Characterization and Mapping of Rice Mutants, \textit{des2} and \textit{des5}

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Abstract

Identification of key regulator genes in the control of spikelet development in panicle, one of the main agronomic traits to determine rice yield, is important to investigate the underlying molecular mechanism and provide the molecular basis for analysis of the agronomic traits. In this study, we have identified a group of mutants with conspicuous reduction of spikelet number, which are designated \textit{decreased spikelets} (\textit{des}). Two of the \textit{des} mutants, \textit{des2} and \textit{des5}, were analyzed in detail. We showed that a single recessive locus was responsible for \textit{des2} phenotype, which is located on the long arm of chromosome 6. The \textit{des2} locus was cloned and it was found that \textit{des2} is a new allele of moc1. Positional cloning and sequence analysis showed that there is a point mutation in the HLH domain-encoding region of LAX gene in \textit{des5}, indicating that \textit{des5} is a new allele of lax. Our data indicate that spikelet development is regulated by the same pathway in the control of development of axillary branches in rice.

Keywords

Rice mutants, \textit{des2}, \textit{des5}, Characterization, Mapping
locus in the control of tillering in rice. Positional cloning revealed that MOC1 encodes a transcription factor belonging to the GRAS family. It is demonstrated that MOC1 may function as a master regulator to determine tillering by modulating the expression of the other two transcription factors, OSH1 and OsTB1 (Li et al., 2003). It has been reported that there were multiple genetic components in the control of branching during rice development. lax panicle (lax) possesses the phenotype of reduced number of axillary branches (Komatsu et al., 2001; 2003). It has been shown that LAX encodes a bHLH transcription factor. Another mutant, small panicle (spa) displays an abnormal phenotype with a smaller panicle but with normal tiller number. However, when the spa was introduced into lax-1 background, a weak allele of lax, the double mutant exhibited extremely deficiency in tiller number and had a single main culm bearing a barren panicle. It was proposed that all axillary branch development should be controlled by a single genetic mechanism (Komatsu et al., 2003). In aberrant panicle organization 1 (apo1), the precocious termination of the main-axis meristem and the precocious conversion of primary branch meristem into spikelet meristem results in short rachis and reduces the number of primary branches (PB) and spikelets significantly (Ikeda et al., 2005; 2007). APO1 has been cloned and found to encode an F-box protein, indicating that ubiquitin-proteasome pathway may be involved in regulating branch development (Ikeda et al., 2005; 2007). It has been well established that SCF complex constituted by Skp1p, Cullin and F-box protein can ubiquitinate and target specific substrate to 26S proteasome for degradation (Deshaies, 1999; Smalle and Vierstra, 2004; Moon et al., 2004; Gagne et al., 2002; Pickart, 2001; Risseeuw et al., 2003; Cardozo and Pogano, 2004; Zheng et al., 2002). Thus, posttranslational modification of some key factors or the interaction at protein level should play an important role during axillary branching. Recently, a pair of enzymes regulating cytokinin level but with opposite function were identified to control panicle morphology (Ashikari et al., 2005; Kurakawa et al., 2007), indicating phytohormones is implicated in axillary branch development.

Although quite a few regulatory genes controlling axillary branch development have been identified, the underlying genetic network just emerges and more key regulators are expected to be discovered. In this study, we identified a group of mutants, decreased spikelets (des), which have conspicuous effect to reduce the spikelet number. Among these des loci, DES2 was cloned by map-based cloning approach and analyzed in detail. It was found that des2 is allelic to MOC1. In des2, a point mutation occurred in the 3’ end of coding region in MOC1, leading to a premature translation stop and giving rise to a deletion of the last 5 amino acid residues in C terminus of MOC1. des5 was identified as a new allele of lax, and carries a point mutation at the HLH domain. Furthermore, it was found that des2 and des5 could interact with each other, abolishing all axillary branch development. Out data indicate that MOC1 and LAX should interplay with each other in the control of development of axillary branches in rice.

1 Materials and methods
1.1 Plant materials
Four des mutants, des1, des2, des3 and des4, were identified from an M2 mutagenized japonica rice cultivar Zhonghua 11 (ZH11) by γ-ray irradiation and des5 from another one by EMS mutagenesis. The mapping population was derived from F2 segregation population of the cross between des2 and the indica rice cultivar Longtefu.

1.2 DNA extraction
Total DNA was extracted from young rice leaves according to the CTAB method (Murray and Thompson, 1980), with some modifications. About 0.1 g rice leaves were ground into powder in liquid N2. Add 400 μL extraction buffer and incubate in 65°C water bath for 30–60 min with occasional gentle mixing. Add 400 μL chloroform and shake followed by centrifugation at 12 000 rpm for 5 minutes. Transfer the supernatant into a new centrifugal tube and add 0.8~1 volume of cold isopropanol with mixing. Place it at -20°C for 15 min followed by centrifugation at 12 000 rpm for 5 minutes. Pour off the supernatant and wash the pellet with 500 μL 70% ethanol once. After drying in the air, add 100 μL TE or sterile water to dissolve DNA.
1.3 Marker analysis by polyacrylamide gel electrophoresis (PAGE)

The PCR reaction system (20 μL) consists of 2 μL 10×buffer (supplied with Taq polymerase), 0.2 mmol/L of dNTP, 5% dimethyl sulfoxide (DMSO), 20 ng of DNA, 0.1 μmol/L of primers and 1 U of Taq polymerase. The PCR program for SSR and dCAPS markers is as follows: 94℃ for 3 min, followed by 35 cycles of 94℃ for 30 s, annealing (temperature adjusted with different primers) for 30 s, 72℃ for 30 s, and finally 72℃ for 10 min. The primer sequences of these SSR and STS markers were downloaded from Gremene (http://www.gramene.org) and RGP (http://r-gp.dna.affrc.go.jp/E/index.html) respectively. The restriction endonuclease digestion for dCAPS markers was conducted in a 20 μL reaction mixture containing 2 μL 10×Buffer (supplied with enzyme), 0.2 μL 100×BSA when necessary, 0.1~0.5 μg of PCR products and 15~20 U of restriction enzyme at the optimal temperature for 2~3 hours. The polymorphisms of these markers were analyzed on 6% polyacrylamide gel stained with 0.1% silver nitrate.

1.4 New molecular markers in des2 region

SSR markers listed in Gramene database are normally tested and used in this study. In the cases when new markers were needed, they were developed by either optimizing the ones from the database or comparing the public available japonica and indica sequences (Feng et al., 2002; Goff et al., 2002; Kikuchi et al., 2003; International Rice Genome Sequencing Project, 2005; Sasaki et al., 2002b; The Rice Chromosome 10 Sequencing Consortium, 2003; Shimamoto and Kyozuka, 2002; Yu et al., 2002; Zhang et al., 2004) so as to design new markers including SSR and dCAPS in potential polymorphic sites (Table 1).

Table 1 Newly-developed molecular markers

| Marker   | Forward primer  | Reverse primer         |
|----------|-----------------|------------------------|
| RM20422d | CCTCTTGCTTATGCTCCTC | CTGGCTCCTCCACGACT      |
| RM20402d | CAACAACCCAAGTAACGAT | AGGTGAATAAAAGGAAAGAGA |
| RM20384d | GCTCCAAACTCTTACTCTT | TATCTATCATGCGACCTT    |
| RM20371d | TTTTCTTTCCACTTGTG | TAATGCCTGCTGACTT      |
| RM20388d | TTTGGAAGCCTCCGATAC | CCATTGACGGCGAAGAGT    |
| RM20378d | GGAATGTCCTTCTCGAGAT | GAAACCTTGGCTAATGGC    |
| OsSSR35k | GACATTCCGTAAGAAAGTT | AGCAGTCTCTTCTAATAGG   |
| OsSSR60k | GTCAAGTTCCGCCGAGA | TCGGCTTCTTCGTTG        |
| RSL0081  | ACACCAACAACCTCAAATCTG | CTAATGTCAGATTCAACACTAGC |
| OsdCAPS1 | CGTAGCAAAGACAGAGAAACGTCTA | GACATACAAAAGGAGGCGAATA  |
| OsdCAPS2 | AACCTGAAATATGGCTTGTG | GTAGCTGACGTGACGACAACATG |
| OsdCAPS3 | CACGCCGCTGCCGCGCGCGCG | CGCTTCCGTTCGTCTC     |

1.5 Construction of linkage map

Linkage map was constructed by using MapDraw V2.1 (Liu and Meng, 2003).

2 Results

2.1 Phenotype of des mutants

To investigate the key regulators in the control of spikelet development, we screened rice mutants with the phenotype of significant reduction of spikelet number, which were designated decreased spikelets (des). Five complementary groups of des mutants, des1, des2, des3, des4 and des5, were identified in the mutagenized M2 population of ZH11. In the des mutants, the reduction rate of spikelets was typically about 30%~40% reduction in comparison to the wild type (Figure 1 and Table 2). However, in des mutants the development of tillers, as well as primary branches (PB) and secondary branches (SB), were also affected to various extents (Figure 1 and Table 2). Among these des mutants, des2 and des5 were characterized in detail in this study.
2.2 Genetic analysis and primary mapping of des2

Genetic analysis was conducted to determine whether des2 is controlled by a single locus. In a cross between des2 and ZH11, the segregation ratio of wild type and mutants in the F2 population was in accordance with 3:1 (n=126, $\chi^2=0.01<\chi^2_{0.05,1}=3.84$). This indicates that a single recessive locus is responsible for des2 (Table 3).

Table 3 Segregation analysis among F2 population

| Cross       | F2  | WT | Mutant | $\chi^2 (3:1)$ |
|-------------|-----|----|--------|----------------|
| des2×ZH11  | 126 | 95 | 31     | 0.01           |
| des5×ZH11  | 140 | 108| 32     | 0.35           |

A mapping population was constructed by crossing des2 with an indica variety, Longtefu. 147 individuals with typical des2 phenotype in F2 population were selected for des2 mapping. Primarily, 15 mutant individuals were randomly selected to comprise a DNA pool, with which 92 SSR markers evenly distributed in 12 chromosomes were subjected to tests for linkage with des2 locus. As a result, two SSR markers RM3827 and RM528 on chromosome 6 were found to be linked with des2, which was further confirmed by testing some F2 individuals with these two markers. Then the candidate region was further framed between RM162 and a STS marker R3879. Furthermore, new molecular markers including SSR and dCAPS were developed by analyzing the japonica and indica genome sequences and their polymorphisms between ZH11 and Longtefu were analyzed in PAGE (Table 1). By developing the new markers, des2 was confined between RM162 and RM20384d (short for RM20384-derived. See Materials and Methods) with estimated genetic distances of 1.9 cM and 1.6 cM respectively (Figure 2).
2.3 Fine mapping of des2

A larger mapping population was constructed for fine mapping of des2, from which 500 mutant plants were subjected to the analysis. Then the locus was delimited to a 28 kb region between two markers OsdCAPS2 and OsdCAPS3, and the des2 locus was found to co-segregate with a SSR marker RM20378d since there was no recombinant among the 500 mutant plants. A PAC contig was found to span the des2 locus (Figure 3). Within the delimited region, only one gene encoding a hypothetic protein and the previously identified MOC1 gene were found, according to the annotation in NCBI (http://www.ncbi.nlm.nih.gov). No mutation was detected in the unknown gene by sequencing, however, there was a point mutation was found in the 3’ end of MOC1 coding region, leading to a premature translation stop and giving rise to a truncated MOC1 protein with a deletion of the last 5 amino acid residues in C terminus (Figure 4A). Therefore, des2 phenotype should be caused by the point mutation in MOC1 gene.

![Figure 3 The fine map encompassing des2 locus on chromosome 6 and PAC contig spanning des2 locus](image)

2.4 des5 being allelic to LAX

Apart from the reduction of spikelet number, defects on panicle development with less SB and SP are also observed in des5 (Table 2). Genetic analysis showed that a single locus should be mutated and lead to the defects in des5 in a recessive manner (n=140, \(\chi^2=0.35<\chi^2_{0.05,1}=3.84\)) (Table 3). Genetic analysis of des5 locus was conducted and positional cloning delimited des5 to a region between markers RSL0081 and RM3640 on the long arm of chromosome 1 (data not shown). Sequence analysis indicated that LAX is located inside the region, which has been shown to play a key role in the control of branch formation during panicle development in rice (Komatsu et al., 2001). Later it was found that in des5 there is a point mutation in the coding region of LAX gene, giving rise to a substitution of Phe-63 for Leu-63 in its HLH domain (Figure 4B). It is expected that the mutated lax should not have a proper function and be consistent with the abnormal panicle development in des5. Therefore, we conclude that des5 should be a new allele of lax mutant.

3 Discussion

In this study, we identified a few des mutants, which mainly affect the spikelet development but with relatively minor effects on development of tiller and branch in panicle. Two loci, des2 and des5, were cloned and the others are subjected to analysis in detail at the moment. It is found that des2 and des5 are allelic to the regulatory genes, MOC1 and LAX, respectively.
Figure 4 The mutation sites in des2 and des5

Note: A schematic representation of MOC1 gene (A) and LAX gene (B), respectively; The mutation site (indicated in bold) has a base substitution leading to premature translational stop in des2 (A) and substitution of Phe for Leu (B), respectively; +1 indicates the starting site of translation; +1 326 (A) and +645 (B) indicate the termination site of translation respectively; The black box represents exon and the blank line indicates GRAS (A) and HLH domain (B) respectively; the left and right gray boxes (B) represent 5'UTR and 3'UTR, respectively.

The des5 displays very similar or consistent phenotype as the one of lax1 (Komatsu et al., 2001). It has been found that LAX encodes a bHLH transcription factor and regulates panicle development by monitoring the initiation of axillary primordium (Komatsu et al., 2001; 2003). In des5, there is a point mutation at the HLH domain of LAX, which presumably has a key function for protein-protein interaction and forms either homodimer or heterodimer (Heim et al., 2003). For example, SPCH, MUTE and FAMA are three key bHLH-type transcription factors regulating stomatal development (MacAlister et al., 2007; Pillitteri et al., 2007; Ito and Bergmann, 2006). It has been shown that their individual function requires physical interaction with two other bHLH-type transcription factors SCREAM1 and SCREAM2; moreover, yeast two-hybrid demonstrates that MUTE itself can strongly form homodimer (Kanaoka et al., 2008). Consistently, our data suggest that the Leu-63 at the HLH domain should be essential for the protein function of LAX.

MOC1 has been previously reported as a key factor in the control of tiller development (Li et al., 2003). However, in the case of des2, there are significant differences between the phenotypes of des2 and the previously reported moc1. The phenotype of a single culm without tiller in moc1 is contrasted to the relatively normal tillering but conspicuous effect on spikelet number in des2. These could be due to the different genetic background, or alternatively could be explained by the allelic difference. The lesion of MOC1 protein in des2 mutant is subtle in comparison with the one in moc1. In des2, the last 5 amino acid residues in the C terminus of MOC1 is missing, whereas the last 125 residues were deleted in moc1 mutant with an extra 1.9 kb insertion of a retrotransposon (Li et al., 2003). Therefore, des2 can be considered as a weak allele of moc1. Nevertheless, the serine homotrimeric stretch is comprised in the last 5 residues of MOC1 which is lost in des2. It is likely that these serine residues are extra potential phosphorylation sites besides the conserved tyrosine (Li et al., 2003). Our data indicate the importance of the C terminus for the protein function of MOC1. In des2 mutant, the conspicuous phenotype of substantial reduction of the spikelet number in panicle, but relatively minor effect on the number of tiller and branches in panicle, suggest that the action of MOC1 could be differentiated depending on the developmental stages. During rice development, the axillary meristem acquires distinct identities in vegetative and reproductive development. It is possible that different axillary meristem development requires differential levels of MOC1 activity, or different domains of MOC1 protein play different roles, which should be essential to interact with other key regulators in the control of formation of tiller, PB, SB and spikelet, respectively.

While the strong phenotype of moc1 could be an obstacle to investigate the genetic interaction between...
MOC1 and other key regulators, the weak phenotype of *des2* was explored in this study. Our preliminary data indicate that different combinations of different *des* double mutants display enhanced phenotype (He et al., unpublished data), suggesting that the interplay between different *DES* genes should be a common theme. The synergistic effect of different double *des* mutants could shed some light on the potential mode of *DES* function. It is possible that different *DES* genes could be involved in several distinct pathways and act independently to control axillary branch development. Alternatively, MOC1 and LAX should function as partners in a common pathway. However, these two possibilities are not exclusive. In phytochrome A signaling pathway, two positive regulators LAF1 and HFR1 have both independent and interdependent functions with respect to the inhibition of hypocotyl elongation by far-red light (Jang et al., 2007). It was shown that COP1-targeted LAF1 and HFR1 protein degradation was inhibited by their physical association (Jang et al., 2007). There have been several reports indicating the regulation of shoot branching at protein level and when an F-box protein, APO1 is mutated, the development of PB and spikelet is affected (Ikeda et al., 2005; 2007). Thus, the posttranslational modification of some key factors or interaction at protein level might play an important role in regulating the formation of branching and spikelet in panicle. MOC1 belongs to the GRAS family and consistently is found to be localized in nucleus. LAX is a bHLH transcription factor and its function could be dependent on its heterodimeric status. In the future work, it is feasible to test whether there is direct protein-protein interaction between LAX and MOC1. It has been proposed that development of axillary branches is controlled by a single genetic mechanism (Komatsu et al., 2003). Therefore, our data is consistent with this notion since most of the spikelets are initiated as the axillary primordium in the periphery zone in the apical meristems of PB and SB. Our data indicates that spikelet development is regulated by the same pathway in the control of development of axillary branches in rice. With more des loci being identified and more *DES* genes cloned, the complexity of the genetic network in the control of spikelet development should be investigated in both genetic and protein levels.

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