

# **Identification of quantitative trait loci (QTL) associated with sheath blight tolerance in rice**

## **Ashok Koshariya, Indra Kumar, Ashish Pradhan, Umesh Shinde<sup>1</sup> , S. B. Verulkar<sup>1</sup> , Toshy Agrawal<sup>1</sup> and Anil Kotasthane**

Department of Plant Pathology, <sup>1</sup>Department of Plant Molecular Biology and Biotechnology CoA, Indira Gandhi Krishi Vishwavidyalaya, Raipur

(Received: May 2017; Revised: February 2018; Accepted: March 2018)

#### **Abstract**

**Sheath blight, caused by the pathogen Rhizoctonia solani Kühn, is one of the most serious diseases of rice and leads to severe yield losses worldwide. A recombinant inbred line (RIL) population consisting of 122 lines was constructed from a cross between Danteshwari a high yielding popular rice cultivar but moderately susceptible to water stress, susceptible to sheath blight and Dagad Deshi a tall deep rooted poor yielder and tolerant to water stress and sheath blight. Five traits, namely infected tillers per hill, lesion size (total coalescing lesions) and individual lesion (one spot) and lesion's length and width) were used to evaluate sheath blight resistance. Using the RIL population and 162 molecular markers, 11 quantitative trait loci (QTLs) were identified for the five traits. Identification of map position was accomplished by identifying BAC or PAC clones that simultaneously contained a hit from the microsatellite / HvSSR marker further helped us to generate a high resolution molecular marker map of the identified QTL region. Identified QTLs were located on seven chromosomes. A major QTL for infected tillers per hill on chromosome 1 was identified with phenotypic variance of 18.02%. Six QTL's with positive additive effect and one with negative additive effects was identified indicating alleles at these loci are being contributed by either of the parents respectively.**

**Key words:** QTL, rice, sheath blight, markers

#### **Introduction**

Sheath blight (ShB) disease of rice (Oryza sativa L.), caused by Rhizoctonia solani Ku¨hn, is particularly important in intensive rice production systems worldwide (Ou 1985; Teng et al. 1990; Savary et al. 2000, 2006). Rice crops with high attainable yields (Savary and Mew 1996) are especially affected. Yield loss estimates of 5-10% for tropical lowland rice in Asia has been reported (Savary et al. 2000). Breeding for sheath blight resistance has been difficult, owing to the lack of identified resistant donors in cultivated varieties (Bonman et al. 1992) and semi-saprophytic character and wide host range of the pathogen affecting 32 plant families and 188 genera (Gangopadhyay and Chakrabarti 1982). Few varieties with varying levels of rice sheath blight resistance were identified after large-scale screening of cultivated germplasm and related wild species (Eizenga et al. 2002; Prasad and Eizenga 2008). Resistance to rice ShB is a complex, quantitative trait controlled by polygenes (Sha and Zhu 1990; Li et al. 1995; Pinson et al. 2005). So far, nearly 70 QTLs for sheath blight resistance have been identified in rice, distributed on all 12 chromosomes (Li et al. 1995; Zou et al. 2000; Che et al. 2003; Han et al. 2002; Kunihiro et al. 2002; Pinson et al. 2005; Tan et al. 2005; Xiang et al. 2007; Xie et al. 2008; Liu et al. 2009; Sharma et al. 2009; Channamallikarjuna et al. 2010). However, a number of researchers (Xie et al. 1992; Pan et al. 1999) proposed that ShB resistance in some rice varieties is controlled by a only a few major genes. The quantitative resistance in some of the most resistant varieties, such as Tetep and Tadukan, can offer excellent protection against the pathogen under field conditions (Groth and Nowick 1992). The development of molecular markers has led to rapid progress in understanding the mechanisms underlying resistance to sheath blight using the quantitative trait locus (QTL) mapping method. Identification of genomic loci governing complex traits has been facilitated by the development of quantitative trait locus (QTL) mapping approaches using segregating bi-parental

\*Corresponding author's e-mail: kotasthaneaj@yahoo.com

Published by the Indian Society of Genetics & Plant Breeding, A-Block, F2, First Floor, NASC Complex, IARI P.O., Pusa Campus, New Delhi 110 012; Online management by indianjournals.com; www.isgpb.org

populations. Limited population size with low density marker coverage usually proves sufficient to identify QTLs for different traits. These roughly estimated QTL intervals extend over several cM, a genetic distance that translates into genomic regions with large number of candidate genes. This limited resolution results mainly from the small number of recombination events that are accumulated over the few generations needed to develop a bi-parental mapping population (Balasubramanian et al. 2009). In this study an attempt was made to map QTLs for sheath blight tolerance using 122 RILs in rice.

#### **Materials and methods**

#### **Mapping population and phenotyping**

The population used in this study consisted of 122  $F_{13}$  recombinant inbred lines (RILs) derived by single seed descent method from a cross between Danteshwari  $(D_1)$  (high yielding popular rice cultivar but moderately susceptible to water stress, susceptible to sheath blight) and Dagad Deshi (D<sub>2</sub>) (a tall deep rooted poor yielder and tolerant to water stress and sheath blight). The  $D_1 \times D_2$  derived population was phenotyped at Indira Gandhi Krishi Vishwavidyalaya , Raipur during 2014 wet monsoon. Raipur is located at  $21^{\circ}$  16' N and 81 $^{\circ}$  36' E at an altitude of 289.6 m above sea level. Each line having 3 rows of 1.5 m length were screened. Standard agronomic practices were followed to raise the crop in nursery and field conditions.

#### **Inoculum production and inoculation procedure**

Multinucleate compatible isolate of R. solani isolate rice, belonging to the AG-1 IA anastomosis was grown on potato dextrose agar at  $28\pm1\textdegree C$  for 6 days for mass multiplication. Rice bran supplemented with dextrose (@ 17g/kg), was moistened, thoroughly mixed and filled to 3/4th the volume of glass bottles. These were then sterilized at  $121.6^{\circ}$ C for 45 minutes. Small bits of PDA blocks containing actively growing mycelium of R solani was inoculated in bottles containing presterilized cooled rice bran and incubated at  $25^{\circ} \pm 1^{\circ}$ C. Growth of R solani appeared over night and the fungus completely colonized the rice bran within one week at  $25^{\circ}$ ±1 $^{\circ}$ C. No sclerotial were produced in the colonized rice bran. R solani colonized rice bran was harvested and pooled in a tray. Clumps in rice bran were formed due to colonization of  *solani and were further* pulverized by passing them through a wire mesh. This resulted in the breaking of the mycelium into small fragments. Every particle of the pulverized rice bran

was of uniform size and contained colonized mycelial bits of R. solani and therefore served as inoculum. The method of inoculum deposition in our present investigation mimics high contact frequency between tissues essentially required for sheath blight epidemics. Inoculation was done at maximum tillering stage. Pulverized rice bran containing colonized mycelial bits of R. solani were deposited uniformly in the whorls of plant hills. The inoculum also got deposited near the base of the plant. The incubation period (IP) was estimated as the period from inoculation to appearance of approximately 50% water soaked lesions (Yeh and Bonman 1986). Observations were recorded for number of infected tillers per hill, and by measuring the total (coalescing lesions) and individual (one spot) lesion length and width. Two disease criteria were assessed for each inoculated plant i.e. number of infected tillers per hill represents the horizontal spread of the disease between the tillers and measurement of lesions size represents the vertical and horizontal spreading degree of the disease on individual tiller and severity.

#### **Construction of linkage map and QTL analysis**

Genotypic data generated from SSR HvSSR markers were used for linkage map construction using computer software MAPMAKER/EXP, version 3.0. Kosambi function was used to calculate the genetic distances between the markers (Kosambi, 1943). QTL mapping (Composite Interval Mapping) was carried out (Zeng et al. 1994) with a threshold value of 2.5 to 3.0 LOD, was used for declaring the presence of a suggestive QTL. Contribution rate  $(R^2)$  was estimated as the percentage of the total phenotypic variation explained by each locus. Graphical presentation of the linkage map was carried out by using MapChart2.2 (http:// www.biometris.wur.nl).

The genotypic data and field based phenotypic data of sheath blight lesion size (number of infected tillers per hill, total (coalescing lesions) and individual lesion (one spot) lesion length and width) was analyzed using QTL cartographer 2.5.

#### **Results and discussion**

#### **QTL analysis**

The parents along with RILs exhibited marked variation for the reaction to sheath blight. The two parents (Danteshwari and Dagaddeshi) showed differences for number of infected tillers per hill but not for lesion size (total (coalescing lesions) and individual lesion (one spot) lesion length and width) (Table 1, Fig. 1). The

for number of infected tillers per hill and lesion size in total (coalescing lesions) lesion width and length, S. No. individual lesion length and width Parents Total no. of All coalescing Individual tillers infec- lesions besion ted/hill Length Width Length Width Danteshwai 41.73913 4.5625 0.625 2.0375 0.6 Dagaddeshi 26.37363 5.125 0.6875 2.275 0.625 Min. 13.38583 1.7 0.3875 1.3375 0.425 Max. 95.65217 9.5875 1.057143 4.425 0.886 Min. = Minimum; Max. = Maximum  $CH #$  No. of QTLs 1 2 2 3 1 3 4 1 4 5 1 5 6 1 6 7 1 7 12 4 Total 11

**Table 1.** Reaction of parents, Danteshwari and Dagaddeshi **Table 2**. Identified QTL on D X D derived RIL population

**Fig. 1. Frequency distributions of 122 genotype (RIL) for sheath blight lesion size**

RILs exhibited transgressive segregation in both directions, which indicated that neither parent contained all the alleles for resistance or susceptibility. Reaction of RILs, for sheath blight, could not be classified into discrete classes of resistance and susceptibility as they showed continuous variation and skewed distribution that suggested the inheritance is quantitative. Transgressive segregations among the RILs were observed for the number of infected tillers per hill, total (coalescing lesions) and individual lesion (one spot) lesion length and width.

The genotypic data and field based phenotypic data of sheath blight lesion size (number of infected tillers per hill, total lesion width, total lesion length, individual lesion length and individual lesion width) was analyzed using QTL cartographer 2.5. Eleven QTL's were identified on chromosomes 1, 3, 4, 5, 6, 7 and 12 for sheath blight reactions (number of infected tillers per hill but not for lesion size {total (coalescing lesions) and individual lesion (one spot) lesion length and width} under artificial inoculated conditions (Table 2). QTL mapping was carried out following composite interval mapping (Zeng et al. 1994) with a threshold value of 2.5 and 3.0 LOD, was used for declaring the presence of a suggestive QTL. Contribution rate  $(R_2)$  was estimated as the percentage of the total phenotypic variation explained by each locus. The LOD score and

phenotypic variance for QTL (LOD 3 and above) ranged from 3.00 to 5.13 and 4.18 to 18.02, respectively (Table 3). QTL's with positive additive effect influencing: number of tillers infected per hill on Ch# 1, two each for lesion width (Total) on Ch#3 and 12, lesion length (individual) on Ch#1 and 12, and one lesion width (individual) on Ch# 5 were identified. A QTL with negative additive effects influencing individual lesion width on Ch# 5, was identified indicating. This suggests that alleles at these loci are contributed by either of the parents respectively (Table 3).

The LOD score and phenotypic variance for QTL (LOD 2.5 and above but below 3) ranged from 2.51 to 2.83 and 1.16 to 10.95 respectively (Table 3). Two QTLs influencing total (coalescing lesions) width on Ch# 4, 12 were with positive additive effect were identified. Two QTLs with negative additive effects influencing total (coalescing lesions) width on Ch# 6, 7 was identified indicating that alleles at these loci are being contributed by either of the parents respectively (Table 3). The location of QTLs reported for sheath blight varies in different populations and at different environments. Many important traits are quantitatively expressed and influenced by the environment and tend to show varied degree of genotype x environment (GxE) interaction (Zhuang et



**Fig. 2. Mapped QTLs on different chromosomes for sheath blight tolerance using 122 RILs in rice**

al. 1997). Over the past two decades several ShB resistance quantitative trait loci (QTL) have been mapped, but consistency across results from different studies has not always been observed (Pinson et al. 2005).

**Table 3.** Identified QTL influencing sheath blight lesion size on D<sub>1</sub> x D<sub>2</sub> derived genotyped RIL population under artificial inoculated conditions

S.No. Trait		Ch.	Marker	Position	<b>LOD</b>	a <sub>0</sub>	$R^2$
	QTL's with threshold value 3 and above						
	QTL's with Positive additive effect						
1	Number of tillers infected per hill	1	<b>HySSR1-87</b>	155.11	4.23	9.44	18.02
2	Lesion Width (Total)	3	RM232	50.91	5.13	0.02	4.18
3	Lesion Width (Total)	12	RM260	69.11	3.23	0.03	11.85
4	Lesion length (individual one spot)	1	RM243	43.21	3.42	0.18	10.31
5	Lesion length (individual one spot)	12	<b>RM277</b>	75.81	3.00	0.23	9.28
6	Lesion width (individual one spot)	5	RM459	77.91	3.31	0.04	11.21
	QTL's with negative additive effect						
$\overline{7}$	Lesion width (individual one spot)	12	<b>HySSR12-48</b>	108.61	3.08	$-0.03$	10.70
	QTL's with threshold value 2.5 and above but <3						
	QTL's with positive additive effect						
$\mathbf{1}$	Lesion Width (Total coalescing lesions)	4	RM564	66.21	2.51	0.01	1.16
2	Lesion Width (Total coalescing lesions)	12	<b>HySSR12-40</b>	91.91	2.83	0.02	3.14
	QTL's with negative additive effect						
1	Lesion Width (Total coalescing lesions)	6	EM1_72	10.41	2.52	$-0.02$	3.17
2	Lesion Width (Total coalescing lesions)	7	RM3394	3.01	2.73	$-0.03$	10.95

Ch.= Chromosome No; \*= figures in parenthesis indicate trait number

#### **QTL's with threshold value 3 and above**

Rice doubled haploid lines and RILs have been extensively used for QTL mapping for sheath blight resistance (Kunihiro et al. 2002; Han et al. 2002). Two QTLs have been identified for sheath blight resistance on chromosomes 3 and 12 in rice line WSS2 which is derived from Tetep (Sato et al. 2004). The accurate measurement of ShB resistance under field conditions i.e., of disease intensity and observed susceptibility (Yuen and Forbes 2009) depends on a range of environmental factors (Ou 1985; Castilla et al. 1996; Eizenga, Lee, and Rutger 2002) and plant morphological traits, such as plant height (Li et al. 1995; Zou et al. 2000; Pinson et al. 2005), which interact, resulting in the observed variation in resistant (susceptible) phenotypes. During the present investigation quantitative trait loci on D X D derived genotyped RIL population under artificial inoculated condition was identified which influenced number of tillers infected per hill with LOD value 4.23 which explained 18.025 % phenotypic variation. Two QTL's for lesion width (total) with LOD value 5.13 (Maker RM232) and 3.23 (Maker RM260) explained 4.18 and 11.85 % phenotypic variation on chromosome 3 and 12, respectively (Table 3). Only two QTLs were identified for lesion length (individual) with LOD value 3.42 (Maker RM243), 3.00 (Maker RM277) and one QTL for lesion width (individual) with LOD value 3.31 (Maker RM459) explained 10.31, 9.28, and 11.21 % phenotypic variation on chromosomes 1, 12 and 5 respectively (Table 3). QTL on D X D derived genotyped RIL population under artificial inoculated condition was identified which influenced lesion width with LOD value 3.08 which explained 10.70 % phenotypic variation respectively was positioned on CH#12 (Table 3).

### **QTL's with threshold value 2.5 and above but <3**

Four QTL's for Lesion Width (Total) (3) with LOD value 2.51 (Maker RM564), 2.83 (Maker HvSSR12-40), 2.52 (Maker EM1\_72) and 2.73 (Maker RM3394) explained 1.16, 3.14, 3.14, and 10.95 % phenotypic variation on chromosomes 4, 12, 6 and 7 respectively (Table 3). Absolute resistance to R. solani is not available in any of the rice germplasm grown worldwide. However, it has been reported that resistance to  $R$ . solani is a typical quantitative trait controlled by polygenes in rice (Sha and Zhu 1990). There are some rice lines such as Tetep, Tadukan, Teqing, Jasmine 85, ZYQ8, Minghui 63, LSBR-5 and LSBR-33 in which a high degree of quantitative resistance is available against this pathogen under field conditions (Khush 1977; Xie et al. 1992; Groth and Nowick 1992; Li et al. 1995; Pan et al. 1999).

### **Tying genetic linkage map to physical map**

Identification of map position was accomplished by identifying BAC or PAC clones that simultaneously contained a hit from the microsatellite / HvSSR marker. Forward primer sequences of genotyped polymorphic marker(s) (Supplementary Table S1) were used for blast analysis to detect the physical position of the molecular markers and the BAC / PAC clones to which they contained a hit. By way of these co-mapped markers, the map in this study is tied to the physical and sequence map developed by the International Rice Genome Sequencing Project (http://rgp.dna.affrc.go.jp/ ;http://www.usricegenome.org/;http://genome.arizona. edu/fpc/rice/;http://www.gramene. org/) and the principal mapping populations used by the rice scientific community.

#### **Authors' contribution**

Conceptualization of research (SBV, ASK); Designing of the experiments (SBV, ASK, TA); Contribution of experimental materials (SBV, ASK); Execution of field/ lab experiments and data collection (AK, AP, VS, IK); Analysis of data and interpretation (ASK, TA); Preparation of manuscript (ASK, SBV).

#### **Declaration**

The authors declare no conflict of interest.

#### **References**

- Balasubramanian S., Schwartz C., Singh A., Warthmann N., Kim M. C., Maloof J. N. et al. 2009. QTL mapping in new Arabidopsis thaliana advanced intercrossrecombinant inbred lines. PLoS One, **4**: e4318.
- Bonman J. M., Khush G. S., and Nelson R. J. 1992. Breeding rice for resistance to pests. Ann. Rev. Phytopathol., **30**: 507-528. http://dx.doi.org/10.1146/ annurev.py.30.090192.002451.
- Castilla N. P., Leaño R. M., Elazhour F. A., Teng P. S. and Savary S. 1996. Effects of plant contact, inoculation pattern, leaf wetness regime, and nitrogen supply on inoculum efficiency in rice sheath blight. J. Phytopathol., **144**: 187-192. http://dx.doi.org/ 10.1111/j.1439-0434.1996.tb01512.x.
- Channamallikarjuna V., Sonah H., Prasad M., Rao G. J. N., Chand S., Upreti H. C. et al. 2010. Identification of major quantitative trait loci qSBR11-1 for sheath blight resistance in rice. Mol. Breed., **25**: 155-166.
- Che K., Zhan Q., Xing Q., Wang Z., Jin D., He D., et al. 2003. Tagging and mapping of rice sheath blight

resistant gene. Theor. Appl. Genet., **106**: 293-297.

- Eizenga G. C., Lee F. N., and Rutger J. N. 2002. Screening Oryza species plants for rice sheath blight resistance. Plant Dis., **86**: 808-812. http://dx.doi.org/10.1094/ PDIS.2002.86.7.808.
- Gangopadhyay S. and Chakrabarti N. K. 1982. Sheath blight of rice. Review of Plant pathol., **61**: 451-460.
- Groth D. E. and Nowick E. M. 1992. Selection for resistance to rice sheath blight through number of infection cushions and lesion type. Plant Dis., **76**: 721-723.
- Han Y. P., Xing Y. Z., Chen Z. X., Gu S. L., Pan X. B., Chen X. L., et al. 2002. Mapping QTLs for horizontal resistance to sheath blight in an elite rice restorer line, Minghui 63. Yi chuan xue bao. Acta Genetica Sinica, **29**: 622-626.
- Khush G. S. 1977. Disease and insect resistance in rice. Adv. Agron. Acad. Press, **29**: 265-341.
- Kunihiro Y., Qian Q., Sato H., Teng S., Zeng D.-L., Fujimoto K. et al. 2002. QTL analysis of sheath blight resistance in rice (Oryza sativa L.). Yi chuan xue bao. Acta Genetica Sinica, **29**: 50-55.
- Li Z., Pinson S. R. M., Marchetti M. A., Stansel J. W. and Park W. D. 1995. Characterization of quantitative trait loci (QTLs) in cultivated rice contributing to field resistance to sheath blight (Rhizoctonia solani). Theor. Appl. Genet., **91**: 382-388. http://dx.doi.org/ 10.1007/BF00220903.
- Liu G., Jia Y., Prado G. A., Yeater K. M., Mcclung A., Correll J. C. et al. 2009. Mapping auantitative trait loci responsible for resistance to sheath blight in rice. Phytopathol., **99**: 1078-1084. http://dx.doi.org/ 10.1094/PHYTO-99-9-1078.
- Ou S. H. 1985. Rice diseases Commonwealth Mycological Institute, Kew, Surrey.
- Pan X. B., Rush M. C., Sha X. Y., Xie Q. J., Linscombe S. D., Stetina S. R. et al. 1999. Major gene, nonallelic sheath blight resistance from the rice cultivars jasmine 85 and teqing. Crop Sci., **39**(2): 338-346.
- Pinson S. R. M., Capdevielle F. M. and Oard J. H. 2005. Confirming QTLs and finding additional loci conditioning sheath blight resistance in rice using recombinant inbred lines. Crop Sci., **45**: 503-510.
- Prasad B. and Eizenga G. C. 2008. Sheath blight disease screening methods to identify resistant Oryza spp. accessions. Plant Dis., **92**: 1503-1509.
- Sato H., Ideta O., Ando I., Kunihiro Y. and Hirabayashi H. 2004. Mapping QTLs for sheath blight resistance in the rice line WSS2. Breed. Sci., **54**(3): 265-271.
- Savary S. and Mew T. W. 1996. Analyzing crop losses due to Rhizoctonia solani: rice sheath blight, a case study. In: Rhizoctonia Species: Taxonomy, molecular biology, ecology, pathology and disease control (Eds: Carling D. E., Sneh B., Jabaji-Hare S., Neate S. and Dijst G.). Springer Science and Business Media. p. 237-245.
- Savary S., Teng P. S., Willocquet L. and Nutter Jr F. W. 2006. Quantification and modeling of crop losses: A

review of purposes. Annu. Rev. Phytopathol., **44**: 89- 112.

- Savary S., Willocquet L., Elazegui F. A., Castilla N. P. and Teng P. S. 2000. Rice pest constraints in tropical Asia: quantification of yield losses due to rice pests in a range of production situations. Plant Dis., **84**: 357-369.
- Sha X. Y. and Zhu L. H. 1990. Resistance of some rice varieties to sheath blight (ShB). IRRIN. **15**: 7-8.
- Sharma A., McClung A. M., Pinson S. R. M., Kepiro J. L., Shank A. R., Tabien R. E. et al. 2009. Genetic mapping of sheath blight resistance qtls within tropical rice cultivars. Crop Sci., **49**: 256-264.
- Tan C.-X., Ji X.-M., Yang Y., Pan X.-Y., Zuo S.-M., Zhang Y.-F. et al. 2005. Identification and marker-assisted selection of two major quantitative genes controlling rice sheath blight resistance in backcross generations. Acta Genetica Sinica, **32**: 399-405.
- Teng P. S., Torres C. Q., Nuque, F. L. and Calvero S. B. 1990. Current knowledge on crop losses in tropical rice. In: International workshop on crop loss assessment to improve pest management in rice and rice-based cropping systems in South and Southeast Asia 11-17 Oct. 1987, Manila (Filipinas) (No. 633.180631 I61c 1987). International Rice Research Institute, Manila (Filipinas).
- Xiang X. C., Li J. H., Zhang K. Z., Zhao P. and Li P. 2007. Genetic analysis of a rice mutant with resistance to sheath blight and its preliminary gene mapping. J Southwest Sci Tech Univ (Nat Sci). **22**: 76-81.
- Xie Q. J., Linscombe S. D., Rush M. C. and Jodari-Karimi F. 1992. Registration of LSBR-33 and LSBR-5 sheath blight-resistant germplasm lines of rice. Crop Sci., **32**: 507.
- Xie X.-W., Xu M.-R., Zang J.-P., Sun Y., Zhu L.-H., Xu J.-L. et al. 2008. Genetic background and environmental effects on QTLs for sheath blight resistance revealed by reciprocal introgression lines in rice. Acta Agronomica Sinica, **34**: 1885-1893. http:// www.sciencedirect.com/science/article/pii/ S1875278009600134.
- Yeh W. H. and Bonman J. M. 1986. Assessment of partial resistance to Pyricularia oryzae in six rice cultivars. Plant Pathol., **35**: 319-323.
- Yuen J. E. and Forbes G. A. 2009. Estimating the level of susceptibility to Phytophthora infestans in potato genotypes. Phytopathol., **99**: 782-786.
- Zhuang J.-Y., Lin H.-X., Lu J., Qian H.-R., Hittalmani S., Huang N. et al. 1997. Analysis of QTLx environment interaction for yield components and plant height in rice. Theor. Appl. Genet., **95**: 799-808.
- Zou J. H., Pan X. B., Chen Z. X., Xu J. Y., Lu J. F., Zhai W. X. et al. 2000. Mapping quantitative trait loci controlling sheath blight resistance in two rice cultivars (Oryza sativa L.). Theor. Appl. Genet., **101**: 569-573. http://dx.doi.org/10.1007/s001220051517.

# May, 2018] **COL** for sheath blight tolerance in rice (i) (i)

# **Supplementary Table 1.** Tying genetic linkage map to the Physical map (BAC or PAC clones)









Chr. = Chromosome number