



Genetic control of panicle architecture in rice

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ABSTRACT

Rice panicle architecture affects grain number per panicle and thereby grain yield. Many genes involved in control of panicle architecture have been identified in the past decades. According to their effect on phenotype, these genes are divided into three categories: panicle branch and lateral spikelets, multifloret spikelets, and panicle type. We review these genes, describe their genetic regulatory network, and propose a strategy for using them in rice breeding. These findings on rice panicle architecture may facilitate related studies in other crops.

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1. Introduction

Rice (*Oryza sativa* L.) plays an essential role in global food security, serving as the primary nutritive source for almost half of the world population [1]. Rice yield is a complex trait multiplicatively determined by three main factors: grain weight, panicle number per plant, and grain number per panicle [2]. In breeding practice over the past 40 years in China, an increase in yield reflects an increase in grain number per panicle (Fig. 1). Dissection of the genetic mechanism controlling grain number per panicle would thus be an efficient way for breeders to improve rice yield.

Grain number per panicle is determined largely by panicle morphology, which is determined mainly by panicle branching. The rice panicle belongs to a kind of inflorescence with limited growth, consisting of primary branches, secondary branches, and spikelets on the branches. The development process of rice panicle is complex. In the reproductive growth period, the shoot apical meristem (SAM) of rice transforms into an inflorescence meristem (IM), differentiates into a primary branch meristem (PBM), and then aborts. Secondary branch meristems (SBM) are produced successively on the primary branch, further differentiating into spikelet branch meristems (SM) and lateral spikelet meristems (LSM). In the same period, the top of the PBM differentiates into a terminal spikelet meristem (TSM) [3,4]. These branches and their differentiated spikelet meristems will eventually form the basic structure of rice panicle and determine the grain number of the panicle (Fig. 2).

Here we review recent advances in elucidating the genetic mechanisms underlying rice panicle development, including panicle branches, spikelets, and panicle type.

2. Panicle branch and lateral spikelet

In rice panicle, primary branches are produced on the central axis, known as the rachis, and secondary branches are produced on the primary branches. Many genes involved in panicle branch formation have been identified and can be classified into several core regulator-dependent pathways, including GRAIN NUMBER 1a (*Gn1a*), IDEAL PLANT ARCHITECTURE 1 (*IPA1*), LAX PANICLE1 (*LAX1*), FRIZZY PANICLE (*FZP*), MONOCULM 1 (*MOC1*) and TAWAWA1 (*TAW1*) as shown in Fig. 3.

Gn1a, encoding cytokinin oxidase/dehydrogenase *OsCKX2*, is the first major QTL associated with grain number per panicle. Rice cultivars with reduced *OsCKX2* expression produce more primary branches and higher yield owing to the increased cytokinin content in panicles [5]. In contrast to *OsCKX2*, *LONELY GUY* (*LOG*) encodes a cytokinin-activating enzyme, directly converting non-active cytokinin and its nucleotide complex to an active form. The *log* mutant, which has a defect in the synthesis of active CKs, produces a smaller panicle with fewer primary branches than the wild type [6]. A zinc finger protein, DROUGHT AND SALT TOLERANCE (*DST*), directly regulates the expression of *OsCKX2*. The mutant allele *DST^{reg1}* interrupts the directed regulation of *OsCKX2* expression and elevates CK levels in the young panicle [7]. *OsER1* is a negative regulator of grain number per panicle, acting upstream of the *OsMKK10-OsMKK4-OsMPK6* cascade and regulating the

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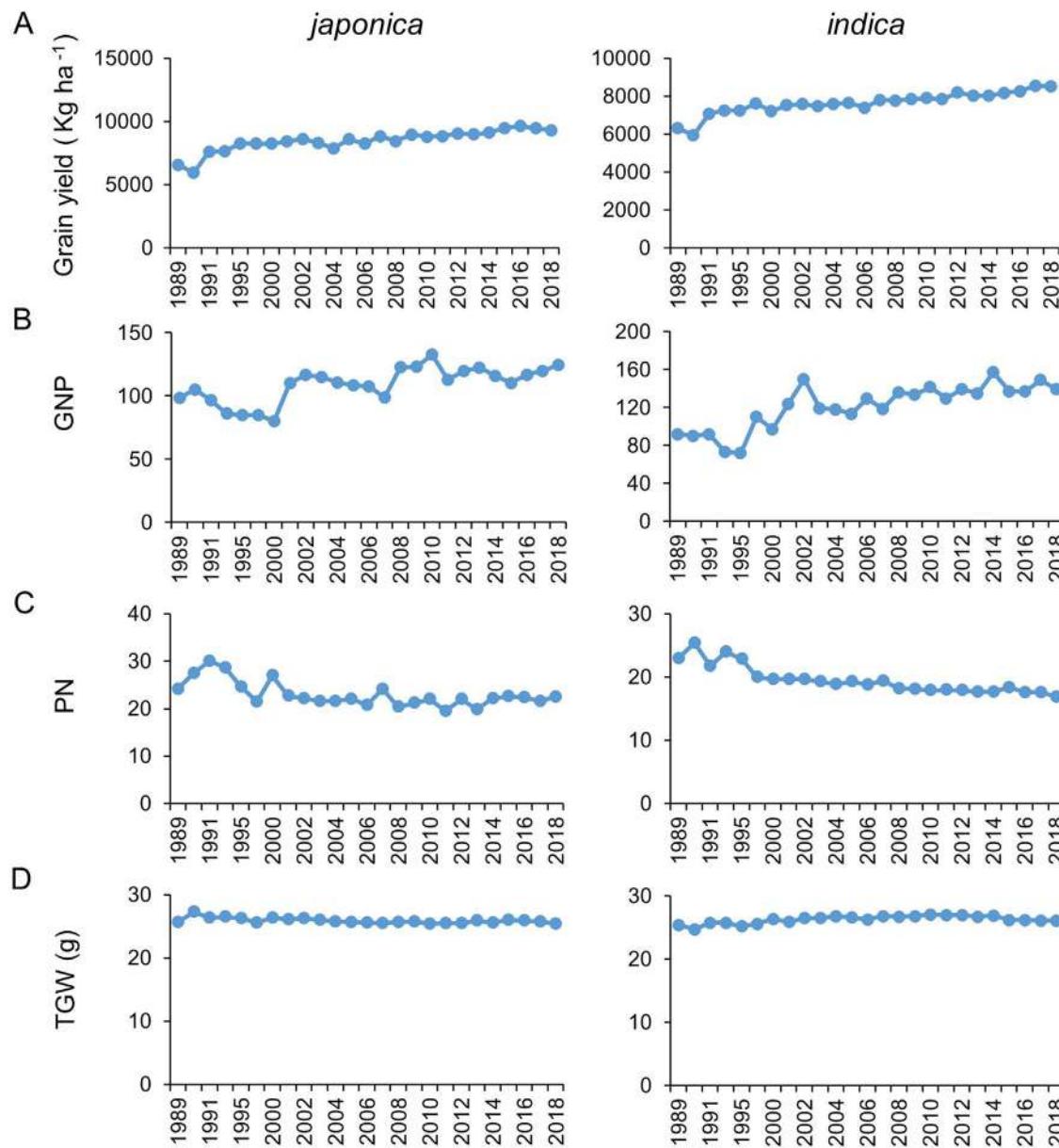


Fig. 1. Statistical analysis of yield characters for released rice cultivars from 1989 to 2018 in China. Statistical results for grain yield (A), GNP (B), PN (C), and TGW (D) for released *japonica* (left panel) and *indica* (right panel) rice cultivars from 1989 to 2018 in China. GNP, grain number per panicle; PN, panicle number; TGW, thousand-grain weight. Data retrieved from CHINA RICE DATA CENTER (<http://www.ricedata.cn/>).

phosphorylation level of OsMPK6 together with OsMKK10 and OsMKK4. It participates in the morphogenesis of rice panicles by regulating local cell division and metabolism [8]. *GRAIN SIZE AND NUMBER1* (*GSN1*) encodes the mitogen-activated protein kinase phosphatase OsMPK1, which can interact with OsMPK6 and lose its activity through dephosphorylation, thus negatively regulating the OsMKK10-OsMKK4-OsMPK6 cascade system, and ultimately affecting the number of secondary branches and grain size [9–11]. OsMPK6 can interact with DST and phosphorylate it, thereby enhancing the transcriptional activation ability of DST of the downstream cytokinin oxidase gene *OsCKX2*, promoting the degradation of cytokinin during the development of the young panicle, and maintaining normal levels of cytokinin. The OsER1-OsMKK10-OsMKK4-OsMPK6 signaling pathway is genetically dependent on the DST-OsCKX2 regulatory module, affecting rice panicle development by maintaining homeostasis of cytokinin, and ultimately determines the formation of grains [8].

DENSE AND ERECT PANICLE1 (*DEP1*) encodes the G protein γ subunit, a major locus regulating rice panicle architecture and panicle branch number [12]. Its dominant allele *dep1-D* downregulates *OsCKX2* and leads to a dense and erect panicle with shortened inflorescence internodes and increased primary branch number. Genes like *ERECT PANICLE 2* (*EP2*)/*DEP2*, *EP3/LARGER PANICLE* (*LP*), and *DEP3*, showing similar function with *DEP1*, have also been cloned successively [13–17]. A recent study shows that *DEP1* is a direct target of IPA1 (also known as Wealthy Farmer's Panicle, WFP) [18]. IPA1/WFP encodes a SQUAMOSA promoter binding protein-like (SBP) box protein *OsSPL14* and has a target site of *OsmiR156* in its coding region. A point mutation in the *OsmiR156* targeting site within *OsSPL14* would disturb the negative effect of *OsmiR156* on its expression, leading to the increase of *OsSPL14* expression, promoting primary branching, and repressing tiller formation [19–20]. The transcriptional activation activity of IPA1 was repressed by SHORT INTERNODES1 (*OsSHI1*) via physical

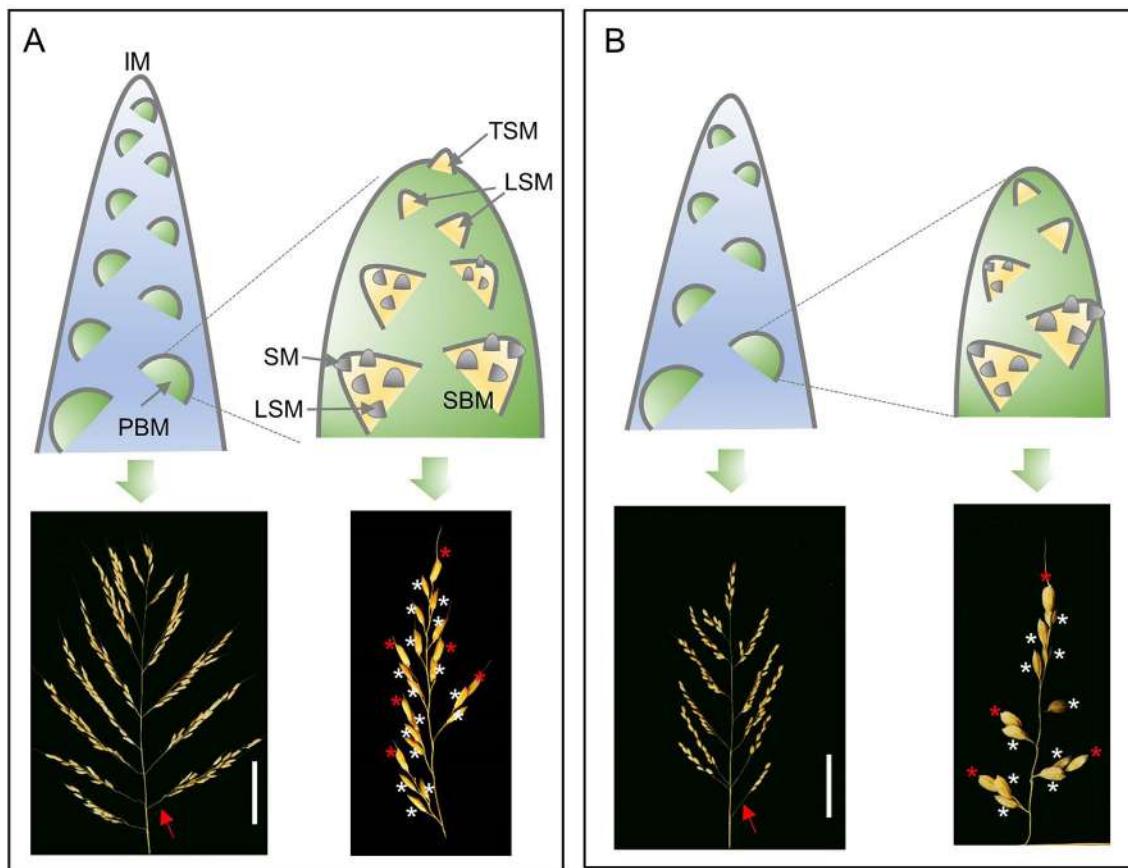


Fig. 2. Schematic representation of panicle formation. The left (A) and right (B) panels show the development process of a panicle with respectively more and fewer grain number. The panicles of cultivars 93–11 and Nipponbare were photographed for panels A and B, respectively. Scale bar, 5 cm. IM, inflorescence meristem; PBM, primary branch meristem; SBM, secondary branch meristem; SM, spikelet meristem; LSM, lateral spikelet meristem; TSM, terminal spikelet meristem. Red arrow indicates the primary branch. Red and white asterisks represent respectively terminal and lateral spikelets.

interaction [21]. Another protein, ovarian tumor domain-containing ubiquitin aldehyde-binding protein 1 (OTUB1), can interact with IPA1 and promote its K48Ub-dependent proteasomal degradation [22]. Loss of function of *OsSHI1* or *OsOTUB1* leads to an increase in primary branch number. *IPA1 INTERACTING PROTEIN 1* (*IPI1*), which encodes a RING-finger E3 ligase, can interact with IPA1. IPI degrades IPA1 in panicles while stabilizes it in shoot apexes. The loss-of-function mutant *ipi1* displayed increased shoot and panicle branches [23]. *UNBRANCHED3* (*UB3*) is an ortholog of *OsSPL14* in maize. Overexpression of *UB3* in rice dramatically suppressed tillering and panicle branching as a result of a sharp decrease in active cytokinin level. These results suggest that *OsSPL14* regulates rice shoot and panicle branching by regulating cytokinin biosynthesis and signaling [24]. *OsSPL7* and *OsSPL17* both show a similar genetic effect on rice panicle development to *OsSPL14/ IPA1* [25,26]. A NAC (for NAM, ATAF1/2, and CUC2) transcription factor *OsNAC2* may control primary branches via an IPA1-DEP1-related pathway, as the expression levels of *IPA1* and *DEP1* were upregulated in *OsNAC2*-overexpressing plants. Overexpression of *OsmiR164b*-resistant *OsNAC2* increases both primary and secondary branches, increasing rice yield [27].

FRIZZY PANICLE (*FZP*), encodes a transcription factor with ERF structure and regulates rice panicle morphogenesis. Its function includes negative regulation of axillary meristem production and positive promotion of floret meristem establishment [28]. An 18-bp insertion at 5.3 kb upstream of *FZP* resulted in enhancement inhibition of *OsBZR1* on its expression and reduced *FZP* expression, leading to an increase of secondary branching in rice panicles [29].

FZP expression can also be repressed by overexpression of *MOTHER OF FT AND TFL1* (*OsMFT1*) and thus produce more secondary branches [30]. *NARROW LEAF 1* (*NAL1*) promoted *FZP* degradation by physical interaction [31]. *NAL1/LSCHL4/SPIKE* encodes a serine/-cysteine protease, the first *japonica* allele found to increase the number of grains per panicle in *indica* rice. The *LSCHL4/SPIKE* allele of *japonica* rice can better balance the trade-off between sink and source, leading to increased secondary branch number and rice yield [32,33].

TAWAWA1 (*TAW1*), which encodes an ALOG (*Arabidopsis LSH1* and *Oryza G1*) transcription factor containing a conserved domain and a nuclear localization signal, is an essential regulatory factor of rice inflorescence morphology. *TAW1* regulates the number of secondary branches by promoting IM activity and suppressing of the phase change to SM identity. Reduction in *TAW1* activity leads to precocious IM abortion and reduced secondary branch number, whereas the phenotype of the dominant gain-of-function mutant *tawawa1-D*, is just the opposite [34]. A novel functional chimeric gene *GRAINS NUMBER 2* (*GN2*), originated from the insertion of a 1094-bp fragment from *LOC_Os02g45150* into the coding region of *LOC_Os02g56630*, influences secondary branch number by increasing the expression level of *TAW1* [35]. The expression of *TAW1* may also be controlled by the *OsmiR396b-Growth Regulating Factor 6* (*OsGRF6*) module. Blocking *OsmiR396* or overexpressing *GRF6* increases rice yield by increasing panicle branching [36].

In rice, lax panicle has a distinct feature that all lateral grains are absent from the secondary branch. To date, three genes have been found responsible for the generation of lateral spikelets on

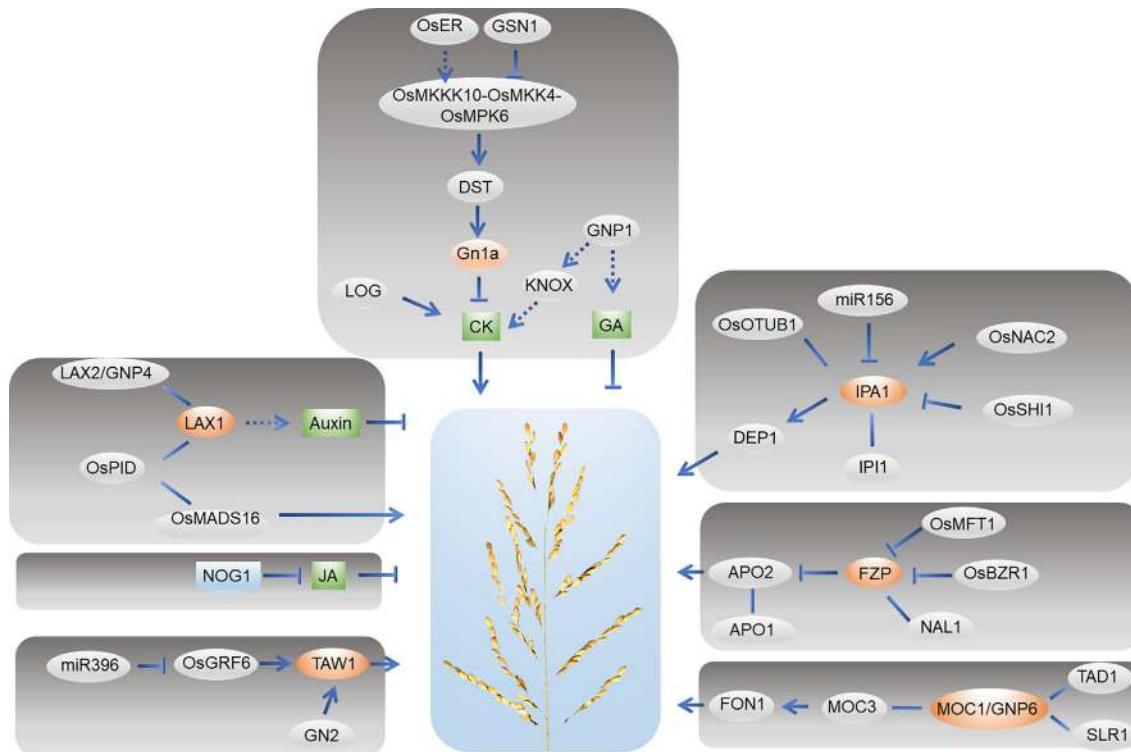


Fig. 3. A schematic representation of regulatory pathways controlling panicle branch and spikelet formation in rice. The core regulators are indicated by orange ellipses and four plant hormone-associated pathways are indicated by green boxes. Solid lines indicate known protein–protein interactions; arrows and blocked arrows indicate positive and negative regulation of transcription. Dotted lines or arrows indicate indirect regulation. JA, jasmonic acid; GA, gibberellic acid; CK, cytokinin.

secondary branches. *LAX1*, an ortholog of maize *BARREN STALK1* (*BA1*) and *Arabidopsis REGULATOR OF AXILLARY MERISTEM FORMATION* (*ROX*), encodes a plant-specific basic helix-loop-helix (bHLH) transcription factor. *lax1* mutant plants have severely reduced branches, especially the lateral spikelets on secondary branches [37]. *LAX1* transcripts are specifically enriched in the junction between initiating AM and SAM, then *LAX1* protein is trafficked toward the initiating AM and promotes cell proliferation [38]. *LAX PANICLE 2* (*LAX2*, also known as *GNP4*) encodes a nuclear protein containing a RING-finger and WD40-associated ubiquitin-like (RAWUL) domain shows a similar function to *LAX1*. Loss of function of *LAX2/GNP4* leads to a phenotype resembling the *lax1* mutant phenotype, including the absence of lateral spikelets. The phenotype of the *lax1lax2* double mutant is more severe, indicating that *LAX1* and *LAX2* play important and similar roles in regulating secondary branch and lateral spikelet formation [39–41]. A recent study showed that *OsPID* also interacts with *OsMADS16* and/or *LAX1* to regulate floral organ development by modulating auxin polar transport in rice [42].

MOC1 (also known as *GNP6*) encodes a GRAS-family transcription factor controlling tiller and panicle branch formation. Several alleles of *MOC1/GNP6* were isolated using different mutants: *moc1-1*, *moc1-3*, *moc1-5*, and *moc1-6/gnp6*, all showing a defect in panicle branch formation [37–39,43,44]. *FON1* and *FON2/4* encode leucine-rich repeat receptor kinases and are orthologous to *Arabidopsis CLAVATA1* (*CLV1*) and *CLAVATA3* (*CLV3*), respectively. Both *fon1* and *fon4* mutants displayed enlarged inflorescence and floral meristems and increased numbers of both primary branches and floral organs [45,46]. *MOC1* functions as a co-activator of *MOC3*, activating the expression of *FON1*; however, the direct target of *MOC1* has not yet been identified [47]. Physical interaction between *SLR1* and *MOC1* suppresses its degradation at the post-transcriptional level; however, physical interaction between *TAD1/TE* with *MOC1* promotes its degradation [48–50]. Both the

moc1 lax1 and *moc1 lax2* (*gnp4*) double mutants display only a single tiller, indicating that *MOC1* is essential for tiller formation [39].

In the above regulatory pathways, *Gn1a*- and *LAX1*-associated pathways are involved in cytokinin and auxin, respectively. Gibberellin (GA) and jasmonic acid (JA) may also be involved in rice branch development. *Grain Number per Panicle1* (*GNP1*) encodes a GA₂₀ oxidase. The increased *GNP1* transcript levels in the rice inflorescence meristem results in the increased expression of *KNOX* gene and increases cytokinin activity via *KNOX* mediated feedback regulation, thus affecting the activity of meristem and finally leading to the increase of the secondary branches and the number of grains per panicle [51]. A recent study showed that the combination of *NAL1* and *GNP1* increased grain yield in *japonica* rice cultivars [52]. *NUMBER OF GRAINS 1* (*NOG1*), which encodes an enoyl-CoA hydratase/isomerase, increases panicle branching (especially secondary branches) without a negative effect on panicle number or grain weight, and thus leads to increased grain yield. *NOG1* may be involved in the JA pathway, given that upregulated *NOG1* expression reduced endogenous JA levels and JA treatment downregulated *NOG1* gene expression [53].

3. Multifloret spikelets

Generally, the inflorescence of grasses is composed of branches and spikelets [54,55]. The spikelet, comprising a pair of bracts and one or multiple florets, is a grass-specific primary inflorescence unit. The number of florets in a spikelet varies with the species of grass. In rice, one spikelet harbors a fertile floret and a pair of sterile lemmas (also known as ‘empty glumes’) [56]. Wild-type rice produces only one grain per spikelet, as the spikelet has only one fertile floret. The origin of sterile lemmas is controversial. One hypothesis ‘rice three-floret spikelet’, suggests that the sterile lemmas are originated from the degeneration of the lemma of lateral

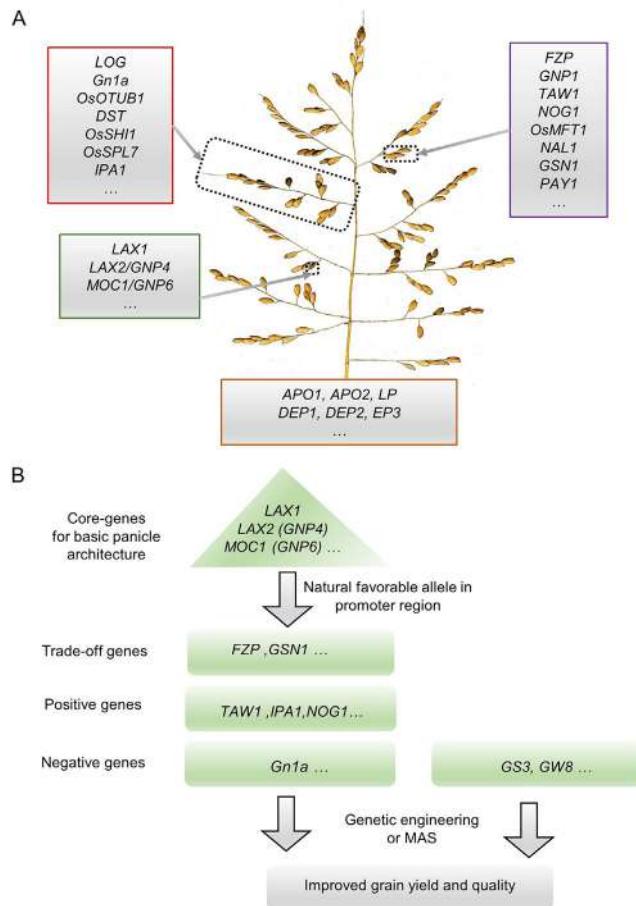


Fig. 4. Summary of key genes that determine rice panicle branching. In the upper panel (A), genes influencing grain number per panicle are identified according to the characters they mainly affect. Names of genes influencing primary branch number are enclosed in the red box; those influencing secondary branch number in the purple box, those influencing the lateral spikelet in the green box, and those influencing panicle type in the orange box. The lower panel (B) shows a proposed strategy for increasing rice grain number and quality. The favorable alleles of core genes containing natural variation in the promoter region should first be selected to maintain the basic backbone of the rice panicle, after which trade-off genes could be used to balance the grain size and number, and lastly favorable alleles for grain number and grain size could be pyramided using MAS or genetic engineering to maximize grain yield and quality.

florets, has been confirmed, with supporting evidence continuing to be reported in recent years [57].

ASP1 acts as an essential regulator of panicle branches and also influences sterile lemmas identity. In the *asp1* mutant, the sterile lemmas identities are altered and turn into a lemma-like sterile lemma. The *asp1* mutant phenotype indicates that *ASP1* can prevent the transformation of the sterile lemma to a lemma [58,59]. *LONG STERILE LEMMA* (*G1*), *EXTRA GLUME 1* (*EG1*), *PANICLE PHYTO-MER 2* (*PAP2*)/*OsMADS34*, and *FLORAL ORGAN NUMBER 4* (*FON4*) are similar to *ASP1* in the regulation of sterile lemma identity [60–63]. The expression of *G1* is positively regulated by *MULTI-FLORET SPIKELET1* (*MFS1*). *MFS1* encodes a nuclear protein of the APETALA2/ethylene-responsive factor (AP2/ERF) family, which plays an important role in floral organ identity. In *mfs1* mutant, the sterile lemma was homeostatically transformed into a rudimentary glume [64]. The other two AP2/ERF domain proteins, *SUPERNUMERARY BRACT* (*SNB*) and *OsIDS1*, were also shown to prevent the sterile lemma's degeneration into a rudimentary glume [65,66]. In maize (*Zea mays*), loss of *INDETERMINATE SPIKELET 1* (*IDS1*) function results in more than two florets in one spikelet

[67]. These genes are positive evidence for the rice three-floret spikelet hypothesis. However, they cannot be used in breeding, as none of these mutants develops fertile lateral florets. A recent study reported that a single-nucleotide substitution in *EG1* leads the spikelets to produce two fertile florets [68]. This newly discovered allele of *EG1* suggests that genes with similar functions as *EG1* may potentially increase floret number per spikelet in rice. *LATERAL FLORET 1* (*LF1*) encodes the rice homeodomain leucine zipper class III

(HD-ZIP III) transcription factor *OsHB1*, responsible for the transformation of a sterile lemma to a fertile lateral floret. In the gain-of-function mutant *lf1*, the spikelet had one fertile terminal floret and one or two fertile lateral florets with proper floral organ identities in the axil of the sterile lemma. The elevated expression of *LF1* in the *lf1* mutant, which is caused by a single-nucleotide substitution in the target site for *OsmiRNA165/166* in the *LF1* gene, results in ectopic expression of the meristem maintenance gene *OSH1* and then induces lateral florets formation in the axils of the sterile lemmas. *LF1* not only provides strong evidence for the three-floret spikelet hypothesis but also provides an efficient way to increase grain number per panicle by breeding rice cultivars with three-floret spikelets [69].

4. Panicle type

In rice, many genes affect not only the number of branches but also the arrangement of branches and spikelets. Mutations in these genes may lead to large changes in panicle type. The *dep1* mutant also exhibits a dense and erect panicle type with shortened inflorescence internodes and an increased number of primary branches [12]. Owing to the continuous generation of secondary branch meristems where the spikelet formed initially, the *fzp* mutant phenotype forms a fuzzy panicle [28]. Mutation in *SPED1*, which encodes a pentatricopeptide repeat protein, can result in a clustered-spikelet panicle owing to the shortened pedicels and secondary branches [70]. *Aberrant Panicle Organization 1* (*APO1*), the ortholog of *Arabidopsis UNUSUAL FLORAL ORGAN* (*UFO*), encodes an F-box protein and plays a key role in the temporal regulation of meristem fate. The *apo1* mutant exhibits aberrant panicle type with a reduced number of primary branches and spikelets [71]. *SCM2* is a new allele of *APO1*, increasing stem thickness and effective panicle number [72]. *ABERRANT PANICLE ORGANIZATION 2/RFL*, the rice ortholog of *Arabidopsis LEAFY*, shows a similar function to *APO1*, as the *apo2* mutant also exhibits aberrant panicle type with a reduced number of primary branches [73]. In rice, *APO2/RFL* regulates the transition from inflorescence meristem to spikelet meristem to control branch number and particularly influences heading date and tiller number. *APO2/RFL* interacts with *APO1* at the protein level to control inflorescence and flower development. Further analysis indicated that *APO1* function depends on *APO2/RFL*, as overexpression of *APO1* in the *apo2-1* background did not rescue the *apo2-1* panicle [73]. *APO2/RFL* expression is positively regulated by a transcriptional activator *Short Panicle3* (*SP3*) [74]. The *sp3* mutant had a short panicle with markedly reduced secondary branches. Unlike the *sp3* mutant, the *short panicle1* (*sp1*) mutant exhibits the short-panicle phenotype with extremely reduced primary branch number and length [75]. *SP1* encodes a putative peptide transporter with transmembrane domains. *ABERRANT SPIKELET AND PANICLE1* (*ASP1*)/*OsREL2* encoding a WD40 protein, which is homologous to *TPL/TPR* in *Arabidopsis* and *REL2* gene in maize. The *asp1/osrel2* mutant shows a disorganized branching pattern with an increased number of primary branches and reduced number of secondary branches [58,59]. A recent study indicates that *ASP1* is a genetic enhancer of *FLORAL ORGAN NUMBER 2/4* (*FON2/4*) in regulating stem cell maintenance [76]. *PAP2/*

Table 1

Cloned genes influencing grain number per panicle in rice.

| Function | Gene name | Protein | Accession number | References |
|----------------------|--------------------|---|------------------|--|
| Primary branch | <i>Gn1a</i> | Cytokinin oxidase | LOC_Os01g10110 | Ashikari et al. [5] |
| | <i>LOG</i> | Cytokinin activating enzyme | LOC_Os01g40630 | Kurakawa et al. [6] |
| | <i>DST</i> | Zinc finger protein | LOC_Os03g57240 | Li et al. [7] |
| | <i>IPA1/WFP</i> | SPL transcription factor | LOC_Os08g39890 | Jiao et al. [19], Miura et al. [20] |
| | <i>OSSH1</i> | SHI family transcription factor | LOC_Os09g36160 | Duan et al. [21] |
| | <i>OsOTUB1</i> | Human OTUB1-like deubiquitinating enzyme | LOC_Os08g42540 | Wang et al. [22] |
| | <i>OsSPL7</i> | SPL transcription factor | LOC_Os04g46580 | Dai et al. [25] |
| | <i>OsSPL17</i> | SPL transcription factor | LOC_Os09g31438 | Wang et al. [26] |
| | <i>OsNAC2</i> | NAC transcription factor | LOC_Os04g38720 | Jiang et al. [27] |
| | <i>FON2/4</i> | Leucine-rich repeat receptor kinase | LOC_Os11g38270 | Chu et al. [73] |
| Secondary branch | <i>FON1</i> | Leucine-rich repeat receptor kinase | LOC_Os06g50340 | Suzaki et al. [74] |
| | <i>FZP</i> | ERF transcription factor | LOC_Os07g47330 | Bai et al. [28], Huang et al. [29] Komatsu et al. [31] |
| | <i>OsMFT1</i> | Phosphatidylethanolamine-binding proteins | LOC_Os06g30370 | Song et al. [30] |
| | <i>NAL1/SPIKE</i> | Serine / cysteine protease | LOC_Os04g52479 | Fujita et al. [32], Zhang et al. [33] |
| | <i>GNP1</i> | GA20 oxidase | LOC_Os03g63970 | Wu et al. [49] |
| | <i>TAW1</i> | ALOG transcription factor | LOC_Os10g33780 | Yoshida et al. [34] |
| | <i>GN2</i> | OsWAK receptor-like protein kinase | LOC_Os02g56630 | Chen et al. [35] |
| | <i>OsGRF6</i> | Growth-regulating factor | LOC_Os03g51970 | Gao et al. [36] |
| | <i>GSN1</i> | MAPK phosphatase | LOC_Os05g02500 | Guo et al. [10] |
| | <i>GLW7</i> | SPL transcription factor | LOC_Os07g32170 | Si et al. [82] |
| Lateral spikelets | <i>NOG1</i> | Enoyl-CoA hydratase/isomerase | LOC_Os01g54860 | Huo et al. [51] |
| | <i>LAX1</i> | bHLH transcription factor | LOC_Os01g61480 | Komatsu et al. [37] |
| | <i>LAX2/GNP4</i> | RAWUL domain protein | LOC_Os04g32510 | Tabuchi et al. [39], Zhang et al. [41] |
| Multifloret spikelet | <i>MOC1/GNP6</i> | GRAS family transcription factor | LOC_Os06g40780 | Li et al. [43], Zhang et al. [44] |
| | <i>G1</i> | ALOG transcription factor | LOC_Os07g04670 | Yoshida et al. [61] |
| | <i>EG1</i> | Putative lipase | LOC_Os01g67430 | Li et al. [58], Ren et al. [66] |
| | <i>PAP2</i> | MADS-box protein | LOC_Os03g54170 | Lin et al. [59] |
| | <i>MFS1</i> | ERF transcription factor | LOC_Os05g41760 | Ren et al. [62] |
| | <i>SNB</i> | ERF transcription factor | LOC_Os07g13170 | Lee et al. [64] |
| | <i>OsIDS1</i> | ERF transcription factor | LOC_Os03g60430 | Lee et al. [63] |
| | <i>LF1</i> | HD-ZIP III transcription factor | LOC_Os03g01890 | Zhang et al. [67] |
| | <i>DEP1</i> | Atypical Gy subunit | LOC_Os09g26999 | Huang et al. [12] |
| | <i>SPED1</i> | PPR protein | LOC_Os06g39650 | Jiang et al. [68] |
| Panicle type | <i>APO1</i> | F-box protein | LOC_Os06g45460 | Ikeda et al. [69] |
| | <i>APO2/RFL</i> | Homolog of LFY | LOC_Os04g51000 | Ikeda-Kawakatsu et al. [71] |
| | <i>SP1</i> | Putative peptide transporter | LOC_Os11g12740 | Li et al. [73] |
| | <i>ASP1/OsREL2</i> | WD40 protein | LOC_Os08g06480 | Yoshida et al. [57] |
| | <i>EP2/DEP2</i> | Plant-specific protein | LOC_Os07g42410 | Li et al. [13], Zhu et al. [17] |
| | <i>EP3/LP</i> | F-box protein | LOC_Os02g15950 | Li et al. [14], Piao et al. [15] |
| | <i>DEP3</i> | PLA2 domain-containing protein | LOC_Os06g46350 | Qiao et al. [16] |

OsMADS34 encodes a MADS-box protein that belongs to the SEPALLATA (SEP) subfamily. The *pap2* mutant produces more rachis branches, as early-arising spikelet meristems are transformed into rachis branch meristems [77].

5. Utilization and perspectives

The practice of rice breeding has shown that increasing grain number per panicle is the most effective way to increase rice yield. In the past decades, there has been great progress in researching number per panicle in rice. Several genes influencing grain number per panicle have been identified, and these genes control one or more panicle characters (Fig. 4A; Table 1). These genes influencing grain number per panicle can be used as potential resources for breeders.

Marker-assisted selection (MAS) has been employed as an effective tool for gene pyramiding in the past years [78]. *Gn1a*-type 3 and *OsSPL14^{WFP}* alleles were pyramided by repetitive backcrossing with MAS. The pyramiding lines showed increased grain number per panicle [79]. The pyramiding of *GNP1* and *NAL1* could increase rice yield by increasing grain number per panicle [52]. Compared with Kongyu 131, the yield of new Kongyu 131 containing the *Gn1a* allele from a large-panicle *indica* rice cultivar GKBR was increased [80]. It should be noted that only some of the genes could be used in MAS, even though dozens of genes have been isolated. There are still challenges in applying these genes in breeding,

as some genes exert undesirable effects in rice. *Ghd7* delays flowering time, *FZP* reduces grain size, *IPA1* reduces tiller number, and excessive overexpression of *IPA1* leads to reductions in both tiller number and panicle branching [26]. There is also a negative correlation between grain number and size [81]. This phenomenon can be observed in rice, as *lax* panicle mutants such as *lax1*, *lax2* (*gnp4*), and *moc1* (*gnp6*) usually produce large grains. The coding sequence region of these genes is usually conserved both in wild and cultivated rice, implying their indispensable role in maintaining the basic panicle architecture, so that these may be considered core genes essential for panicle development (Fig. 4B). The identification of new alleles of core genes with variations in the promoter region could be used for precisely controlling their expression and they could then be used in gene pyramiding.

How to balance these traits with grain number per panicle awaits further study. More detailed information about the regulatory networks underlying these genes will facilitate the elimination of unfavorable effects. *IPA1* suppresses rice tillering mainly by positive regulation of a negative regulator of rice tillering *Ostb1* [18,82]. Thus, it is feasible to mutate the *IPA1* binding sites in the *Ostb1* promoter region by CRISPR/Cas9 or to restrict *IPA1* expression in rice inflorescence using a tissue-specific promoter, thereby eliminating the effect of *IPA1* on rice tillering. In addition, natural variation of these genes should be characterized. In a recent study, *qWS8/ipa1-2D*, a new allele of *IPA1* caused by a tandem repeat upstream of *IPA1*, having a weak effect on reducing tiller number caused by tissue-specific up-regulation of *IPA1*, was identified

[83]. Genes responsible for coordinating the trade-off between grain number and grain size in rice have been cloned, such as *FZP* and *GSN1*. Tuning the expression of these genes to maximize grain yield is a challenge. Some genes increase grain number without reducing grain size; *NOG1* increases grain number without any adverse effect on grain weight [53]. There also exist genes such as *OsSPL13/GLW7* that can increase grain number and grain size simultaneously [84]. Further identification of genes with similar functions to *NOG1* or *OsSPL13/GLW7* will ultimately benefit rice breeding.

In recent years, a rational design approach based on the extensive accumulated information about genes governing agronomic traits (including grain number) has been proposed and put into practice [85,86]. Tailored rice with superior haplotypes for grain yield and quality have also been proposed, including *DEP3-H2*, *DEP1-H2*, and *SP1-H3* for long panicles and *LAX1-H5*, *OSH1-H4*, and *LP-H13* for increased panicle branching [87]. Rational design for yield and quality improvement faces two tasks: discovery of favorable genes or alleles in rice germplasm and their evaluation in diverse genetic backgrounds, and characterization of the genetic interactions between these favorable alleles and their pyramided effect on grain yield by MAS or genetic engineering (Fig. 4B).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Gangling Li wrote the manuscript, **Hongliang Zhang and Jinjie Li** revised the manuscript, **Zhanying Zhang** wrote and revised the manuscript, **Zichao Li** conceived the project.

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