. IIP 4, one of *ILA1*'s phosphorylation substrates, interacts with the secondary wall synthesis-up regulator NAC29/NAC31, inhibiting the expression of *OsMYB61* and secondary wall cellulose synthesis gene *CESA* to repress secondary wall synthesis, changing the content of cellulose and xylan in the secondary wall of SCs [86]. Further study showed that the expression of *ILA1* is controlled by auxin response factors *OsARF6* and *OsARF17*. The double mutants *osarf6/osarf17* displayed reduced composition of the secondary cell wall in sclerenchymatous cells, leading to an exaggerated flag LA [87].

In addition, the KNOX (knotted-like homeobox) protein OSH15 functions as a transcription activator to activate the expression xylan synthesis gene *OsIRX9* (*IRREGULAR XYLEM 9*), and increasing the content of xylan in secondary wall [88]. OVATE family proteins 6 (*OsOFP6*) interact with OSH15, enhancing the transcriptional activity of OSH15. Knockdown *OsOFP6* expression results in increased LA due to a thinner secondary cell

wall, characterized by reduced cellulose and lignin levels, while overexpression of *OsOFP6* leads to a the thicker secondary cell wall with increased lignin content [88].

It was identified that *OsWRKY53* positively regulates rice BR signaling involved in LA control [80]. Further research showed that *OsWRKY53* functions as a transcriptional repressor and regulates *OsMYB63* to exert inhibitory control over its expression. The overexpression of *OsWRKY53* and knockout of *OsMYB63* resulted in thinner SC walls, suggesting that the two transcriptional factors cooperate in regulating the differentiation of SC [81]. Meanwhile, *OsWRKY53* is a substrate of *OsMAPKKK10*-*OsMAPKK4*-*OsMAPK6* cascade in LA control, mediating the crosstalk between BR signaling and the MAPK (mitogen-activated protein kinase) pathway [89]. In addition, *OsMKKK70* might be another MAPKKK function upstream of the *OsMAPKK4*-*OsMAPK6* *OsWRKY53* pathway, which regulates LA in rice [90].

5. Regulation by Environmental Factors

Environmental factors such as nutrients, light, temperature, and drought, to a greater or lesser extent, have an impact on leaf LJ development [3].

Some evidence comes from the study of inorganic ions, such as the reported negative regulatory role of Pi starvation-induced proteins *SPX1* and *SPX2* on leaf inclination by binding and inhibiting transcription factor *RLI1* (REGULATOR OF LEAF INCLINATION1) [91]. *RLI1* activates the downstream genes directly, such as *BUI1*, which is upregulated by BR and control elongation of the LJ cells [91]. Further study found that the alternative splicing of *RLI1* produces two protein isoforms, namely *RLI1a* and *RLI1b*, both of which play crucial roles in regulating Pi starvation signaling by directly activating genes involved in brassinolide (BL) biosynthesis and signaling [92]. As the major nitrogen resource, NH_4^+ exerts profound effects on rice growth and yields. It has been revealed that a crosstalk mechanism exists between NH_4^+ and BR, whereby NH_4^+ promotes BR biosynthesis through *miR444*. Subsequently, *miR444* positively regulated BR biosynthesis by targeting *MADS-box* proteins, which directly repress the transcription of *OsBRD1* (*BR-deficient dwarf 1*), a key BR biosynthetic gene [96]. In addition, there are complex relationships between different ions: Pi and Fe consistently induce antagonistic physiological effects in plants. Fe deficiency promotes Pi accumulated, and inhibits the transcriptional level of BR biosynthesis and signal-related genes in rice, thereby promoting the LJ cell elongation and enhancing the LA [75,82]. In addition, endogenous levels of SL (strigolactones) in rice increased in response to nitrogen, phosphate, or sulfate (-N, -P, or -S) deficiency, suggesting that elevated endogenous SLs might regulate LA in rice, negatively. Correspondingly, the rice SL mutants show increased LA [97].

The rates of plant cell elongation exhibit variations in response to day-night alternation, the nexus between cell wall remodeling and phytohormone signaling during plant cell elongation had been revealed by the study on *cell wall-associated kinase11* (*OsWAK11*). *OsWAK11* interacts with and phosphorylates *OsBRI1*, a BR receptor, leading to the inhibition of BR signaling. Meanwhile, the extracellular domain of *OsWAK11* exhibits enhanced binding capacity with methyl-esterified pectin. The stability of *OsWAK11* is light-dependent; while its degradation is by proteasome in darkness, *OsWAK11* serves as a crucial link between the changes in cell wall pectin methyl-esterification changes and BR signaling in LA control [93]. Besides the impact of day and night on the LJ development, UV-B intensity also affects plant morphology, including rice LA. Under field-enhanced UV-B radiation treatment, the contents of BR and GA increased, while auxin content decreased compared with that in natural light. The content of cellulose and hemicellulose both reduced in the pulvini, while the areas of thick-walled cell and vascular bundle in leaf pulvini declined with increasing radiation intensity, owing to the significantly altered expression of LA regulation genes, such as *OsBUL1*, *OsGSRI1*, and *OsARF19*, and the cell wall fiber level control gene *ILA1* [98].

6. Perspectives

The regulation mechanisms of important rice agronomic traits have received significant attention due to its dual role as a main staple crop and a model plant. LA is an important trait in rice architecture, which is closely related to yield. For example, the function of *OsBAK1* is related to some important agricultural traits of rice, including LA, plant height, grain morphology, and disease resistance responses. Suppressing the expression level of *OsBAK1* can generate new rice varieties with erect leaves and normal reproduction. Therefore, *OsBAK1* is looked at as a promising molecular breeding tool for improving rice grain yield through the modification of rice architecture [25]. Moreover, the LA architecture of smart canopy 1 (*ZmLAC1*) was used to contribute smart-canopy-like plant architecture in maize, which was providing a one-step haploid induction editing technique system and accelerated strategy for developing high-density-tolerant cultivars [99].

In recent years, the outstanding progress in elucidating the underlying genetic mechanisms implicated in rice LJ development and subsequently regulating LA has been made. The development of rice LJ is finely controlled by various factors including plant hormones, environmental factors, biological and abiotic factors. Various kind of TFs and protein kinases participate in regulating the corresponding biological processes of LJ development, which forming intricate associations with different hormones. In addition, different environmental factors such as nutrients, light, and temperature also affect the development process of LJ tissue. Although some reviews have discussed the development of leaf LJ and the regulation of LAs from different perspectives [3,5,6,25,100], new insights, such as the relationship between auxin signaling and MAPK cascade signaling, in which auxin signaling regulates the development of parenchyma tissues by regulating MAPK cascade signaling, have been discovered recently (Figure 3). Overall, there are still many gaps that need to be filled; the complete genetic network of LJ development remains unknown, and the precise mechanism is still an urgent need to be revealed.

Rice LA is determined by the development of the LJ, especially the proliferation and elongation PCs, and the proliferation, differentiation, and secondary wall synthesis of SCs. Up to now, most studies focus on the developmental regulation of PCs, including phytohormones, the functions of BR, auxin, and the synergism and antagonism between different hormones, such as BR and auxin and BR and GA. In contrast, there is relatively little research on the developmental regulation of SCs, and the underlying mechanism is not yet systematic.

The currently known genes related to the development of leaf LJ mainly include hormone synthesis and signaling genes, downstream TFs, MAPK cascades components, and various enzymes involved in cell wall synthesis. Most of these genes are involved in the regulation of multiple agronomic traits, restricting the application of specific gene resources in terms of crop improvement to some extent. Accordingly, how to use these known genes reasonably and selectively modify the phenotype of rice to cultivate elite rice varieties is the focus of subsequent research. The advent of genome editing has made it possible, with the support of CRISPR/Cas9 and other technologies, to cultivate high-yield, high-quality, and environmental stress-tolerant/-resistant rice varieties effectively by precise editing of genes related to LJ development.

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