miR156f integrates panicle architecture through genetic modulation of branch number and pedicel length pathways

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Abstract

Background: Rice (Oryza sativa) panicle architecture is the major determinant of the ideal plant architecture that directly influence yield potential. Many genes influencing development of primary branches, secondary branches, spikelet and pedicel would also influence panicle architecture, which is thus a complex trait regulated by genes from various aspects. miR156, an extensively studied miRNA, has recently emerged as promising target for crop improvement because of its role in plant architecture regulation, such as the number of tillers, plant height and the panicle architecture. Increasing evidence suggests that miR156 might play an important role in panicle architecture regulation.

Main body: To study the detailed function of miR156 in rice panicle architecture regulation, we examined the genetic interaction or transcriptional regulation of miR156/OsSPL to other panicle regulating genes. Our results revealed that expression of many panicle related genes were influenced by miR156. Through biochemical analysis, we further proved that miR156 directly regulated the axillary meristem regulating gene, LAX1, at the transcription level. And the intimate relations between miR156 and LAX1, and miR156 and LAX2 were also uncovered by genetic analysis. On the other hand, a tight genetic linkage between miR156 and RCN2, the panicle branch promoting gene, was also detected, which suggested a buffering mechanism for the miR156 mediated panicle architecture regulation. Furthermore, genetic analysis also demonstrated that miR156 functioned in the same pathway with OsRA2 to regulate pedicel length.

Short conclusion: Altogether, miR156 integrates several genetic pathways mediated by genes such as LAX1, LAX2, RCN2 and OsRA2, and comprehensively regulates panicle development in rice. Based on these analysis, we concluded that miR156 acts as an important regulator for panicle architecture through influencing various aspects of panicle development.

Keywords: Oryza sativa, miR156, Panicle development, LAX1, LAX2, RCN2, OsRA2

Background

As one of the most important cereal crops, rice (Oryza sativa) provides food for more than half of the world population. And still greater challenge would be faced to meet the need of the increasing population under condition such as shrunk arable land, changing climate and less water (Wang et al. 2018). Increasing world population calls for high yield from crop plants, therefore, factors that affect rice yield always attract the major research attention. Panicle architecture (inflorescence patterning) is one of the specific morphological characters of rice, which comprehensively coordinates panicle length, the primary branches (PB) and secondary branches (SB), the spikelet, and pedicel length (Itoh et al. 2005). In agriculture, panicle architecture is the major determinant of the ideal plant architecture that directly influence rice yield potential. Therefore, many studies have focused on the genes associated with the panicle development and the underlying mechanism.

Panicle architecture is mainly determined by the timing of identity transition of different type of meristems...
microRNAs (miRNAs) have emerged as a new force in regulating plant development and physiology, increasing evidence suggests them to be coordinated integrator of complex traits, with the potential in crop improvement (Tang and Chu 2017; Wang et al. 2015). Ever since the clarification of its role in regulating developmental timing, miR156 and its targets are extensively studied (Wu et al. 2009). In rice, the miR156/SQUAMOSA PROMOTER BINDING PROTEIN-like (SPL) module proves to be good target for crop improvement for its role in plant architecture regulation, such as the number of tillers, plant height and the panicle architecture (Wang and Wang 2015; Wang and Zhang 2017). Specifically, OsSPL14 positively regulates the number of PBs in the panicle (Jiao et al. 2010; Miura et al. 2010). And generally, miR156 regulates plant height and tiller number in rice (Dai et al. 2018; Xie et al. 2006). In regulating panicle development, miR156 is revealed to regulate the coordinated development of the branching in vegetative and reproductive stage, together with several other miRNAs and factors (Wang et al. 2015). However, since panicle development is a complex processes involving multiple pathways and many regulating factors with the underlying crosstalk less revealed, still many genetic relations needs to be studied to further understand the genetic regulation of panicle development. Although miR156 is a vegetative specific factor, the target genes of miR156, OsSPLs, function in the reproductive stage, indicating the possible function of miR156 in panicle traits determination that remains to be further revealed.

In this study, we proved that miR156 regulated panicle development, with the cs mutant plants in which miR156f was over expressed showing small panicle, and MIM156fOE plants in which miR156 was down-regulated showing increased panicle length and decreased number of SBs, and expression of many genes functioned in panicle regulation was influenced by miR156. In genetic analysis, we revealed the possible relation between miR156 and LAX1, LAX2, RCN2 and OsRA2. Through these analysis, we investigated the possible pathways through which miR156 regulated auxiliary development, the number of branches, and pedicel development in the panicle.

**Main text**

Sequestering miR156 resulted in sparse panicle with less SBs and longer pedicel

In our previous study, it was proved that miR156f regulated plant height and tiller number in rice through the auxin signaling pathway (Dai et al. 2018). Furthermore, we found that miR156f also had effects on the panicle morphology. Specifically, the constitutively expressed
MIM156f in the MIM156fOE transgenic plants, sequestered the native miR156, and induced sparse panicles (Fig. 1a). The panicle architecture related characteristics in the MIM156fOE plants such as the panicle was longer, the number of the SBs but not the PBs was decreased, as compared with those in the wild type (WT) plants (Fig. 1a, b, c). These changes on the panicle indicated that the transition from SBs to spikelets was affected when miR156 was down-regulated. In addition, the pedicels of the MIM156fOE plants were also elongated as compared with that of the WT (Fig. 1d). Altogether, these morphological features of the panicle changes made the panicle in the MIM156fOE plants a sparse phenotype.

Further, we had isolated a T-DNA insertion mutant named cs mutant, in which the expression of miR156f was significantly up-regulated (Dai et al. 2018). The cs mutant had a smaller and shortened panicle as compared with the WT (Fig. 1b, e), accompanied with less PBs and SBs (Fig. 1c), these characters are in consistence with the significantly decreased plant height of the cs mutant (Dai et al. 2018). In contrast to the panicle changes in the mutant, the pedicel length showed no significant difference as compared with that of the WT plants (data not shown).

In summary, changes of the morphological features of in the panicle of the MIM156fOE plants and the cs mutant indicated that although miR156 is extensively studied as a vegetative factor, it was also involved in panicle architecture regulation in rice.

miR156f regulated the expression level of genes related to panicle development

We have shown that miR156f played a role in panicle development. Further, we wanted to know if its function

![Figure 1](https://example.com/fig1.png)
in the panicle was mediated by regulation on the panicle related genes. The microarray data of the cs mutant (Dai et al. 2018) revealed that the expression level of several panicle development related genes, such as LAX2, RCN1, RCN3 and OsCKX2 was greatly changed compared with WT (Table 1).

The expression level of selected panicle development related genes in the MIM156fOE plants and the cs mutant was analyzed by quantitative real-time PCR (qRT-PCR). Genes detected included those involved in axillary meristems development, such as LAX1(Oikawa and Kyozuka 2009; Komatsu et al. 2003) and LAX2 (Tabuchi et al. 2011), pedicel length regulation, such as OsRA2 (Lu et al. 2017), those regulating PB and SB development, such as RCN1, RCN2, RCN3, RCN4 (Nakagawa et al. 2002), GHD8 (Yan et al. 2011) and MOC1 (Li et al. 2003), and those in spikelet development, such as MFS1 and OsIDS1 (Ren et al. 2013; Lee and An 2012). It was revealed that the expression level of these selected genes was significantly changed by miR156f over expression (the cs mutant) and/or miR156 down regulation (the MIM156fOE plants) when compared with WT plants (Fig. 1f). This analysis further indicated the possible involvement of miR156f in panicle development.

**Genetic analysis revealed interaction between miR156f and LAX1**

In rice, LAX1 gene is responsible for AM initiation and maintenance. The lax1 mutant showed reduced high-order branches and spikelets, which was due to the defects in initiation or maintenance of the lateral panicle development (Komatsu et al. 2003). To investigate the potential interaction between miR156f and LAX1, we crossed the MIM156fOE plant with the lax1 mutant, a natural genetic mutant in the ZH11 background. The positive F2 hybrid showed sparser panicle, much less number of SBs and sterile spikelets (Fig. 2d) compared with the parents (Fig. 2b, c) and the WT (Fig. 2a), but no change of the number of PBs. This synergistically enhancement on the panicle defects indicated the possible genetic interaction between miR156f and LAX1.

**SPL protein could directly regulate LAX1 expression at the transcription level**

The genetic analysis had confirmed the interaction between miR156f and LAX1 in panicle development, and then we wanted to test whether LAX1 could be regulated by miR156 at the transcriptional level. Firstly, the expression level of LAX1 in the MIM156fOE plants and the cs mutant was analyzed. It showed that the expression of LAX1 was down-regulated in the MIM156fOE plants, while up-regulated in the cs mutant (Fig. 1f). This confirmed that LAX1 gene was affected by miR156 at the transcriptional level.

miRNAs function through negatively regulating their targets SPL genes. In previous study, we had showed that the target gene, OsSPL7, mediated miR156f’s regulation on rice plant architecture (Dai et al. 2018). When the fusion protein of OsSPL7 and myc tag was over expressed, the transgenic plants showed phenotype in panicle architecture, with shorter panicle (Fig. 3a, b), more PBs and less SBs (Fig. 3c). Then, we analyzed the expression level of LAX1 in the OsSPL7 over expressed SPL7Flag plants, and RNAi plants to see whether LAX1 expression was also regulated by OsSPL7 (Dai et al. 2018). Consistent with the results in miR156f transgenic plants (Fig. 1f), LAX1 gene was down-regulated in the SPL7Flag plants, while up-regulated in the SPL7RNAi plants (Fig. 4a). These data demonstrated that OsSPL7 gene might also mediated the function of miR156f in regulating LAX1 expression.

To investigate how OsSPL7 regulated LAX1 gene expression at the transcriptional level, the promoter sequence of LAX1 gene was analyzed by searching the database of Plant Cis-acting Regulatory DNA Elements, PLACE, (http://www.dna.affrc.go.jp/PLACE/) to look for the potential SPL binding motifs. In the 3 Kb sequence upstream of the “atg” start codon of LAX1 gene, there are 19 “GTAC” motifs for potential SPL binding (Fig. 4b). We then checked the binding affinity of the OsSPL7 protein to the 18th and 19th motifs using a yeast one hybrid (Y1H) assay. When two copies of both 18th and 19th motifs were used (“2(18 + 19)” in Fig. 4c), the yeast clones turned sharp blue, when two copies of mutant 18th and normal 19th motifs were used (“2(m18 + 19)” in Fig. 4c), the clones turned faint blue; when one copy of mutant 18th and mutant 19th motif, and one copy of normal 18th and 19th motif were used (“(18 + 19) + (m18 + m19)” in

| Table 1 Expression of some panicle related genes in the microarray data of the cs mutant |
|---------------------------------|---------------------------------|
| **Gene names and ID** | **Log2 fold change (+"indicates up-regulation; "_" indicates down-regulation)** |
| LOC_Os06g40780(MOC1) | -1.5 |
| LOC_Os01g61480(LAX1) | +1.0 |
| LOC_Os04g32510(LAX2) | -2.0 |
| LOC_Os11g05470(RCN1) | +7.5 |
| LOC_Os12g05590(RCN3) | +4.7 |
| LOC_Os04g33570(RCN4) | -1.2 |
| LOC_Os01g07480(OsRA2) | +1.3 |
| LOC_Os08g39890(OsSPL14) | +1.3 |
| LOC_Os03g06430(OsDS1) | -1.7 |
| LOC_Os05s41760(MFS1) | -1.4 |
| LOC_Os08g07740(GHD8) | +1.1 |
| LOC_Os01g10110(OsCKX2) | +2.6 |
Fig. 4c), the clones were also faint blue, only when two copies of both mutant 18th and mutant 19th motifs were used (“2(m18 + m19)” in Fig. 4c), the clones were not blue any more. Altogether, the Y1H assay demonstrated that the OsSPL7 protein could bind to the 18th and 19th “GTAC” motifs in the LAX1 promoter region. In addition to OsSPL7, OsSPL13 protein also showed binding ability to the same LAX1 promoter region in the Y1H assay (Fig. 4c). These direct evidences showed that both miR156f targets, OsSPL7 and OsSPL13, could bind to the promoter region of LAX1 promoter.

Next, we carried out chromatin immunoprecipitation (ChIP) assay with the transgenic SPL7::SPL7Flag plants, in which OsSPL7 protein was fused with Flag tag and driven by its own promoter (Dai et al. 2018). It was revealed that the DNA fragments of the LAX1 promoter region were pulled-down by the OsSPL7Flag protein (Fig. 4d). It further confirmed the direct binding of OsSPL7 to the promoter region of LAX1 gene.

Genetic analysis revealed interaction between miR156f and LAX2
LAX2 gene encodes a nuclear protein that regulates axillary development, and lax2 mutant showed similar phenotype as lax1 mutant (Tabuchi et al. 2011). To investigate the genetic relation between miR156f and LAX2, we crossed the MIM156fOE plants to a lax2 mutant in ZH11 background. The positive F2 hybrid showed less SBs and spikelets (Fig. 2a, b, e, f) compared with the parents, but no change in the number of the PBs as compared with the lax2 mutant. Then, we analyzed the expression level of LAX2 gene in the cs mutant and the MIM156fOE plants.
Consistently, LAX2 gene was also down-regulated in the MIM156fOE plants and up-regulated in the cs mutant (Fig. 1f). By searching the PLACE database, we also found 10 “GTAC” motifs in the 3 Kb promoter of the LAX2 gene. However, different from the result from LAX1, when we carried out Y1H assay with LAX2 predicted binding motifs and OsSPLs proteins, there were no binding affinity detected between them (data not shown). It indicated that LAX2 was not directly regulated by OsSPL7 at the transcriptional level, although they showed genetic interaction. Further, OsSPL7 and LAX2 could directly interact with each other in the yeast two hybrid (Y2H) system but not OsSPL7 and LAX1 (Fig. 4e). In summary, it suggested the direct regulation of OsSPL7 to LAX1, but not LAX2.

Genetic analysis revealed interaction between miR156f and RCN2

Over expression of several RCN genes could greatly increase the number of PBs and SBs (Ikeda-Kawakatsu et al. 2012). According to the microarray data, the expression level of RCN1, RCN3 and RCN4 were significantly up-regulated in the cs mutant (Table 1). We used 2 cm young panicle to perform the qRT-PCR analysis, the expression of all four RCN genes were down-regulated in
the MIM156fOE plants while up-regulated in the cs mutant, and the up-regulation of the RCN2 and RCN4 genes was the most dramatic (Fig. 1f). It indicated that miR156f regulated the expression of RCN4 genes, through which the number of panicle branches was influenced.

In previous study, we had isolated a T-DNA insertion mutant, A989, in which RCN2 was constitutively over expressed (Li et al. 2010). A989 showed high-density panicle with significantly increased number of PBs and SBs. To investigate the genetic interaction between miR156f and RCN2 gene, we also crossed A989 with the cs mutant and the MIM156fOE plants. The cs/A989 hybrid showed longer panicle (Fig. 5a, d) and more PBs and SBs (Fig. 5c) compared with the cs mutant. In contrast, the MIM156fOE/A989 hybrid showed shorter panicle (Fig. 5b, d) and more PBs and SBs (Fig. 5c) than the MIM156fOE plants. So that, over expression of RCN2 increased the number of SBs in both the cs mutant and the MIM156fOE plants. Other than the panicle number, the plant height and tiller number of the cs/A989 hybrid were similar to the cs mutant, while the less tiller number phenotype of the MIM156fOE plant was maintained in the MIM156fOE/A989 plants (Additional file 1: Figure S1).

Genetic analysis revealed possible interaction between miR156f and OsRA2
Since MIM156fOE plants showed elongated pedicels (Fig. 1d), and OsRA2 gene was reported to have function in the pedicel elongation (Lu et al. 2017), we suspected that miR156 also had relation with OsRA2. To test this hypothesis, we detected the expression level of OsRA2 in the MIM156fOE plants and the cs mutant. Similarly, the expression of OsRA2 gene was down-regulated in the MIM156fOE plants, while up-regulated in the cs mutant (Fig. 1f). The pedicel phenotype of the hybrid from the MIM156fOE and OsRA2RNAi plants was similar to that of the OsRA2RNAi plants (Fig. 6a, b). We also carried out Y1H assay to see whether SPL proteins can directly bind to the promoter regions of OsRA2 gene, but the result turned out to be negative (data not shown). These results indicated that OsRA2 might function downstream of miR156f in regulation of pedicel elongation but not through its target OsSPLs genes.

Discussion
Rice panicle is a complex trait under regulation from various aspects including miRNAs (Wang et al. 2015). In this study, we provided genetic evidences to elucidate the relationships between miR156/SPL module and some panicle development related genes, such as LAX1, LAX2, RCN2 and OsRA2. MIM156fOE plants showed decreased number of SBs (Fig. 1c), which were partially due to the down regulation of axillary meristem regulating genes LAX1 and LAX2 (Fig. 1f). And these regulations was mediated through the direct binding of OsSPL7 and OsSPL13, the miR156 target genes, to the promoter regions of LAX1 gene, and interaction between OsSPL7 and LAX2 protein (Figs. 1f and 4). Moreover, OsSPLs might have genetic interaction with LAX2, and LAX1 and LAX2 could also interact (Tabuchi et al. 2011), thus a triple protein of LAX1/LAX2/SPL complex might be formed (Figs. 2 and 4e). Altogether, axillary meristem regulating genes plays pivotal roles in the miR156f-regulated panicle architecture development. Although OsSPL14 has been proved to function in ideal plant architecture including panicle (Jiao et al. 2010; Miura et al. 2010), in the cs mutant, OsSPL14 is down-regulated to a degree not that obvious as that of OsSPL7 or OsSPL13 (Table 1, Dai et al. 2018), and further analysis proved OsSPL7 to be the mediating factor of miR156 in regulating LAX1 expression in this study, this conclusion does not exclude the role of other OsSPLs in panicle trait determination.

miR156f also regulate pedicel length in panicle development (Fig. 1a), with the MIM156fOE plants showing longer pedicle (Fig. 1d). OsRA2 was reported to regulate pedicel length in rice (Lu et al. 2017). In genetic analysis, it was revealed that miR156f and OsRA2 functioned in the same pathway in regulating pedicel development (Fig. 6). However, how miR156f/OsSPL regulates the expression of OsRA2 remains unclarified.

In addition, the RCN2 genes mainly affect the number of PBs and SBs in rice panicle development (Ikeda-Kawakatsu et al. 2012). MIM156fOE plants showed less SBs (Fig. 5b, c), this phenotype was restored in the MIM156fOE/A989 hybrid by over expression of RCN2 (Fig. 5b, c), indicating that miR156 regulated the number of branches in the panicle through RCN2. In the cs mutant, where RCN2 was up-regulated (Fig. 1f), the panicle was not dense, however, when crossed with A989, the panicle was dense (Fig. 5a, c). Indicating that regulation of RCN2 on the number of branches might have dosage effect. An example for this is that when RCN2 was driven by double 35S promoter, the panicle failed to develop, which might due to too much delayed spikelet meristem transition (Li et al. 2010).

Theoretically, the cs mutant would show a much dense panicle, with increased expression of several RCN genes which would increase the number of PBs and SBs; increased expression of OsRA2, which would decrease pedicel length, and increased expression of IDS1 and MFS1, which would increase the number of spikelets (Fig. 1f). However, the cs mutant showed a panicle architecture quite to the contrary (Fig. 1c, e). One explanation is that there might be some buffering mechanism under control of miR156f, since if the cs mutant develop accordingly to the expression level of these panicle related genes, it
would be an extreme dense panicle that run out of control, not to mention that many genes have dosage effect. This buffering mechanism might be a result of the interwoven interaction among different panicle development related factors, which deserve more investigation.

**Conclusions**

We revealed that miR156f influenced expression of many panicle related genes. miR156f/SPL directly regulated LAX1 at the transcription level. And tight genetic relations existed between miR156f and LAX1, and miR156f and LAX2. Meanwhile, there was genetic relation between miR156f and RCN2, which might provide a buffering mechanism for miR156f in mediating panicle architecture regulation. miR156f might function in the same pathway with OsRA2 in pedicel regulation. We concluded that miR156f acts as an important regulator of panicle architecture through influencing various aspects of panicle development.

**Material and methods**

**Plant species and growth conditions**

Wild type rice species ZH11 (*Oryza sativa* L. subsp. japonica cv. Zhonghua No. 11) was used as the host for transgenic transformation in this study. ZH11, the cs mutant the A989 mutant, the lax1 mutant, the lax2 mutant, and all the transgenic plants were grown in the green house, with 10 h light and 14 h dark, or in the field...
under natural conditions in summer, Shanghai, China. The construction of the respective transgenic plants was described in respective references.

**qRT-PCR analysis**
Total RNA was extract from different tissues using TRIzol (Invitrogen), followed by DNase I digestion. For qRT-PCR, cDNA was synthesized from 1 μg of total RNA using One Step SYBR PrimeScript RT-PCR Kit (TaKaRa), and 1 μl of cDNA was used as template for real-time analysis. Sampling and expression measurement was repeated three times. The *actin* gene was used as internal reference.

**Y1H assay**
The full-length cDNAs of *OsSPL7* and *OsSPL13* were amplified with gene-specific primers (Additional file 2: Table S1), and then fused into the activation-domain (AD) of vector pPC86. Fragments containing “GTAC” in *lax1* gene promoter were amplified with gene-specific primers (Additional file 2: Table S1) and fused into the vector p178 at the *XhoI* site. The p178 and pPC86 constructs were transformed into the yeast strain EGY48 together. The yeast strain was growth on SD selective medium (SD-His-Leu) and observed in blue on Chromogenic medium. The transformants containing void plasmid pPC86 and p178 constructs were used as a negative control. Y1H assay was carried out as described (Matchmaker One-hybrid System; Clontech).

**Y2H assay**
The open reading frame (ORF) of *OsSPL7* was amplified and cloned into the prey vector pGAD-T7. The ORFs of *LAX1* and *LAX2* were amplified and cloned into the bait vector pGBK-T7. The Y2H assay was performed according to the manufacturer’s instructions (Clontech).

**ChiP analysis**
ChiP analysis was carried out as previously described (Dai et al. 2018).
Measurement of panicle traits
At least 20 panicles from each line were used for the analysis of panicle characters, including panicle length, the number of PBs and SBs. Fifty spikelets from each line were used to analyze the pedicell length. Data was shown as mean ± SD.

Measurement of plant height and tiller number
Plant height of at least 20 plants were measured at the mature stage, and effective tillers (tillers that bear panicles) were counted at the same time. Data was shown as mean ± SD.

Additional files

Additional file 1: Plant height and tiller number of the cross between A989 and MIM156fOE plants, and A989 and the cs mutant. (TIF 12961 kb)

Additional file 2: Primer sequences used in this study. (XLSX 11 kb)

Abbreviations
AM: Axillary meristem; CK: Cytokinin; DST: DROUGHT AND SALT TOLERANCE; GA: Gibberellin; IM: Inflorescence meristem; LAX1: LAX PANICLE 1; LAX2: LAX PANICLE 2; LP: LARGER PANICLE; MM156f: Mimicry miR156f; MIK156fOE: Mimicry miR156f over-expression; miRNA: microRNA; MOC1: MONOCOT 1; OsCKX2: CYTOKININ OXIDASE; OsRA2: OsRAMOSA2; PB: Primary branches; SAM: Shoot apical meristem; SAM: Shoot apical meristem; SB: Secondary branches; SM: Spikelet meristem; SPL: SQUAMOSA PROMOTER BINDING PROTEIN-like; SPL7Flag: OsSPL7 fused with flag tag driven by its own promoter; SPL7myc: OsSPL7 fused with myc tag driven by its own promoter; SPL7RNAi: OsSPL7 RNA interference

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Authors’ contributions
YX, DZ and ZX carried out the experiments, ZX and WJ analyzed the data, MX and SZ designed the experiments and wrote the MS. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate
Not applicable

Consent for publication
Not applicable

Competing interests
The authors declare that they have no competing interests.

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