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REVIEW

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Current progress on genetic interactions of rice with rice blast and sheath blight fungi

Abstract Analysis of genetic interactions between rice and its pathogenic fungi *Magnaporthe oryzae* and *Rhizoctonia solani* should lead to a better understanding of molecular mechanisms of host resistance, and the improvement of strategies to manage rice blast and sheath blight diseases. Currently, dozens of rice resistance (*R*) genes against specific races of the blast fungus have been described. Among them, ten were molecularly characterized and some were widely used for breeding for genetic resistance. The *Pi-ta* gene was one of the best characterized rice *R* genes. Following the elucidation of its molecular structure, interaction, distribution, and evolution, user friendly DNA markers were developed from portions of the cloned genes to facilitate the incorporations of the *Pi-ta* mediated resistance into improved rice varieties using marker assisted selection (MAS). However, rice blast is still a major threat for stable rice production because of race change mutations occurring in rice fields, which often overcome added resistance based on single *R* genes, and these virulent races of *M. oryzae* pose a continued challenge for blast control. For sheath blight, progress has been made on the exploration of novel sources of resistance from wild rice relatives and indica rice cultivars. A major quantitative trait locus (QTL), named *qSB9-2,* was recently verified in several mapping populations with different phenotyping methods, including greenhouse methods. The ability to identify *qSB9-2* using greenhouse methods should accelerate the efforts on the *qSB9-2* fine mapping and positional cloning.

Keywords blast, *Magnaporthe oryzae*, *Oryza sativa*, *Pi-ta*, rice, *Rhizoctonia solani*, sheath blight

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1 Introduction

Rice is the staple food that feeds half of the world's population, and plays an important role in global food security. Blast disease of rice (*Oryza sativa* L.) caused by the filamentous ascomycetes fungus (*Magnaporthe oryzae,* formerly *Magnaporthe grisea* (T.T. Hebert) M.E. Barr) (Fig. 1), and sheath blight disease caused by *Rhizoctonia solani* Kühn (Fig. 2) have been the two major damaging fungal diseases of rice worldwide (Savary et al., 2000, 2006; Khush and Jena, 2007). Before genetic resistance

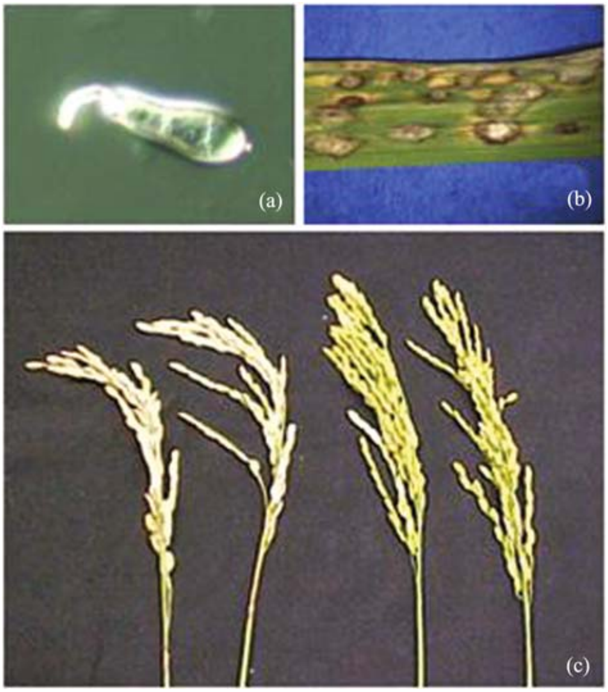

Fig. 1 Blast disease of rice
Note: A germinated conidium of *M. oryzae* (a), typical symptoms of leaf blast (b), and typical symptom of panicle blast (c), left, and panicles at right are healthy (controls). Pictures were taken by a digital camera. Leaf blast was captured 7 days after infection with *M. oryzae*, and panicle blast was collected in an experimental field in Arkansas, USA.

was known, rice farmers knew how to use survived seeds after disease epidemics and knew how to improve cultural practices to reduce crop damage such as adjusting time of planting, rotating crops, avoiding excessive or unbalanced nitrogen application in addition to using survived landrace varieties to prevent blast disease. Current research on blast disease has allowed the identification of several dozens of major blast resistance (*R*) genes; each of them confers race specific resistance. Some of them have been molecularly characterized with user friendly molecular markers for breeding. Sheath blight disease was not a major constraint for rice until the deployment of semi-dwarf varieties in intensified high yielding production system because of the relatively short height of rice plants. Increased deployment of semi-dwarf rice varieties worldwide has made sheath blight one of the most damaging diseases that threaten crop productivity and quality. In contrast to the blast disease, major *R* genes have not been identified in cultivated rice for sheath blight disease. However, genes with major and minor effects have been readily found in rice germplasm, and some of them have been tagged with closely linked molecular markers. In this article, current progress on genetic interactions of rice with *M. oryzae* and *R. solani* will be reviewed.

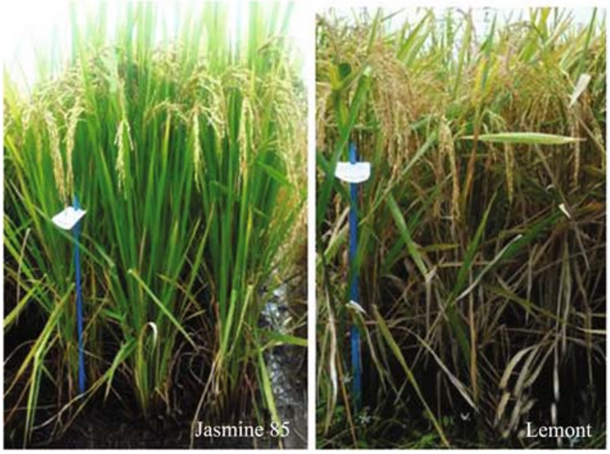

Fig. 2 Sheath blight disease of rice

Note: Rice cultivar Jasmine 85 showing highly resistance, and rice cultivar Lemont showing highly susceptibility. Picture was taken in a sheath blight nursery post anthesis after inoculation with a field isolate from Arkansas, USA.

2 The *O. sativa* - *M. oryzae* interaction

Genetic studies revealed that interaction of *O. sativa* with *M. oryzae* often follows the classical gene-for-gene specificity (Flor et al., 1971; Silue et al., 1992). The *R* gene named as *Pyricularia* (*Pi*) was thought to confer resistance to races of *M. oryzae* containing the corresponding avirulence (*AVR*) genes. *M. oryzae* commences infection immediately after the attachment of a conidium to the surface of rice leaf, and subsequently, germinated conidia produce appresoria for penetration (Fig. 1(a)). Penetration is believed to occur within 24 h depending on strains (Wang et al., 2007a). *M. oryzae* was known to directly penetrate the cell membranes with a high turgor pressure, and growth of mycelia results in subsequent destruction of a living cell (Howard et al., 1991). Before a cell is completely destroyed, mycelia were predicted to move to the next cell via unknown mechanisms including the use of plasmodesmata (Kankanala et al., 2007). Resistance responses mediated by *Pi* genes can be seen within 48 h after infection (Wang et al., 2007a), implying that *Pi* genes act at the frontiers of defense responses. It was then predicted that the Pi proteins are receptors for diverse effectors from the fungus. Some of these effectors are likely encoded by the fungal *AVR* genes.

The *Pi-ta* gene located near the centromere of chromosome 12 encode a predicted cytoplasmic protein with a NBS-LRR domain that recognizes races of *M. oryzae* that contain *AVR-Pita* (Bryan et al., 2000; Jia et al., 2000; Orbach et al., 2000) (Fig 3). *AVR-Pita* is a metalloprotease whose expression in plant is largely unclear. Analysis of natural alleles of the *Pi-ta* gene in international rice collection reveals only one resistant allele, and all other alleles were predicted to be susceptible to races of *M. oryzae* that contain *AVR-Pita* (Wang et al., 2008). The *Pi-ta* allele was widely deployed and was identified in 89 rice germplasm worldwide (Wang et al., 2007b; Wang and Jia, unpublished data). The *Pi-ta* gene in cultivated rice *O. sativa* was predicted to be under strong selection constraint during its domestication (Fig. 4). The frequency of distribution of single nucleotide mutations was examined across the *Pi-ta* region (~2 Mb, Fig. 4(b)) on chromosome 12 with 60 accessions of *O. sativa* and 29 accessions of *O. rufipogon*. In *O. rufipogon* accessions, the significant negative value of Tajima's D ($D = -2.4$, $P < 0.01$) in the genomic region near the *Pi-ta* locus is consistent with recent directional selection (Lee and Jia, unpublished data). Interestingly, the *Pi-ta* gene was predicted to directly recognize the products of *AVR-Pita* in triggering resistance responses (Bryan et al., 2000; Jia et al., 2000). If direct recognition of the fungal effector by an *R* gene product is a general mechanism to activate signaling cascades to prevent further spread of blast fungus, the means of cross-kingdom translocation and processing of products of *AVR* need to be identified (Fig 3). The finding that chaperones in the endoplasmic reticulum (ER) for both *Pi-ta* and *AVR-Pita* mediated resistance and virulence activities of the fungus (Yi et al., 2009) marks an important milestone for the elucidation of molecular mechanisms of *Pi* gene-mediated signal recognition. Another interesting feature at the *Pi-ta* locus is that an unusual large linkage block was identified in several elite rice cultivars due to a large introgression of the *Pi-ta* region into several rice cultivars worldwide. Mechanisms for maintaining a linkage block around the *Pi-ta* gene is unknown but

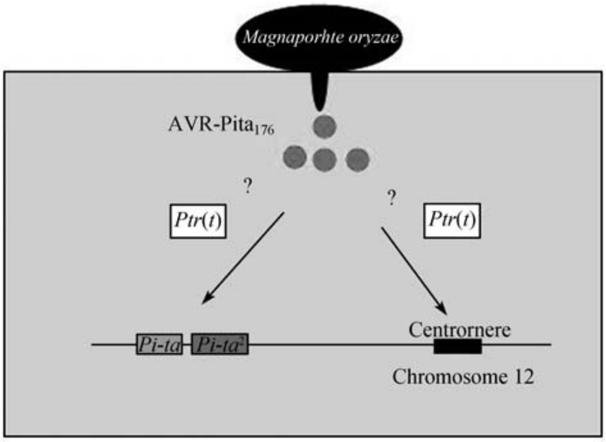

Fig. 3 A model of *Pi-ta* mediated disease resistance showing the existence of additional plant component

Note: *AVR-Pita* was predicted to be translocated to the inside of plant cells in triggering *Pi-ta*/*Pi-ta*^{2}-mediated resistance. *Ptr*(*t*) was identified to be essential for *Pi-ta*/*Pi-ta*^{2}- mediated resistance.

could be due to the effects of the centromere or/and the components in *Pi-ta* resistance resides on different regions of the chromosome 12 (Jia and Martin, 2008; Jia, unpublished data). The presence of a large linkage block would benefit the use of molecular markers for marker assisted *Pi-ta* introgression. Further investigation of molecular mechanisms of recombinant suppression will benefit genetic improvement and enhancement of rice.

As promising alternatives, cloning of other matched pairs of *R* and *AVR* genes may facilitate the investigation of *R* gene-mediated signaling recognition. To this end, nine *Pi*-genes have been molecularly characterized: *Pib* (Wang et al.,1999), *Pi9* (Qu et al., 2006), *Pi2/Piz-t* (Zhou et al., 2006), *Pi-d2* (Chen et al., 2006), *Pi36* (Liu et al., 2007), *Pi37* (Lin et al., 2007), *Pikm* (Ashikawa et al., 2008), *Pi5* (Lee et al., 2009) and *Pit* (Hayashi and Yoshida, 2009) (Fig. 5) and some other *Pi* genes have been tagged with closely linked markers for isolation (Ballini et al., 2008). Cloned *Pi* genes were predicted to encode proteins with the conserved NBS-LRR domains again implying that plant *R* genes possess a common mechanism of signaling recognition or/and transduction. In contrast, cloning and characterization of *AVR* genes in *Magnaporthe oryzae* has lagged behind *R* gene cloning due to the relative difficulties of genetic cross. To date, 25 *AVR* genes in *M. oryzae* were described (Dioh et al., 2000) and six of which were recently cloned: *AVR-Pita*, *AVR1-CO39*, *PWL1*, *PWL2*, *ACE1* and *AVR-Pizt* (Kang et al., 1995; Sweigard et al., 1995; Farman and Leong, 1998; Orbach et al., 2000; Bohnert et al., 2004; Li et al., 2009) (Table 1). *AVR-Pita* encodes a protein with conserved domain indicative of metalloprotease (Orbach et al., 2000). Transient expression of *AVR-Pita* in *Pi-ta* containing plants resulted in hypersensitive cell death, indicative of a gene-for-gene resistant reaction (Bryan et al., 2000; Jia et al., 2000). *AVR-Pita* was not found to express in culture and slight induced expression was observed in susceptible rice plants

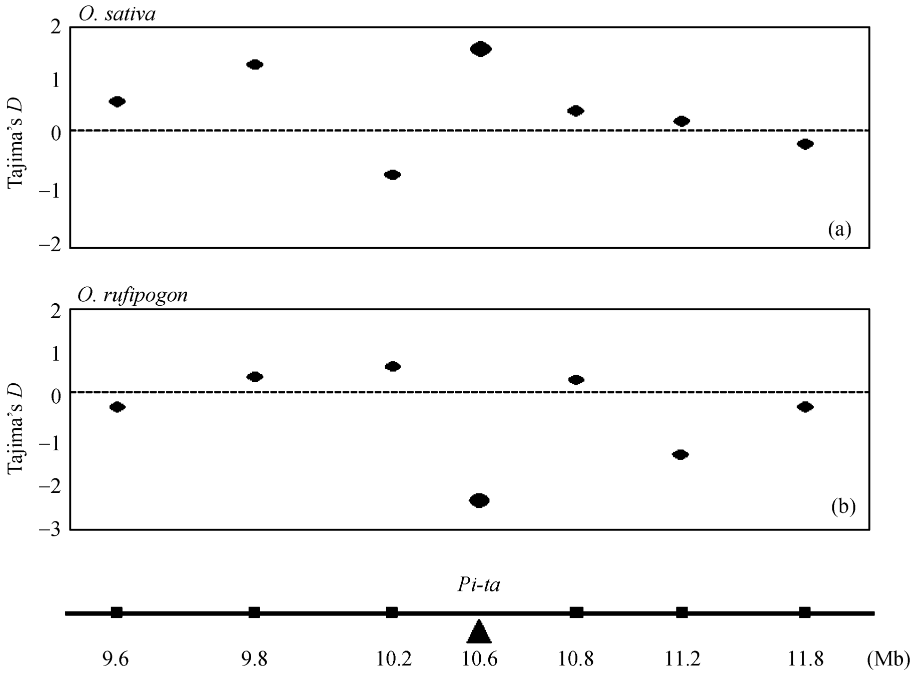

Fig. 4 Levels of Tajima's *D* in accessions of *O. sativa* and *O. rufipogon*

Note: Levels of Tajima's *D* for (a) *O. sativa*, (b) *O. rufipogon*. The arrow indicates the position of the *Pi-ta* gene. The nucleotide diversity of the genomic region around the *Pi-ta* locus in (upper) accessions showing Tajima's *D* statistics of the *Pi-ta* alleles and the six flanking genes spanning the *Pi-ta* locus whose physical locations were shown in the lower panel.

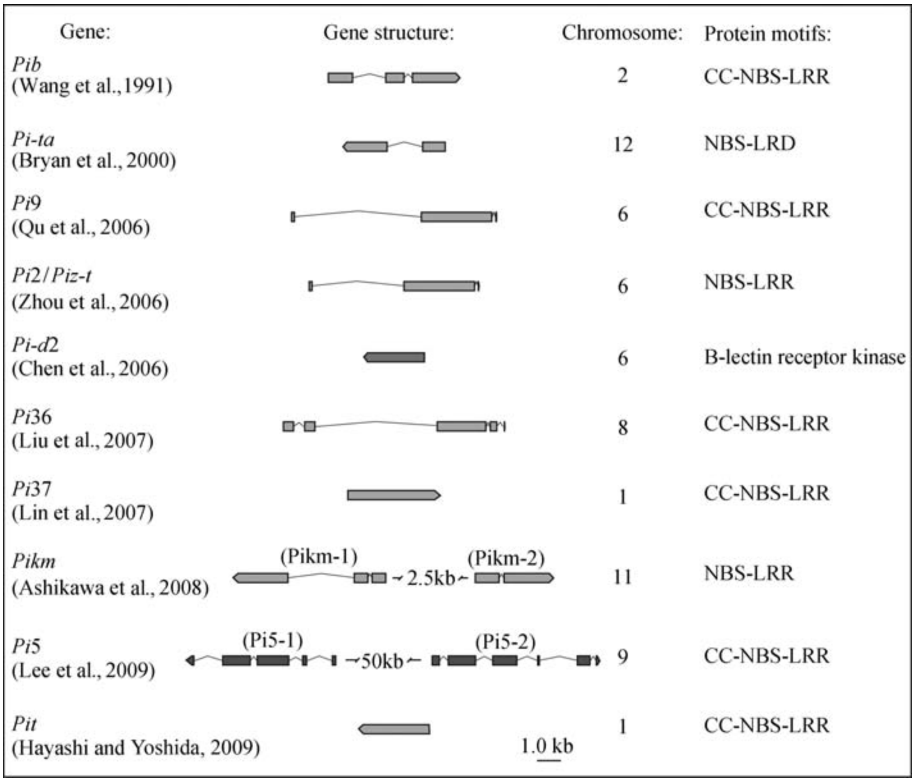

Fig. 5 Diagram of genomic structures of cloned blast resistance genes

Note: Exons are indicated by boxes, and introns by thin lines angled upward. For *Pikm* and *Pi5*, where two genes are required to confer rice blast resistance, their genomic distance in kb is reported at the center of the diagram.

(Jia, unpublished data). Recent surveys of the *AVR-Pita* alleles in *M. oryzae* species complex and in field isolates revealed that transposon insertion at the promoter regions, in the coding region, deletion at 5′ region are likely related to mechanisms to avoid *Pi-ta* recognition and the diversifying selection of *AVR-Pita* allele (Jia et al., 2006; Dai and Jia, unpublished data). Besides *AVR-Pita*, *AVR-Co39*, *ACE1* and *AVR-Pizt* are other three *R* gene-specific *AVR* genes, isolated from *M. oryzae* and all of which have secret signals indicating that their products were secreted out of the fungus (Table 1). Although their modes of actions in plant are still largely unclear, these *AVR* genes all seem to be diversified (Kang et al., 2001; Zhou et al., 2007; Khang et al., 2008).

In the meantime, it has been known that other components in plants are also involved in transducing signals for resistance (Martin et al., 2003). In the *Pi-ta*/*AVR-Pita* interaction, the *Pi-ta* gene was found to require *Ptr*(*t*) for recognition and signaling transduction. *Ptr*(*t*) was recently mapped at the *Pi-ta* region (Jia and Martin, 2008) (Fig. 3). Cloning *Ptr*(*t*) and examining its interaction with both *Pi-ta* and *AVR-Pita*, and other *Pi* and *AVR* gene pairs are being intensely pursued. For a short term benefit, “Perfect” markers can be developed from portions of

Table 1 Summary of molecularly characterized avirulence genes in races of *Magnaporthe oryzae*

name of the *AVR* gene	structural feature	host *R* gene	reference
AVR-Pita	putative neutral zinc metalloprotease	*Pi-ta*	Orbach et al., 2000
AVR1-CO39	putative calcium binding protein	*Co39*	Farman and Leong, 1998
PWL1	a novel protein with 147 amino acids	NA	Kang et al., 1995
PWL2	a glycine-rich, hydrophilic protein with a putative secretion signal sequence	NA	Sweigard et al., 1995
ACE1	a putative hybrid between a polyketide synthase and a nonribosomal peptide synthetase	*Pi33*	Bohnert et al., 2004
AVR-Pizt	a predicted secreted protein	*PiZt*	Li et al., 2009

Note: *PWL1* and *PWL2* are not race specific avriulence genes.

cloned *Pi* genes such as the markers for *Pi-ta* and *Pi-b* (Jia et al., 2002; Jia et al., 2003; Fjellstrom et al., 2004; Jia et al., 2004). These perfect markers are robust and easy to use for marker-assisted selection. In the southern US, rice cultivars with the *Pi-ta* gene are: Katy (Moldenhauer et al., 1990), Drew (Moldenhauer et al., 1998), Kaybonnet (Gravois et al., 1995), Madison (McClung et al., 1999), Cybonnet (Gibbons et al., 2006), Spring (Moldenhauer et al., 2007a), Banks (Moldenhauer et al., 2007b) and Ahrent (Moldenhauer et al., 2007c). The perfect markers for *Pi-ta* (Jia et al., 2002; Jia et al., 2004) were used for the development of some of these cultivars, and were used to verify the *Pi-ta* gene in all *Pi-ta* containing cultivars.

3 The *O. sativa- R. solani* interaction

R. solani belongs to a necrotrophic species complex. Based on anamosis grouping, at least 13 groups infecting different hosts have been identified. The group AG1-IA of *R. solani* causes sheath blight in rice (Wamishe et al., 2007). AG1-IA is one of the largest groups causing the most damages among all other AG groups. Little is known about the pathogenicity and virulence factors of *R. solani.* Although the fungal extracts presumably containing the fungal toxin from *R. solani* was recently shown to induce expression of distinct genetic components in rice, the nature of toxin remains to be identified (Brooks, 2007). Thus far, no major *R* genes have been found to prevent *R. solani* infection; however, host genes each contribute to different levels of resistance as quantitative *R* loci have been mapped onto different chromosomal locations of the rice genome. Several recent accomplishments on the genetic resistance of rice to *R. solani* are summarized below.

Fig. 6 Micro chamber method to evaluate reactions to sheath blight pathogen

Note: Rice seedlings at three to four leaf stages were inoculated with PDA agarose blocks that contain actively grown mycelia (a). An enlarged agarose block that contains mycelia (b) and inoculated plants shown in (a) were covered by soft drink bottles with bottoms removed (c). Disease is rated 3–5 days after inoculation using a rating scale described in Jia et al. (2007).

3.1 Standardized rapid disease evaluation

Traditionally, disease reaction to the pathogen is determined by replicated field experiments. Evaluation of disease reactions in the field is often limited by its location, cost and the minimal time needed for evaluation, and often is the bottle neck for genetic studies and germplasm screening. Recently, breeders in Bangladesh were able to evaluate disease reactions using soft drink bottles and this method has been standardized and improved in a number of labs in the US and South America (Jia et al., 2007) (Fig. 6). In addition, adult plants at early tiller stages were successfully subjected to disease evaluation for mapping quantitative trait loci (QTL) (Liu et al., 2009).

3.2 Candidate genes for sheath blight resistance

Host genes involved in different biochemical pathways have also been identified using DNA microarray and serial analysis of gene expression (Venu et al., 2007). Individual genes known to contribute minor effects in controlling disease are often referred to as defense genes or candidate genes for disease resistance. Rice germplasm with different levels of resistance and rice genes involved in resistance have been recently verified (Manosalva et al., 2009). For example, reduction of germin-like protein (OsGLP) reduced resistance to both rice blast and sheath blight diseases (Manosalva et al., 2009). Some of these differentially and highly expressed genes including the proteins with NBS-LRR domains were identified in some of these QTLs. Using DNA microarray analysis, it was demonstrated that at least 23 rice genes were consistently induced starting from 6 to 10, to 16 h after inoculation (data not shown). Research is underway to utilize these induced genes for developing expression markers to evaluate minor phenotypic responses of rice to the infection by *R. solani*.

3.3 Mapping sheath blight resistance QTLs (ShB-QTL)

A number of chromosomal locations were identified to associate with different levels of sheath blight resistance (Table 2). These QTLs are located on chromosomes 2, 3, 4, 5, 7, 8, 9, 10, 11 and 12. Some of them were confronted with

Table 2 Summary of historical effort on the identification of quantitative trait loci responsible for sheath blight resistance in rice

year	mapping population	molecular markers	ShB-QTLs	chromosome	LOD value$^{a)}$	related to$^{b)}$		references
						PH	HD	
1995	Lemont/Teqing F_4 bulk	113 RFLP	QSbr2a, QSbr3a, QSbr4a, QSbr8a, QSbr9a, QSbr12a	2, 3, 4, 8, 9, 12	⩾2.4	√	√	Li et al., 1995
1999	Lemont/Jasmine 85 DH lines	94 RFLP + SSR	qSB-2, qSB-3, qSB-7	2, 3, 7	>2.0	×	√	Pan et al., 1999
2000	Lemont/Jasmine 85 F_2	118 RFLP + SSR	qSB-2, qSB-3, qSB-7, qSB-9-2, qSB-9-1, qSB-11	2, 3, 7, 9, 11	⩾2.0	×	×	Zou et al., 2000
2002	Zhaiyeqing 8/Jingxi 17 DH lines		qSB-2, qSB-3, qSB-7, qSB-11	2, 3, 7, 11	⩾2.0	√	√	Kunihiro et al., 2002
2002	Zhenshan 97/Minghui 63 $F_{11\text{-}12}$ RILs	RFLP + SSR	qSB-5, qSB-9	5, 9	⩾2.0	–	–	Han et al., 2002
2003	XZX19/4001 (transgenic) F_2	RFLP + SSR	Rsb1	5		–	–	Che et al., 2003
2004	WSS2/Hinohikari (SB resistance from Tetep) BC_1F_1	201 SSR + STS	qSB-3, qSB-12	3, 12	⩾3.0	√	√	Sato et al., 2004
2005	Lemont/Teqing, $F_{10\text{-}11}$ RILs	173 RFLP	qSB-1, qSB-2, qSB-3-1, qSB-3-2, qSB4-1, qSB4-2, qSB-5, qSB-6-1, qSB-6-2, qSB-7, qSB-8-1, qSB-8-2, qSB-9, qSB-10, qSB-12	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12	⩾2.4	×	×	Pinson et al., 2005
2005	Lemont/Teqing F_2	SSR	qSB-9,qSB-11	9, 11	⩾2.0	–	–	Tan et al., 2005
2007	Lemont/Teqing BC_4F_1	Indel	qSB11^{Le}	11		–	–	Zuo et al., 2007
2009	Rosemont/Pecos $F_{2:3}$	149 SSR	Four ShB-QTLs	1, 2, 3, 9	⩾3.6	√	–	Sharma et al., 2009
2009	Lemont/Jasmine 85 F_5 RILs	199 SSR	qShB1, qShB2-1, qShB2-2, qShB3-1, qShB5, qShB6, qShB9-1, qShB9-2	1, 2, 3, 5, 6, 9	⩾2.4	–	–	Liu et al., 2009

Note: a) The LOD values used to declare for a ShB-QTL. b) The sheath blight resistance in rice may or may not be related with plant height (PH) and/or heading date (HD) of rice plants.

QTLs conditioning plant height and heading date in rice (Yano et al., 1997; Yamamoto et al., 2000). Remarkably, the major ShB-QTL *qSB9-2* at the bottom of chromosome 9 was identified and verified in several laboratories using different phenotyping methods (Li et al., 1995; Pinson et al., 2005; Liu et al., 2009). Identification and verification of *qSB9-2* is one of the most significant advancements in genetic resistance to *R. solani*. Two NBS-LRR gene candidates along with dozens of differentially expressed genes were identified at the *qSB9-2* locus. Detecting *qSB9-2* in a mapping population using controlled greenhouse methods makes the fine mapping and cloning of *qSB9-2* more feasible. Near isogenic lines with *qSB9-2* have been developed and more molecular markers are being identified to delimit physical regions harboring *qSB9-2*.

4 Future perspectives

Currently, blast disease has been primarily managed by the use of major *R* genes, and sheath blight has been managed by the use of chemical agents with the deployment of tolerant cultivars in integrated cultural practices. In parallel, research on *Arabidopsis* and other model crops such as tomato has allowed a better understanding of molecular mechanisms of disease resistance (Martin et al., 2003), and resulting knowledge has facilitated the development of strategies to manage both rice blast and sheath blight diseases. Recent studies worldwide have led to a promising future for better genetic control of both rice blast and sheath blight diseases. However, the following questions remain unanswered: 1) the total number of *R* genes against the blast fungus is unknown in rice germplasm. Rice should have plenty of *R* genes or possess elaborate mechanisms to fight against the blast fungus given the fact that the fungus is highly mutable; 2) *AVR* genes in *M. oryzae* were predicted to play important roles in fitness and pathogenicity and also to trigger *R* gene-mediated defense responses; however, cellular targets of *AVR* genes of *M. oryzae* are largely unknown; 3) whether or not there is (are) a master controller(s) in rice either for blast or sheath blight or for both diseases is (are) also unknown, and if so, why has (have) it (they) not been identified? 4) Underlying mechanisms of ineffectiveness of *R* genes to *R. solani* are unknown. However, more new sources of resistance to both *M. oryzae* and *R. solani* have recently been identified from wild rice relatives and they will be used to study the relations of rice with *M. oryzae*, or with *R. solani* (Eizenga et al., 2006; Prasad and Eizenga, 2008; Eizenga et al., 2009). Eventually the above mentioned challenging questions will be addressed with continuous investigation of genetic resistance to both fungal pathogens worldwide. Resulting knowledge will lead to environmentally benign disease management strategies for ensuring global food security.

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