Part II. Section 5

Chapter 1. Molecular biology of disease resistance in rice

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1. Introduction

Plants are continuously exposed to microorganisms in the environment and have evolved multiple layers of preformed and induced defenses to protect themselves against potential pathogens. The use of host plant resistance (HPR) has played an important role in the history of rice development and cultivation. This is particularly true for the last half century, during which time remarkable success has been achieved in combating rice diseases with HPR.

In the early 1960s, rice pathologists and breeders identified large numbers of resistance sources comprising of cultivars from rice germplasm, landraces, and wild rice accessions. Local cultivars with high levels of resistance to rice blast, bacterial blight, the tungro virus complex, and other important diseases were crossed with semidwarf high-yielding breeding materials for transferring their resistance. The derived semidwarf rice cultivars in the 1970s contributed greatly to the success of the Green Revolution in many Asian countries. Genetic mapping of host resistance (***R***) genes in rice began in the early 1970s when a rice genetic linkage map was constructed using trisomic lines (Iwata and Omura 1975). Molecular mapping, however, was initiated only when the RFLP linkage map was constructed (McCouch et al 1988). During the last two decades, tremendous progress has been made in molecular dissection of the host resistance in rice including the mapping and cloning of R genes and characterization of key genes involved in defense signaling and responses. The research has provided not only new insights into the molecular mechanisms of host resistance in rice but has also accelerated breeding of new rice cultivars with broad-spectrum and durable resistance.

The purpose of this section is to review the recent advances in understanding of the molecular biology of rice resistance to major fungal and bacterial diseases. The structure and function of R genes and quantitative trait loci (QTLs) and genes essential for defense signaling are summarized and discussed. This is followed by a discussion of challenges and opportunities for future research on disease resistance in rice.

2. Molecular basis of qualitative resistance

2.1. Concepts of qualitative and quantitative resistance

Based on the effect of resistance on the pathogen and its subsequent reproduction, HPR can be generally classified into two types e.g., qualitative and quantitative resistance. Qualitative resistance, also referred to as vertical resistance or completed resistance, confers strong inhibition against specific pathogen strains, preventing reproduction of the pathogen. Quantitative resistance, synonymous to horizontal resistance or partial resistance, reduces pathogen reproduction within the context of a basic compatible interaction. Usually, qualitative resistance is race-specific and associated with a hypersensitive reaction (HR) at the infection site during the early infection stages. In general, this type of resistance is controlled by a single dominant or recessive R gene and its product can recognize the cognate avirulent protein secreted from the invading pathogen (Flor 1971). In contrast, plants with quantitative resistance or partial resistance

show a low level of infection and delayed disease progress and is controlled by multiple genes or quantitative trait loci (QTLs) with minor effects.

2.2. Structure, function, and evolution of cloned R genes and their interaction with Avr effectors and downstream host proteins in rice

2.2.1. Structure and function of blast* R *genes. Genetic studies of resistance to rice blast began in the early 1960s when I. Goto established the differential system for races of the blast fungus in Japan (Ou 1985). After the first molecular linkage map was published in 1988 (McCouch 1988), efforts to map resistance genes were intensified, especially for those controlling qualitative resistance to rice blast. To date, at least 85 major *R* genes and 49 metaQTLs have been identified and molecularly mapped on the rice linkage map (Ballini 2008, Liu et al 2010) (MB Table 1). The chromosomal positions of the mapped genes are shown in MB Figure 1. Thus far, a total of 21 R genes including 19 major R genes and 2 resistance QTLs have been successfully cloned (MB Table 1). The protein product of the R genes recognize pathogen-secreted avirulence (Avr) proteins either by direct or indirect mechanisms.

Interestingly, except for *Pi-d2*, all R genes encode NBS-LRR proteins, one of the largest conserved families of plant R proteins (Liu et al 2007a), suggesting that a conserved defense mechanism may exist in rice to defend pathogens. The features of the NBS-LRR and non-NBS-LRR genes are summarized below.

2.2.1.1. NBS-LRR genes. *Pi-b* was the first blast R gene that was cloned by a map-based cloning strategy and encodes an NBS-LRR protein (Wang et al 1999). The gene was introgressed independently from two Indonesian and two Malaysian cultivars into various japonica cultivars. This gene confers high levels of resistance to most blast races in Japan. RNA gel blot analysis of the *Pib* family members (*Pib, PibH8, HPibH8-1,* and *HPibH8-2*) revealed that their expression is regulated dramatically by environmental signals such as temperature, light, and water availability, and by chemical treatments (jasmonic acid or JA, salicylic acid or SA, ethylene, and probenazole) (Wang et al 2001).

Pi-ta was mapped within the centromeric region of chromosome 12 and cloned using a map-based cloning strategy (Bryan et al 2000). It encodes a putative 928-amino acid cytoplasmic receptor with a centrally localized NBS and leucine-rich domain (LRD) at the C-terminus. Susceptible rice varieties contain *pi-ta(-)* alleles encoding predicted proteins that share a single amino acid difference relative to the *Pi-ta* resistance protein.

The *Pi2/Pi9* locus is unique because at least 10 R genes (*Pi2, Pi9, Piz-t, Piz, Pigm, Pi26, Pi40, Pi50, Pi2-1,* and *Pi2-2*) are located in a 100-kb genomic region near the centromere of chromosome 6 (Hayashi et al 2004, Wu et al 2005, Qu et al 2006, Zhou et al 2006, Jeung et al 2007, Wang et al 2012, Zhu et al 2012, Jiang et al 2012). These genes have different origins and all confer high levels of resistance to different sets of rice blast strains. *Pi2* and *Piz-t* were introduced from indica rice cultivars 5173 and TKM1, respectively (Mackill and Bonman 1992). *Piz* was originally reported in the U.S. cultivar Zenith (Kiyosawa 1967). *Pi9* was introduced from *O. minuta,* a tetraploid wild species of the genus *Oryza* (Amante-Bordeos et al 1992). *Pigm(t)* was identified in the known blast-resistant cultivar Gumei 4 in China (Deng et al 2006). *Pi26(t)* was identified from the Gumei 4-related cultivar Gumei 2 (Wu et al 2005). The new broad-spectrum *R* gene *Pi40(t)* was introgressed from the EE genome wild rice species *O. australiensis* (Jena et al 1991, Jeung et al 2007). *Pi50(t)* was identified from the cultivar Er-Ba-Zhan(EBZ) with broad-spectrum resistance to rice blast in South China and also mapped in a 59-kb region at the Pi2/9 locus (Zhu et al 2012). *Pi2-1* and *Pi2-2* were recently molecularly mapped at the same locus with *Pi2* from cultivars Jefferson and Tianjingyeshengdao, respectively (Wang et al 2012, Jiang et al 2012).

MB Table 1. Mapping of rice blast major R genes and QTLs.

No.	Gene	Donor(s)	Chr.	Linker marker/gene encoding	References
1	*Pi1*[a]	LAC23	11	RZ536(7.9cM), Npb181(3.5cM)/NBS-LRR protein	Yu et al (1996), Hittalmani et al (2000), Hua et al (2012)
2	*Pi2*[a]	Fukunishiki/ 5173	6	RG64(2.8cM)~R2123(2.7cM) Closely linked to PB8, 3R and 12L/ NBS-LRR protein	Liu et al (2002), Zhou et al (2006)
3	*Pi3*	Pai-Kan-Tao	9	Allelism test; Identical to Pi5	Inukai et al (1996), Jeon et al (2003)
4	*Pi4*	Tetep	12	RG457-RG869	Yu et al (1991), Yu et al (1996),
5	*Pi5* [a]	Moroberekan	9	C1415-S04G03/NBS-LRR protein	Jeon et al (2003), Lee et al (2009c)
6	*Pi6*	Apura	12	RG869-RG397	Yu et al (1996)
7	*Pi7*	Moroberekan	11	RG103A- RG16 S12886 (0 cM)	Bonman and Mackill (1988), Campbell et al (2004)
8	*Pi8*	Kasalath	6	Linkage to *Amp-3* and *Pgi-2*	Pan et al (1996)
9	*Pi9* [a]	*O.minuta/*75-1-127	6	RG64(2.2cM);R2123(2.2cM) pB8(0CM)/NBS-LRR protein	Liu et al (2002), Qu et al (2006)
10	*Pi10*	Tongil	5	RRF6(3.8cM), RRH18(2.9cM); PH18-PF6	Naqvi and Chattoo (1996)
11	*Pi11 (Pizh)*	Zhaiyeqing8	8	BP127A(14.9cM)	Zhu et al (1993)
12	*Pi12(Pih)*	Hongjiaozhan	12	RG869 (5.1cM)	Zheng et al (1996)
13	*Pi12*	Moroberekan	11	---	Inukai et al (1996)
14	*Pi13*	Maowanggu	6	Linkage to Amp-3	Pan et al (1998b)
15	*Pi14*	Maowanggu	2	Linkage to Amp-1	Pan et al (1998b)
16	*Pi15*	GA25	9	CRG3 (0 cM); CRG4 (0 cM)	Lin et al (2007b)
17	*Pi16*	Aus373	2	Linkage to *Amp-1*	Pan et al (1999a)
18	*Pi17*	DJ123	7	Linkage to *Est9*	Rice Genetics Cooperative (1996)
19	*Pi18*	Suweon 365	11	RZ536(5.4cM)	Ahn et al (2000)
20	*Pi19*	Aichi Asahi	12	Closely linkage to *Pi-ta2* or allelic RM27937 and RM1337	Hayashi et al (1998)
21	*Pi20*	IR24	12	XNph88(1.0cM)	Imbe et al (1997)
22	*Pi24*	Zhong156	12	RG241A(0cM), RGA3-620(0cM)	Zhuang et al (2002)
23	*Pi24*[b]	Azucena	1	K5	Sallaud et al (2003)
24	*Pi25*	Yuxi2	1	OSR3(5.8cM)	Yang et al (2001)

MB Table 1. Continued.

No.	Gene	Donor(s)	Chr.	Linker marker/gene encoding	References
25	*Pi25* [b]	IR64	2	RG520	Sallaud et al (2003)
26	*Pi25*	Gumei2	6	A7(1.7cM), RG456(1.5cM)	Zhuang et al (2002)
27	*Pi26* [b]	Azucena	5	RG313	Sallaud et al (2003)
28	*Pi26*	Gumei2	6	B10(5.7cM), R674(25.8cM), Possibly allelic to *Piz*	Wu et al (2005)
29	*Pi27*	Q14	1	RRM259-RM151	Zhu et al (2004)
30	*Pi27* [b]	IR64	6	Linkage to *Est-2*	Sallaud et al (2003)
31	*Pi28* [b]	IR64	10	RZ500, RGA-IR86,identical to Pi11	Sallaud et al (2003)
32	*Pi29* [b]	Azucena	8	RZ617	Sallaud et al (2003)
33	*Pi30* [b]	IR64	11	OpZ11-f, identical to *Pia*	Sallaud et al (2003)
34	*Pi31* [b]	IR64	12	O10-800,identical to *Pita*	Sallaud et al (2003)
35	*Pi32* [b]	IR64;Bala	12	AF6, identical to *Pi12*	Sallaud et al (2003)
36	*Pi33*	IR64	8	Y2643L(0.9cM),RM72(0.7cM); RM3507(0cM)	Berruyer et al (2003), Ballini et al (2007)
37	*Pi36* [a]	Q61	8	CRG3 (0 cM), CRG4 (0 cM); CRG2-RM5647/NBS-LRR protein	Liu et al (2005)
38	*Pi37* [a]	St. No. 1	1	RM543(0.7cM), M319(1.6cM)/ NBS-LRR protein	Lin et al (2007a)
39	*Pi38*	Tadukan	11	RM206,RM21	Gowda et al (2006)
40	*Pi39*	Q15	12	RM27933(0.09cM), RM27940(0.18cM)	Liu et al (2007c)
41	*Pi40*	IR65482-4-136-2-2	6	RM527(1.1cM), RM3330(2.4cM)	Jeung et al (2007)
42	*Pi41*	93-11	12	RM28130 (0 cM)	Yang et al (2009)
43	*Pi41*	GL33	4	RM5586-6679	Cho et al (2007)
44	*Pi42*	28 zhan	6	---	Zhu et al (2007)
45	*Pi42*	DHR9	12	STS5 and RRS6 interval 2cM	Kumar et al (2010)
46	*Pi42*	Zhe733	8	RM310 and RM72	Lee et al (2009b)
47	*Pi43*	Zhe733	11	RM1233 and RM224	Lee et al (2009b)
58	*Pi44*	Moroberekan	11	AF349(3.3cM)	Chen et al (1999)
49	*Pi45*	Moroberekan	4	RM5709- RM3687	Naqvi et al (1996)
50	*Pi46*	SHZ-2	9	---	Liu et al (2009)
51	*Pi46(t)*	H4	11	RM224(1.04cM) and RM27360(1.2cM), K67(0.04cM), T94(0.04cM)	Xiao et al (2011)
52	*Pi47*	Xiangzi3150	11	RM206; RM224 interval 5.5 cM	Huang et al (2011)

MB Table 1. Continued.

No.	Gene	Donor(s)	Chr.	Linker marker/gene encoding	References
53	*Pi48*	Xiangzi3150	12	RM5364;RM7102 interval 0.2cM	Huang et al (2011)
	Pi49	Mowanggu	11		
54	*Pi50*	Er-Ba-Zhan(EBZ)	6	*Pi2/9* allele, GDAP51 and GDAP16	Zhu et al (2012)
55	*Pi54/Pik*^{h}	Tetep	11	RM2191, TRS26, TRS33	Rai et al (2011)
56	*pi55(t)*	Yuejingsimiaoc (YJ2)	8	H2, H66	He et al (2012)
57	*Pi62*	Yashiro-mochi	12	---	Wu et al (1996)
58	*Pi157*	Moroberekan	12	---	Naqvi et al (1996)
59	*Pia* [a]	Aichi Asahi	11	OpZ11-f, RGA-IR14, RM120/ NBS-LRR protein	Causse et al (1994), Okuyama et al (2011)
60	*Pib* [a]	IR24, BL1	2	C379~C2782B; G7010 (0 cM); G7021 (0 cM)/NBS-LRR protein	Miyamoto et al (1996), Wang et al 1999
61	*Pif*	Chugoku 31-1	11	15% recombination value to *Pik*	Shinoda et al (1971), Toriyama et al (1972), Monosi et al (2004)
62	*Pii/Pi5/ Pi3*	Moroberekan	9	S04G03-C1454 (170kb)	Inukai et al (1994)
63	*Pik* [a]	Kusabue/Kanto51/ NBS-LRR protein	11	R543(2.0cM)	Hayashi et al (2006), Zhai et al (2011)
64	*Pikm* [a]	Tsuyuake	11	RM254 (13.4cM)~RM144 (1.2cM)/NBS-LRR protein	Hayashi et al (2006), Ashikawa et al (2008)
65	*Pikp* [a]	K60	11	k3957 (0 cM) Cross K28(0.6cM) and K39(0.07 cM) around 126kb/ CC-NBS-LRR protein	Hayashi et al (2006), Wang et al (2009b), Yuan et al (2010a)
66	*Pikg*	GA20	11	Allelic to *Pik*	Pan et al (1998a)
67	*Piks*	Bengal; M201	11	RM224 (0 cM)	Fjellstrom et al (2004)
68	*Pikh*	Lemont	11	RM224 (0 cM)	Fjellstrom et al (2004)
69	*Pikh*	IRBLkh-K3	11	Cross Kh-45F and Kh-A-3R around 270kb	Xu et al (2008b)
70	*Pish* [a]	Akihikari	1	RM212-OSR3/NBS-LRR protein	Araki et al (2003), Takahashi et al (2010)
71	*Pit* [a]	K59	1	R1613 (0 cM), t256 (0 cM)/ NBS-LRR protein	Rybka et al (1997), Bryan et al (2000), Hayashi et al (2006)
72	*Pita* [a]	Yashiromochi	12	RG241(5.2cM), RZ397(3.3cM); ta3 (0 cM)/NBS-LRR protein	Rybka et al (1997), Bryan et al (2000), Hayashi et al (2006)
73	*Pita-2*	PiNo.4	12	ta3 (0 cM)	Rybka et al (1997), Hayashi et al (2006)

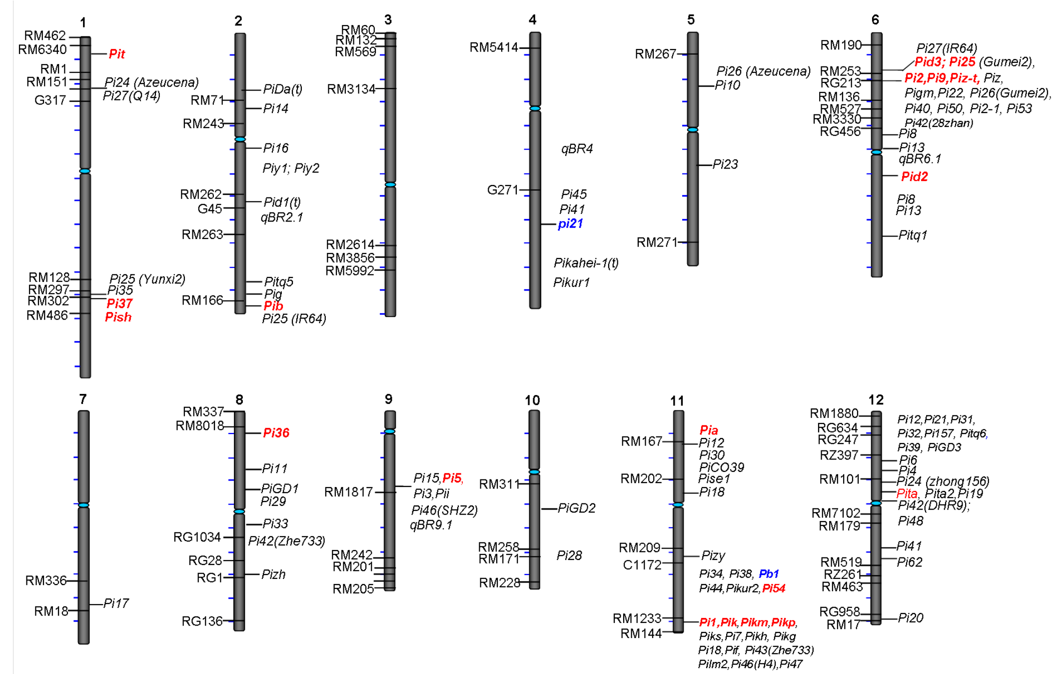
MB Table 1. Continued.

No.	Gene	Donor(s)	Chr.	Linker marker/gene encoding	References
74	*Piz*	Fukunushiki	6	z56592 (0 cM)	Hayashi et al (2006)
75	*Pigm*	Gumei4	6	C5483-C0428; Allelic or closely linked to *Pi2* C5483 ~ C0428(70kb)	Deng et al (2006)
76	*Piz-t* [a]	TKM.1/Toride1	6	RG64(2.8cM)~R2123(2.7cM)/ NBS-LRR protein	Hayashi et al (2006), Zhou et al. (2006)
77	*Pik^h* / *Pi54*[a]	Tetep	11	TRS26(0.7cM)-TRS33(0.5cM)/ NBS-LRR protein	Sharma et al (2005), Sharma et al (2010), Rai et al (2011)
78	*Pid1*	Digu	2	G1314A(1.2cM), G45(10.6cM)	Chen et al (2004)
79	*Pid2* [a]	Digu	6	RM527(3.2cM), RM3(3.4cM)/ B-lectin receptor like kinase	Chen et al (2006)
80	*Pid3* [a]	Digu	6	NBS-LRR	Shang et al (2009)
81	*PiCO39*	CO39	11	S2712(1.0cM)	Chauhan et al (2002)
82	*Pilm2*	Lemont	11	R4-RZ536	Tabien et al (2000)
83	*Pig*	Guangchangzhan	2	RM166(4.0cM), RM208(6.3cM)	Zhou et al (2004b)
84	*Piy*	Yunyin	11	RM202(3.8cM)	Zhang et al (2003)
85	*Piy1*	Yanxian1	2	---	Lei et al (2005)
86	*Piy2*	Yanxian1	2	---	Lei et al (2005)
87	*Pi-zy(t)*	Ziyu44	11	RM206(0cM)	Zhang et al (2009)
88	*Pi2-1*	Tianjingyeshengdao	6	*Pi2/9* allele	Wang et al (2012)
89	*Pi2-2*	Jefferson	6	*Pi2/9* allele	Jiang et al (2012)
90	*pi21* [a,b]	Owarihatamochi	4	G271(5.0cM), G317(8.5cM)/ Proline-containing protein	Fukuoka et al (2001, 2009)
91	*Pi21*[b]	Suweon 365	12	RG869	Ahn et al (1997, 2000)
92	*Pi22* [b]	Suweon 365	6	Possibly allelic to *Pi2* linked with RG213	Ahn et al (1997, 2000)
93	*Pi23* [b]	Suweon 365	5	RM164(19.4cm); RM249(23.9CM)	Ahn et al (1997)
94	*Pi34*[b]	Chubu 32	11	C1172-C30038, RM1596(0cM)	Zenbayashi et al (2002), Zenbayashi-Sawata et al (2007)
95	*Pi35* [b]	Hokkai 188	1	RM1216-RM1003	Nguyen et al (2006)
96	*Pikur1* [b]	Kuroka	4	---	Goto (1988), Monosi et al (2004)
97	*Pikur2* [b]	Kuroka	11	14% recombination value to *la* (lazy growth habit)	Goto (1988), Monosi et al (2004)

MB Table 1. Continued.

No.	Gene	Donor(s)	Chr.	Linker marker/gene encoding	References
98	*Pise1* [b]	Sensho	11	9.5% recombination value to *la*	Goto and Ahmed (1984), Wisser et al (2005)
99	*pb1* [a, b]	Modan	11	C189(1.2cM)/NBS-LRR protein	Fujii et al (2000), Hayashi et al (2010)
100	*Pitq1* [b]	Teqing	6	C236-RG653	Tabien et al (2000)
101	*Pitq5* [b]	Teqing	2	RG520-RZ446b	Tabien et al (2000)
102	*Pitq6* [b]	Teqing	12	RG869-RZ397	Tabien et al (2000)
103	*PiGD1* [b]	SHZ-2	8	XLRfr-8(3.6cM)	Liu et al (2004a)
104	*PiGD2* [b]	SHZ-2	10	r16(3.9cM)	Liu et al (2004a)
105	*PiGD3* [b]	SHZ-2	12	RM179(4.8cM)	Liu et al (2004a)
106	*Pikahei-1(t)* [b]	Kahei	4	RM17494, RM6629	Xu et al (2008a)
107	*qBR2.1* [b]	SHZ-2	2	RM6069	Liu et al (2010)
108	*qBR6.1* [b]	SHZ-2	6	RM584 and RM204	Liu et al (2010)
109	*qBR9.1* [b]	SHZ-2	9	RM24022, LRR-1 and LRR-2	Liu et al (2010)
110	*qBR4-2* [b]	Owarihatamoch	4	RM317, RM3687	Fukuoka et al (2012)

Note: a means the cloned genes; b represents the QTL genes. Chr. is Chromosome.



MB Fig. 1. Chromosomal positions in rice of the blast R genes.

Pi9 was the first gene cloned from the R gene cluster using a map-based cloning strategy (Qu et al 2006). Then, *Pi2* and *Piz-t* were cloned using both the map-based strategy and PCR-based homology cloning (Zhou et al 2006). All three genes encode similar NBS-LRR proteins and are members of a multiple gene family in each haplotype. They share more than a 96% identity in amino acid sequence. Only eight amino acid changes distinguish Pi2 from Piz-t and these changes are confined within three consecutive LRRs. The *Pi2/Pi9* chimeric gene, which is derived from the replacement of the *Pi2* LRR region with that of *Pi9*, confers the *Pi9* resistance specificity, suggesting that the LRR region is the major determinant of the resistance specificity (B. Zhou and G.L. Wang, unpubl.).

The *Pi36* gene was identified on chromosome 8 in the indica rice variety Kasalath. Using both genetic and *in silico* mapping techniques, the gene was fine-mapped within a 17-kb interval and was cloned using a long-range PCR amplification method (Liu et al 2007b). The *Pi36*-encoded protein is composed of 1,056 amino acids, with a single substitution event (Asp to Ser) at residue 590 associated with the resistant phenotype. It is a single-copy gene in rice and is more closely related to the barley powdery mildew R genes *Mla1* and *Mla6* than to the rice blast R genes *Pita, Pib, Pi9,* or *Piz-t*.

The *Pi37* gene in the rice cultivar St. No. 1 was cloned using a similar approach employed for the cloning of *Pi36* (Lin et al 2007a). *Pi37* encodes a 1290-peptide NBS-LRR product and the presence of substitutions at two sites in the NBS region (V239A and I247M) is associated with the resistance phenotype. Semiquantitative expression analysis showed that *Pi37* is only slightly induced by blast infection. Transient expression experiments indicated that the *Pi37* product is restricted to the cytoplasm. The four *Pi37* paralogs are more closely related to maize *rp1* than to any of the currently isolated rice blast *R* genes *Pita, Pib, Pi9, Pi2, Piz-t,* or *Pi36*.

The *Pid3* gene was isolated from the Chinese cultivar Digu using a comparative genomic approach (Shang et al 2009). The *Pi25* gene from the Chinese indica rice cultivar, Gumei 2, was recently cloned and proved to be an allelic gene of *Pid3* (Chen et al 2011). The NBS-LRR gene sequences in the japonica cultivar Nipponbare and indica cultivar 93-11 were analyzed and compared. PCR-based molecular markers specific to the Nipponbare NBS-LRR pseudogene alleles were designed and used for cosegregation analysis with the blast resistance in Digu. The allelic *Pid3* loci in most of the tested japonica varieties were identified as pseudogenes because they have a nonsense mutation at nucleotide position 2208 starting from the translation initiation site. This mutation, however, was not found in any of the tested indica varieties, African cultivated rice varieties, or AA genome-containing wild rice species. These results indicate that the pseudogenization of *Pid3* in japonica occurred after the divergence of indica and japonica subspecies.

The *Pit* gene confers race-specific resistance against *Magnaporthe oryzae* and is a member of the NBS-LRR family in the rice cultivar K59 (Hayashi and Yoshida 2009). The function of *Pit*-mediated resistance is associated with the LTR retrotransposon Renovator that is inserted upstream of its promoter. Compared with the nonfunctional allele in Nipponbare, the functional allele *Pit* in K59 contains four amino acid substitutions and has Renovator inserted upstream. The upregulated promoter activity due to the insertion is essential for *Pit* function, suggesting that transposon-mediated transcriptional activation may play a role in reactivating "silenced" R genes in the rice genome. Recently, the same research group developed PCR-based DNA markers derived from the LTR-retrotransposon sequence and identified five cultivars among 68 rice accessions with the Renovator insertion, all of which have *Pit*-mediated blast resistance (Hayashi et al 2010).

The *Pish* gene was identified by screening 41,119 Tos17 insertional mutants of Nipponbare (Takahashi et al 2010). The screening identified two completed susceptible

mutants that contained the same insertion in the 2nd exon of an NBS-LRR encoding gene (Os01g0782100) located in the genomic region of *Pish*. Complementation showed that this NBS-LRR gene indeed encodes *Pish*, which confers resistance against *M. oryzae* isolates carrying *AvrPish*. Genome analysis indicated that *Pish* and its neighboring three NBS-LRR genes are highly similar and are located in tandem.

The *Pi54* gene, previously known as *Pik^h*, was identified in the indica rice cultivar Tetep and is effective for resistance to *M. oryzae* populations in the northwestern Himalayan region of India (Rai et al 2011). Molecular mapping and cloning showed that *Pi54* locates on chromosome 11 and encodes an atypical NBS-LRR protein. The *Pi54* transgenic plants showed a high level of resistance to multiple *M. oryzae* isolates. Callose deposition analysis indicated that widespread deposition was observed at the infection sites of transgenic resistance plants. The expression of *Pi54* was inducible.

Unlike the R genes discussed in the previous paragraphs, six recently cloned genes (*Pikm, Pi5, Pik, Pik-p, Pi1,* and *Pia*) require two NBS-LRR genes for blast resistance (Ashikawa et al 2008, Lee et al 2009c, Yuan et al 2010a, Okuyama et al 2011, Zhai et al 2011, Hua et al 2012). Fine-mapping at the *Pikm* locus identified two candidate NBS-LRR genes (*Pikm1-TS* and *Pikm2-TS*) (Ashikawa et al 2008). Complementary tests showed that transgenic plants carrying only one of the NBS-LRR genes did not exhibit *Pikm* resistance specificity. The *Pikm* resistance was complemented only when both candidate genes were present in the same plant, suggesting that the *Pikm*-mediated resistance is controlled by two adjacent NBS-LRR genes. Although these two genes (*Pikm1-TS* and *Pikm2-TS*) are not homologous with each other, they both contain all the conserved motifs necessary for an NBS-LRR class gene. *Pikm1-TS* but not *Pikm2-TS* is induced by blast infection**.** A similar case was found at the *Pi5* locus (Lee et al 2009c); transformation experiments showed that two highly homologous NBS-LRR genes, *Pi5-1*, and *Pi5-2,* are required for the *Pi5*-mediated resistance. Gene expression analysis revealed that the *Pi5-1* transcripts accumulate after blast infection, whereas *Pi5-2* is constitutively expressed. Similarly, two highly similar NBS-LRR genes, i.e., *Pik-1* and *Pik-2*, *Pikp-1* and *Pikp-2*, *Pi1-1* and *Pi1-2,* and *SasRGA4* and *SasRGA5* are required for the *Pik-, Pik-p-*, *Pi1-*, and *Pia*-mediated resistance, respectively (Yuan et al 2010a, Okuyama et al 2011, Zhai et al 2011, Hua et al 2012). Understanding how these pairs of R proteins work together and which one interacts with the cognate Avr or other proteins, and whether they form a protein complex during recognition and signal transduction will certainly provide new insight into the molecular mechanisms of this special type of R gene-mediated resistance.

2.2.1.2. The non-NBS-LRR R gene. *Pi-d2* was identified in the Chinese cultivar Digu, a cultivar that is resistant to 156 blast isolates collected from China and Japan and has been widely used in rice breeding programs in China (Chen et al 2004). This gene is located on chromosome 6 and confers resistance to the strain ZB15. The *Pi-d2* gene encodes a novel plasma membrane-associated receptor-like kinase protein with a predicted extracellular domain of a bulb-type mannose-specific binding lectin (B-lectin) and an intracellular serine-threonine kinase domain (Chen et al 2006). A single amino acid difference at position 441 of *Pi-d2* distinguishes the resistant and susceptible alleles. Because of its novel extracellular domain, ***Pi-d2*** represents a new class of plant resistance gene.

2.2.2. Genetic diversity and evolution of blast R genes. Genetic diversity and evolutionary analysis of R genes is essential in understanding the origin and allele distribution of these loci in cultivated and wild rice and will help researchers find new resistance sources in rice germplasm. Several recent studies revealed the allele polymorphism and evolutionary mechanisms of the *Pi-ta* gene in cultivated and wild rice species. Huang et al (2008) analyzed the evolution of the *Pi-ta* locus in 36 accessions of the wild rice *O. rufipogon*. The low nucleotide polymorphism of the *Pi-ta* gene in the wild

species is similar to that in the cultivated species *O. sativa*. The LRR domain has a high nonsynonymous/synonymous ratio, and the amino acid Ala-918 in the LRR domain has a close relationship with the resistant phenotype. Phylogenetic analysis showed that there are two major haplotypes, H1 and H2, in the sequenced alleles. H1 is derived from H2, which is common in wild rice and the ancestral form. A study of the haplotype diversity at the *Pi-ta* locus in 51 accessions of cultivated rice and six wild relatives identified two major clades consisting of 16 different sequences with numerous insertion and deletions (Wang et al 2008). Only one *Pi-ta* resistance allele was identified by rice blast inoculation and molecular analysis. Similarly, research on the intra- and interspecific DNA variations at the *Pi-ta* locus in wild rice (*O. rufipogon*), cultivated rice (*O. sativa*), and two other related wild rice species (*O. meridionalis* and *O. officinalis*) revealed that the resistance *Pi-ta* allele has less variation than the susceptible *Pi-ta* allele (Yoshida and Miyashita 2009). In addition, a more extensive and in-depth sequence analysis of the *Pi-ta* locus and its flanking regions in 159 accessions composed of seven AA genome *Oryza* species identified 33 new *Pi-ta* haplotypes and 18 new Pi-ta protein variants (Lee et al 2009a). Sequence analysis indicated that the *Pi-ta* introgressed fragment is greater than 5.4 Mb in all elite resistant cultivars but not in the susceptible *Pi-ta* cultivars. A 3364-bp transposon in the *Pi-ta* promoter region is associated with the *Pi-ta* resistance. Together, these studies have increased our understanding of the evolutionary mechanism of the *Pi-ta* locus in the *Oryza* genus.

The *Pi2/9* locus contains at least nine NBS-LRR-like genes within a 100-kb region (Qu et al 2006, Zhou et al 2006). Comparative sequence analysis of the complex locus in five rice cultivars revealed contrasting genomic dynamics at the intra- and inter-haplotype levels (Zhou et al 2007). Within each haplotype, the paralogues share as little as 58% similarity in nucleotide sequence, and the 5′ regulatory region and N-terminal intron of the paralogues are quite divergent in sequence and size. In contrast, an obvious orthologous relationship has been maintained among different haplotypes, in which the orthologues are quite similar in sequence and are located at the similar location in the gene cluster. A recent study of the genomic sequences of the *Pi2/9* locus in four wild *Oryza* species representing three genomes (AA, BB, CC, and BBCC) determined that the number of the *Pi2/9* family members in the four wild species ranges from 2 to 12 copies (Dai et al 2010). Although these genes are conserved in structure and categorized into the same subfamily, sequence duplications and subsequent inversions or uneven crossovers were observed, suggesting that the locus in different wild species has undergone dynamic changes. Positive selection was found in the LRR region of most members. Analysis of sequence evolution at the *Pi2/9* locus in cultivated rice cultivars and four wild *Oryza* species has provided new information on the genomic structure and evolution of a multi-*R*-gene cluster in the genus *Oryza*.

2.*2.3. Interaction between R proteins and Avr proteins.* Although six pairs of ***Avr*** and ***R*** genes have been cloned so far (Liu et al 2010), the interactions between the proteins encoded by these two types of genes have not been well established. Transient expression in rice cells of the *Pi-ta* gene together with the *Avr-Pita* induces a resistance response, suggesting an intimate relationship between the two proteins. Truncated Avr-Pita (176) protein binds specifically to the LRD of the Pi-ta protein both in the yeast two-hybrid system and in an *in vitro* binding assay (Jia 2000). However, *in vivo* evidence is still lacking for the AvrPi-ta and Pita protein interaction.

Recently, the *AvrPik* gene was also found to directly interact with Pik and the coiled-coil (CC) domain is required for the *AvrPik-Pik* association (Kanzaki et al 2012). Interestingly, the AvrPik alleles have different interaction specificity to different ***Pik*** alleles. For example, five *AvrPik* alleles (*AvrPik-A, -B, -C, -D, -E*) were identified in a collection of blast isolates. The allele *Pikp* from K60 recognizes *AvrPik-D*, but not *AvrPik-A, -E, -C*. *Pik* from

Kanto51 perceives *AvrPik-D, -E*, but not *AvrPik-A, -C*. *Pikm* from Tsuyuake monitors *AvrPik-D, -E, -A,* but not *AvrPik-C*. These results revealed a significant arms race co-evolution between Avr-Pik and Pik that is maintained by their direct physical interactions.

A map-based cloning strategy was used to clone the *AvrPiz-t* gene, which is the cognate *Avr* gene in *M. oryzae* for the R gene *Piz-t* (Li et al 2009). This gene encodes a predicted 108-amino acid polypeptide with an 18-amino acid secretion signal at the N-terminus. The protein is present in a single copy in the fungus and shows no sequence homology to any known protein. Host targets of *AvrPiz-t* in rice cells were searched for with a yeast-two-hybrid (Y2H) screen using AvrPiz-t as the bait. A total of 12 *AvrPiz-t* interacting proteins (APIPs) were identified. Among them, three APIPs are putative ubiquitin E3 ligases, suggesting that AvrPiz-t may target the ubiquitination-mediated protein degradation pathway when entering rice cells. It is now well established that plants possess a multilayered defense system for protecting themselves against the invading pathogens (Jones and Dangl 2006). Pathogen-associated molecular patterns (PAMPs) are perceived by pattern recognition receptors (PRRs) activating the first level of immune responses called pattern-triggered immunity (PTI). Successful pathogens suppress this level of resistance and are challenged by the second level of immunity facilitated by the recognition of plant resistance (R) proteins by the so-called avirulence (Avr) proteins termed as effectors triggering the effecter-triggered immunity (ETI). However, a complete understanding of the molecular and physiological mechanisms conferring immunity remains elusive. Post-translational modifications result in diversity of the proteins expressed by the genes and this may have relevance for successful plant defense signaling. Recently, APIP6, one of the three RING finger E3 ligases, was demonstrated to be a positive regulator of rice pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) signaling. Silencing of *APIP6* in rice showed a significant decrease of flg22-induced ROS generation and suppression of defense-related genes expression, and increased susceptibility to *M. oryzae.* Surprisingly, the effecter AvrPiz-t inhibits the E3 ligase activity of APIP6; in return, APIP6 ubiquitinates AvrPiz-t *in vitro*. Agroinfection assays reveal that AvrPiz-t and APIP6 are both degraded when coexpressed in *N. benthamiana,* revealing a novel insight that a fungal effecter target suppresses plant PTI by modulating the host ubiquitin proteasome system (Park et al 2012).

2.2.4. Structure and function of bacterial blight R genes. Genetic studies on the inheritance of the bacterial blight R genes began in the late 1980s in Japan and at the International Rice Research Institute (Mew 1987). Currently, 37 bacterial blight R genes have been mapped on different chromosomal loci (MB Table 2; MB Figure 2. Many of these have been widely used in rice breeding programs, and some have been the targets of fine mapping and cloning efforts in the last decade. Thus far, six R genes, i.e., *Xa1,* xa5, *xa13, Xa21, Xa3/Xa26,* and *Xa27,* have been isolated and characterized (MB Table 2). These genes encode various types of proteins that may act in different ways to activate defense responses in rice upon *Xoo* infection.

2.2.4.1. LRR-like receptor kinase R genes Xa21 *and* Xa3/Xa26. *Xa21* was originally identified in the wild species *O. longistaminata* and its subsequent transfer to the cultivated rice IR24 generated the near-isogenic line IRBB21 (Khush et al 1990). *Xa21* was the first R gene to *Xoo* that was cloned in rice (Song et al 1995). The *Xa21* gene encodes a receptor kinase-like protein carrying LRRs in the putative extracellular domain, a single-pass transmembrane domain, and a serine/threonine kinase intracellular domain. Compared with the structure of other cloned R genes from plants, the structure of *Xa21* is unique in carrying both the receptor domain LRR, presumably for recognition, and the kinase domain for subsequent signal transduction (Ronald 1997). Wang et al (1996) inoculated *Xa21*-transgenic plants with isolates from diverse locations to determine

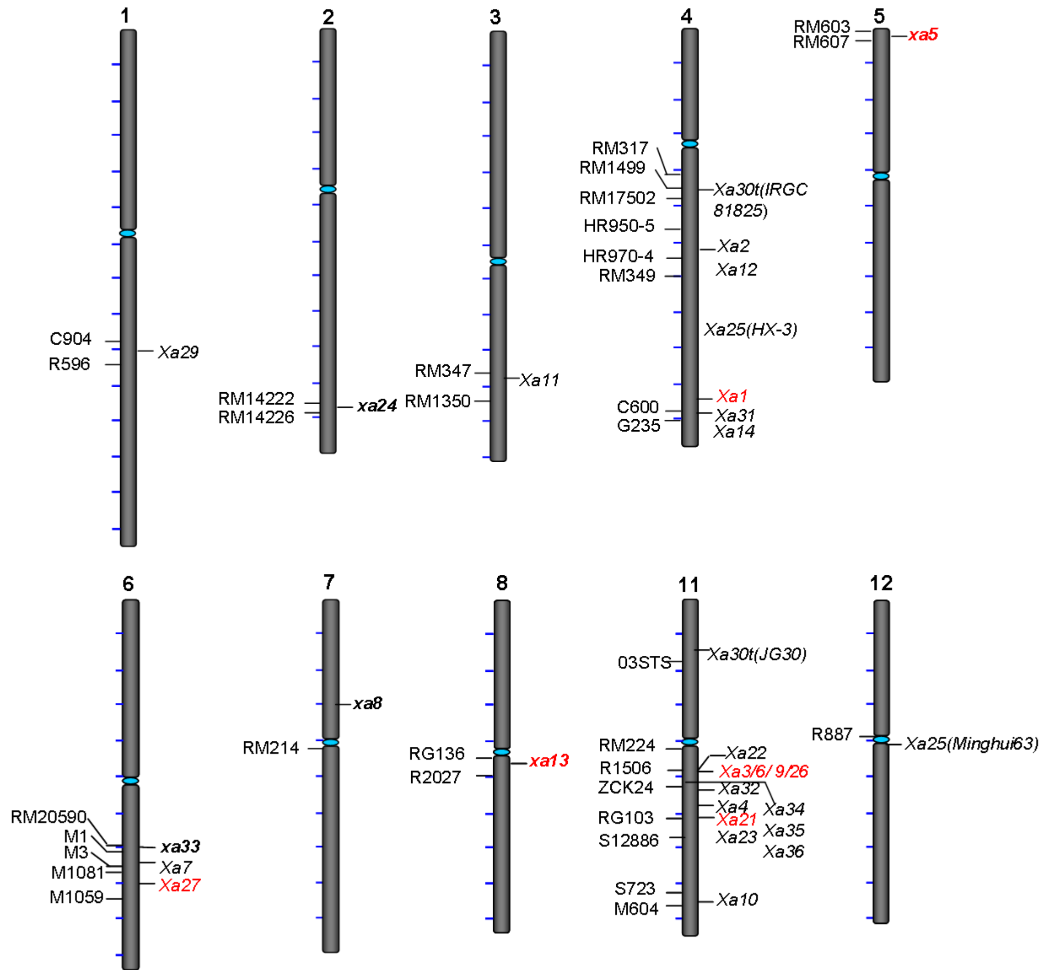
MB Table 2. Mapping of rice bacterial blight R genes.

No.	Gene	Donor	Chr.	Linked marker/Gene encoding	References
1	*Xa1* [a]	IRBB1, Kogyoku	4	Npb235/ NBS-LRR protein	Yoshimura et al (1996), Yoshimura et al (1998)
2	*Xa2*	IRBB2, Wase Aikoku 3	4	V61; E10736, HZR950-5, HZR970-4	He et al (2006)
3	*Xa3/ Xa26/ Xa6/ Xa9* [a]	Zhachang-long, Wase Aikoku,Chukogu-45,IRBB3,IRBB26	11	C481S;Y6855RAR1506, ZCK24/ LRR recptor- like kinase	Sun et al (2004), Xiang et al (2006)
4	*Xa4*	IRBB4	11	Npb181; R1506(0.5cM); S1228(0.5cM)	Robeniol et al (1996), Yoshimura et al (1995), Li et al (1999c)
5	*xa5 a*	DV85, IR1545-248, BJ1, IR291-7,IRBB5	5	RG556,RM603, RM607/ TFIIA Transcription factor	Iyer et al (2004), Jiang et al (2006)
6	*Xa3/ Xa26/ Xa6/ Xa9* [a]	Zhachang-long,Wase Aikoku,Chukogu-45,IRBB3,IRBB26	11	C481S, Y6855RA/LRR recptor-like kinase	Sun et al (2004), Xiang et al (2006)
7	*Xa7*	DV85, DV87,RBB7	6	G1091; M1(2.2cM), M3(0.3cM)	Kaji and Ogawa (1995), Porter et al (2003)
8	*xa8*	P1231129,IRBB8	7	RM214; R M500; RM533	Singh et al (2002)
9	*Xa3/ Xa26/ Xa6/ Xa9* [a]	Zhachang-long,Wase Aikoku,Chukogu-45,IRBB3,IRBB26	11	C481S; Y6855RA/ LRR recptor-like kinase	Sun et al (2004), Xiang et al (2006)
10	*Xa10*	IRBB10,Cas209	11	M491(0.04cM), M604(0cM)	Gu et al (2008)
11	*Xa11*	IRBB11,IR8,RP9-3	3	RM347(2.0 cM), RM1350(7.6cM)	Goto et al (2009)
12	*Xa12*	IRBB12,Java14,Kogyoku	4	--	Ogawa et al (1993)
13	*xa13* [a]	IRBB13	8	RG136; xa13-prom/ Homolog of nodulin MtN3	Yoshimura et al (1995), Chu et al (2006)
14	*Xa14*	IRBB14	4	VAZ190B; RG163; RG620 (20.1cM) and G282(19.1cM)	Taura et al (1991), Tan et al (1998)
15	*xa15*	M41,IRBB15	--	--	Kinoshita et al (1995)
16	*xa16*	IRBB16,IR24,Tetep	--	--	Takahito et al (1989)
17	*Xa17*	IRBB17,Asaminori	--	--	Ogawa et al (1989)
18	*Xa18*	IRBB18,Toyonishiki	--	--	Yamamoto et al (1990)

MB Table 2. Continued.

No.	Gene	Donor	Chr.	Linked marker/Gene encoding	References
19	*xa19*	IRBB19,XM5	--	--	Taura et al (1991), Kinoshita et al (1995)
20	*xa20*	IRBB20,XM6	--	--	Taura et al (1991), Kinoshita et al (1995)
21	*Xa21* [a]	IRBB21, *O. longistaminata*	11	PTA248/LRR-Ser/Thr protein kinase	Ronald et al (1992), Song et al (1995)
22	*Xa22*	IRBB22, Zhangchanglong	11	L363B; P143	Wang et al (2003)
23	*Xa23*	IRBB23, *O. nivara*	11	RG1109; RM254	Zhang et al (2001)
24	*xa24*	IRBB24, Dungano, IR28, DV85, V86,Aus295	2	RM14222(0.07cM); RM14226(0.07cM)	Wu et al (2008c)
25	*Xa25*	IRBB25; Minghui63	12	R887,G1314	Chen et al (2002)
26	*Xa25*	HX-3(Minghui63 mutant)	4	RM6748(9.3cM) and RM1153(3.0cM)	Gao et al (2005)
27	*Xa25*	*O.minuta* (78- 15)	--	---	Ruan et al (2008)
28	*Xa3/ Xa26/ Xa6/ Xa9* [a]	Zhachang-long,Wase Aikoku,Chukogu-45,IRBB3,IRBB26	11	C481S; Y6855RA/LRR recptor-like kinase	Sun et al (2004) Xiang et al (2006)
29	*xa26*	Nep Bha Bong	---	---	Ruan et al (2008)
30	*Xa27* [a]	IRBB27	6	M1081 and M1095/ Novel protein without homolog	Gu et al (2004) Gu et al (2005)
31	*Xa27*	CO39	6	---	Ruan et al (2008)
32	*xa28*	Lota Sail	--	--	Lee et al (2003)
33	*Xa29t*	*O. officinalis*	1	C904, R596	Tan et al (2004)
34	*Xa30t*	*O. nivara* IRGC 81825	4	RM17499 and RM17502; RM317 and RM562	Cheema (2008)
35	*Xa30t*	*O.nivara* (Y238);JG30	11	03STS(2.0cM)	Jin et al (2007)
36	*Xa31t*	Zhachanglong	4	G235 and C600	Wang et al (2009a)
37	*Xa32t*	*O. australiensis*	11	RM27256, RM27274, RM2064, ZCK24,	Zheng et al (2009)
38	*xa33t*	Ba7	6	RM20509	Korinsak et al (2009b)
39	*Xa34t*	Pin Kaset	11	RM224	Korinsak et al (2009a)
40	*Xa35t*	*O.minuta*	11	RM144(0cM), RM7654(1.1cM),RM6293 (0.7 cM)	Guo et al (2010)
41	*Xa36t*	C4059	11	RM2136(3.2cM) RM7443(3.8cM) and RM1233(1.9cM) RM224(1.3cM)	Miao et al (2010)

Note: a represents the cloned genes. Chr. is chromosome.



MB Fig. 2. Bacterial blight R genes mapped on different rice chromosomal loci.

whether the multi-isolate resistance observed for line IRBB21 was due to a single gene or multiple genes at the locus. The transgenic plants expressing the cloned *Xa21* gene conferred resistance to 29 of 32 diverse isolates collected from eight different countries but were susceptible to three isolates from Korea. The resistance spectrum of the engineered lines was identical to that of the donor line, indicating that the single cloned gene is sufficient to confer multi-isolate resistance.

Xa21 is a member of a multigene family cluster, the members of which are designated as A1, A2, B, C, D, E, and F (Song et al 1997). Distinct amino acid differences occur in the LRRs of the *Xa21* gene family members, suggesting that the LRR region is responsible for resistance specificities. To test this hypothesis, Wang et al (1998) investigated the resistance of the transgenic rice plants carrying family members A1, A2, C, D, E, and F to eight *Xoo* races of the Philippines. Members A1, A2, C, E, and F conferred no observable resistance in transgenic plants whereas *Xa21D* conferred the same resistance spectrum as *Xa21*. The resistance level in the *Xa21D* transgenic plants, however, was intermediate to that observed for *Xa21*. The presumed ORF of *Xa21D* encodes a receptor-like molecule lacking the transmembrane and kinase domains due to insertion of a retrotransposon element at the 3' region of the LRR domain, supporting the hypothesis that the *Xa21D* LRR domain is involved in pathogen recognition.

The dominant genes *Xa3* and *Xa26* are the same gene and the locus was renamed *Xa3/Xa26* (Sun et al 2004, Xiang et al 2006). *Xa3* was isolated from the isogenic line IRBB3 while *Xa26* was isolated from the Chinese cultivar Minghui 63. Like *Xa21, Xa3/Xa26* confers high levels of resistance against many *Xoo* strains (Yang et al 2003). *Xa3/Xa26* encodes a protein structurally similar to *Xa21* but with a slightly larger LRR domain (26 vs. 23 repeats). In addition, 87% of the solvent-exposed residues in the predicted extracellular parts of *Xa21* and Xa3/Xa26 differ. Like *Xa21, Xa3/Xa26* is expressed constitutively through development, but unlike *Xa21*'s development-related resistance (Century et al 1999), the resistance level of *Xa3/Xa26* is not dependent on the age of the plant (Sun et al 2004). Uniquely, japonica plants carrying *Xa3/Xa26* regulated by the native promoter showed a broader resistance spectrum and an increased resistance level compared to the indica rice, and unlike resistance in indica rice, resistance in the japonica plants was expressed at all growth stages (Cao et al 2007). The enhanced resistance is associated with an increased expression of *Xa3/Xa26* throughout the growth stages in the japonica plants, which resulted in enhanced expression of defense-responsive genes. Over expression of *Xa3/Xa26* with a constitutive strong promoter also enhanced rice resistance to ***Xoo*** in both indica and japonica backgrounds.

In addition to the *Xa3/Xa26* gene, the locus also contains three paralogs (*MRKa, MRKc,* and *MRKd*) in the indica cultivar Minghui 63 (Sun et al 2006, Cao et al 2007). *MRKa* and *MRKc* are intact genes while *MRKd* is a pseudogene. Constitutive expression of *MRKa* driven by the native promoter in transgenic plants leads to partial resistance to *Xoo*. The kinase domain of *MRKa* partially restores the function in resistance of the truncated *Xa3/Xa26*. The expression pattern for *MRKa* and *MRKc* was similar to that of *Xa3/Xa26*, which was expressed only in the vascular system. Researchers speculated that these two genes might provide templates for generating novel recognition specificity in response to the future evolution of *Xoo*.

For understanding the regulation of resistance during growth of rice plant and the race specificity of resistance to ***Xoo***, four chimeric genes containing different parts of ***Xa3/Xa26*** and ***Xa21*** generated by domain swapping were constructed and transformed into a susceptible rice variety (Zhao et al 2009). The resistance spectra and development-regulated resistance of the chimeric genes against different ***Xoo*** strains together with the wild-type genes were analyzed in the same genetic background. Expression analysis of the transgenes suggested that the gradual increased expression of ***Xa3/Xa26*** and ***Xa21*** contributed to the progressively increased ***Xoo*** resistance during rice development. Although the LRR domains of XA3/XA26 and XA21 are important determinants of race-specific recognition, the juxtamembrane regions of the two R proteins are also important for resistance specificity.

2.2.4.2. NBS-LRR R gene* Xa1**. *Xa1* confers a high level of specific resistance to race 1 strains of ***Xoo in Japan. The gene was identified from the cultivar Kogyoku and mapped onto chromosome 4. *Xa1* encodes an 1802-amino acid NBS-LRR protein (Yoshimura et al 1998). The ***Xa1*** LRR contains six almost perfect repeats, each 93 amino acids long. In each repeat unit, there are six occurrences of a consensus sequence [LXXLXL/IXXN/CXX]. Of the six direct repeat units, the first through the fifth are almost identical and the sixth unit is less similar to the other five units with a 62.7% similarity in amino acid sequence and 73.8% similarity in nucleotide sequence. *Xa1* expression is unique in that it is induced by inoculation with *Xoo* or by wounding, in contrast to the constitutive manner of expression by Xa21.

***2.2.4.3. Transcription factor recessive* R *gene* xa5.** The recessive *xa5* gene is a naturally occurring mutation that is most commonly found in the Aus-Boro group of rice (*Oryzae sativa* L.) varieties from the Bangladeshi region of Asia (Garris et al 2003). The

gene encodes the gamma subunit of transcription factor IIA (TFIIAγ) (Iyer and McCouch 2004). Sequencing of the resistant and susceptible alleles revealed two nucleotide substitutions that resulted in an amino acid change (from valine to glutamic acid at residue 39) between resistant and susceptible cultivars. Inoculations indicated that *xa5* in indica background sustains high levels of bacterial populations compared to that in the susceptible background, yet the resistant plants restrict symptom expression (Iyer-Pascuzzi et al 2008). In contrast, in a japonica background, the bacterial population is 50-fold higher in the susceptible plants than that in the resistant ones. However, both resistant indica and japonica plants delay bacterial movement down the leaf. These results support a model in which *xa5*-mediated recessive resistance is the result of restricted bacterial movement, but not restricted multiplication. TFIIA is known to stabilize the binding of the TATA box-binding protein to the TATA box and plays a role in transcriptional activation, but it has not been associated with plant defense. Therefore, identification of the target genes of *xa5* will increase our understanding of this recessive gene-mediated resistance to *Xoo* in rice.

***2.2.4.4. Nodulin MtN3-like recessive gene* xa13.** The *xa13* gene is fully recessive, conferring resistance only in the homozygous state. It was first discovered in the rice variety BJ1 and was mapped on the long arm of rice chromosome 8 (Chu et al 2006). It encodes a novel protein that has no sequence similarity with any known R proteins, but it shows 50% sequence identity and 68% sequence similarity to the nodulin MtN3 protein in legumes that is induced by Rhizobium in legumes during nodule development (Gamas et al 1996). In the absence of knowledge on the mechanisms of root nodule symbiosis in the presence of active plant immunity (Toth and Stacey 2015), this may serve as a tool in studying the evolutionary aspects of host genes in maintaining a balance between the symbiont and the pathogen. There is no difference in the coding regions of *Xa13* and *xa13* alleles. Although both alleles are normally expressed at low levels in leaves, the functional allele of *xa13* remains expressed at low levels following pathogen attack, whereas the nonfunctional allele is induced. Sequence analysis revealed that all 11 *xa13*-carrying lines had insertion, deletion, or substitution in the promoter region. Thus, the promoter mutations may abolish the induction activity of *xa13* expression by pathogen infection. This allele uniquely plays two divergent roles in disease resistance and in sexual reproductive development of the plant. While the recessive allele confers resistance, the dominant allele governs pollen development (Chu et al 2006). Interestingly, both *xa13* and *Xa13* transcripts are highly expressed in male reproductive tissues and silencing of the locus results in abortive pollen and male sterility, suggesting a mechanistic link between reproductive development and disease resistance in rice.

2.2.4.5. Novel R gene* Xa27.** The *Xa27* locus conferred a high level of resistance to many ***Xoo strains and was mapped on the long arm of chromosome 6 (Gu et al 2004). Except for a 300-bp insertion in the promoter region of the *Xa27* gene in the resistant line, the promoter and coding sequences in the resistant and susceptible plants are identical (Gu et al 2005). The XA27 protein does not contain any known domain in the databases except that a signal-anchor-like sequence is predicted at the amino (N)-terminal region of XA27. Subcellular localization assays showed that the XA27-GFP fusion protein accumulates in vascular elements, the host sites where the bacterial blight pathogen multiplies (Wu et al 2008b). The fusion protein XA27-FLAG is secreted from electron-dense vesicles in the cytoplasm to the apoplast via exocytosis. Deletion of the hydrophobic region or substitution of the triple arginine motif with glycine or lysine residues in the signal-anchor-like sequence abolishes the localization of the mutated proteins to the cell wall and impairs plant resistance, suggesting that the N-terminal signal-anchor-like sequence in *Xa27* is important for ***Xa27***-mediated resistance to *Xoo*.

2.1.5. Evolution of bacterial blight R genes. In the analysis of the evolution of the *Xa21* gene family, seven members, designated A1, A2, B, C, D, E, and F, were cloned and sequenced (Song et al 1997). They are grouped into two classes (*Xa21* and A2) based on sequence similarity. The *Xa21* class contains *Xa21* as well as members D and F. The A2 class contains members A1, A2, C, and E. While nucleotide sequences within each class are nearly identical (98.0% average identity for the members of the *Xa21* class; 95.2% average identity for the members of the A2 class), sequence identity between members of the two classes is low (63.5% identity between *Xa21* and A2). A highly conserved (HC) 233-bp sequence exists among the seven family members such that recombination at the HC region between family members results in a precise swapping of the promoter regions. Large sequence duplications are generated by a presumed unequal crossover event in the intergenic regions. Furthermore, 15 transposon-like elements are located within the gene cluster and two of them are located in the ORFs of members D and E, suggesting that these elements may play a role in the diversification of the *Xa21* family members. These results indicate that duplication, recombination, and transposition contribute to the amplification and diversification of the *Xa21* gene family.

Xa3/Xa26 is also a member of a clustered family of paralogs (Sun et al 2006). Comparisons of the haplotype of this locus in several rice lines revealed an unusually high degree of diversity among paralogs relative to the *Xa21* family. Analysis indicated that point mutations and positive selection are largely responsible for this diversity and may be the most important force in the evolution of new specificities for R genes of this type. The family is formed by tandem duplication followed by diversification through recombination, deletion, and point mutation.

2.1.6. Interaction between bacterial blight R proteins and Avr effectors. The bacterial blight R gene, *Xa27,* and its cognant avirulence gene, *AvrXa27,* are the first gene pair that have been isolated from rice (Gu et al 2005). The transcription activater-like (TAL) type III effecter *AvrXa27* manipulates the function of the corresponding R gene *Xa27* (Gu et al 2005). Only the *Xa27* functional allele is responsive when the rice plants are infected by an *Xoo* strain expressing the cognate *avrXa27* gene. Gu et al (not cited) hypothesized that *AvrXa27* activates transcription of *Xa27* directly or indirectly through activation of a specific host transcription factor. Recently, another study (Gu et al 2009) showed that AvrXa27-activated *Xa27* transcription requires host general transcription factor OsTFIIA gamma 5. The V39E substitution in OsTFIIA gamma 5, encoded by the recessive R gene *xa5* in rice, greatly reduces this activation in plants containing *xa5* and *Xa27* after inoculation with *AvrXa27*-containing strains. The *xa5* gene also causes attenuation in the induction of *Xa27* by *AvrXa27* expressed in rice. Unexpectedly, *xa5*-mediated resistance to *xa5*-incompatible strains is also down-regulated in the *xa5* and *Xa27* plants. These results show that TAL effectors target host general transcription factors to directly manipulate the host transcriptional machinery for virulence or avirulence.

Because like other R receptor proteins, *Xa21* encodes a typical LRR receptor-like kinase, XA21 has been thought to be involved in Avr recognition and defense. Surprisingly, *Xa21* can recognize a PAMP-like molecule Ax21 (activator of XA21-mediated immunity), which is conserved in all analyzed *Xanthomonas* species (Lee et al 2009d). The 194-amino acid protein Ax21 was identified from ***Xoo*** supernatants using LC-MS. A sulfated, 17-amino acid synthetic peptide [axY(S)22] derived from the N-terminal region of Ax21 is sufficient for activity. A co-immunoprecipitation assay showed that XA21 is required for axY(S)22 binding and recognition. This study demonstrates that a PAMP effecter can be an Avr protein and can be recognized by a typical R protein in plants. Conceivably, this forms the first level of response known as pathogen-triggered immunity (PTI) in the multilayered

host immunity as AvrXa21/Xa21 interaction constitutes a PAMP/PRR perception (Nicaise et al 2009) according to the innate immunity models proposed by Chisholm et al (2006) and Jones and Dangl (2006).

3. Molecular basis of quantitative resistance

While the gene-for-gene model concerns genes involved in the initial host-pathogen recognition, many genetic factors contributing to incremental resistance do not entail a high level of specificity. After pathogen infection, the cultivars with quantitative resistance show partial resistance instead of HR. The inheritance of this type of resistance is polygenic and controlled by many genes, often referred to as QTLs. Because of their small and quantitative effects, QTLs are difficult to define genetically. The development of molecular makers, e.g., RFLP, RAPD, AFLP, SSR, and SNP, makes it possible to investigate the inheritance of these complex traits and to locate individual genetic factors associated with them. Many QTLs for major rice diseases have been mapped, and two blast QTLs have been recently cloned. As we are approaching the saturation of the rice genome with high-density SNP markers and applications of advanced mapping and ultra-fast and low-cost sequencing technologies, more QTLs for resistance are likely to be precisely mapped and cloned in the near future.

3.1. Mapping and cloning of rice blast QTLs

The first set of QTLs for rice blast (see Part II, Section 1, Chapter 2) was identified in the durably resistant upland cultivar Moroberekan using a recombinant inbred line (RIL) F_8 population derived from a cross between Moroberekan and the susceptible cultivar CO39 (Wang et al 1994). Since then, at least 20 independent populations have been developed for mapping of rice blast QTLs under greenhouse and field conditions. These studies identified a total of approximately 362 QTL loci distributed over the 12 chromosomes of rice (Ballini et al 2008, Jia and Liu 2011, Liu et al 2010). Considering the redundant or overlapping QTL loci in different mapping populations, a total of 49 metaQTLs were identified as real partial-resistance metaQTLs through meta-analysis (Ballini et al 2008) (MB Table 1).

The candidate gene approach has been successfully used to localize blast QTLs. Liu et al (2004a) used candidate defense response (DR) genes to dissect quantitative resistance in rice using RIL and advanced backcross (BC) populations derived from the blast-resistant cultivar Sanhuangzhan 2 (SHZ-2). Five DR genes, encoding putative oxalate oxidase, dehydrin, PR-1, chitinase, and 14-3-3 protein, account for 60.3% of variation in diseased leaf area (DLA) and colocalized with a resistance QTL identified by interval mapping. Across environments, the average phenotypic contributions of oxalate oxidase, dehydrin, PR-1, chitinase, and 14-3-3 protein in the BC lines to blast resistance were 26.1, 19.0, 18.0, 11.5, and 10.6%, respectively.

Among the identified QTLs, several have been fined mapped in a less than 500-kb genomic interval, and these include *Pb1* (Fujii et al 2000), *pi21* (Fukuoka and Okuno 2001), *Pi34* (Zenbayashi et al 2002, Zenbayashi-Sawata et al 2007), *Pif* (Monosi et al 2004), *Pikur1* and *Pikur2* (Goto et al 1988, Monosi et al 2004), *Pi-se1* (Wisser et al 2005), *Pi35* (Nguyen et al 2006), *Pikahei-1(t)* (Miyamoto et al 2001, Xu et al 2008a), *qBR6.1* (Liu et al 2010), and *qBR9.1* (Liu et al 2010) and *qBR4-2* (Fukuoka et al 2012) (MB Table 1). The recessive partial resistance gene *pi21* located on chromosome 4 was identified from the upland cultivar Owarihatamochi (Fukuoka and Okuno 2001). Recently, the *pi21* gene was successfully cloned via a map-based cloning method (Fukuoka et al 2009). The dominant *Pi21* gene encodes a proline-rich protein that includes a putative heavy metal-binding domain and protein-protein interaction motifs. The wild-type *Pi21* appears to slow plant defense responses, whereas a deletion in its proline-rich motif leads to the gain of resistance and

activation of the defense response against the infection. Although how proline-rich pi21 protein interacts with effectors from the blast fungus and triggers the subsequent defense cascade are unclear, isolation of the partial resistance gene suggested that the *pi21*-mediated partial resistance might represent a novel plant defense mechanism in plants.

The *Pb1* gene, which originated from the indica rice cultivar Modan that has been cultivated for more than 30 years, is the second cloned blast partial resistance gene (Fujii et al 1999, Hayashi et al 2010). It confers more effective resistance at the adult stages and shows stronger panicle blast resistance. Molecular characterization reveals that *Pb1* also encodes an NBS-LRR protein and that the increased expression level of *Pb1* is correlated with the adult and panicle resistance (Hayashi et al 2010). Surprisingly, the increased expression is caused by a duplication of another *Pb1* promoter. A similar phenomenon was observed in the *Pit* gene, whose expression is enhanced by the insertion of a transposon (Hayashi and Yoshida 2009); insertion of a transposon may represent a conserved mechanism of enhancing *R* gene function through promoter modification.

The successful isolation of two blast QTL genes is exciting and promising but several important questions remain to be answered. Are QTLs recognized by a single or by multiple Avr genes? How do the defense pathways mediated by QTLs and by major R genes differ? Is resistance based on a QTL durable and broad-spectrum in a genetic background that lacks major R genes?

3.2. Mapping and cloning of bacterial blight QTLs

Although most of the identified bacterial blight R genes in rice are qualitative genes, several studies have identified some minor resistance QTLs to *Xoo* (MB Table 3). For example, through QTL mapping using an RIL population derived from the cross between Lemont x Teqing, Luo et al (1998) identified a major gene (*Xa4*), 10 QTLs, and nine pairs of epistasis loci conferring partial resistance to three *Xoo* strains. Similarly, using an F_2 population between Minghui63 (containing *Xa3/X26*) and Mudanjiang 8, Zhou et al (2009) identified eight QTLs responsible for bacterial blight resistance in addition to the major *R* gene *Xa3/Xa26*. Several QTLs were also identified using the candidate gene approach. Ramalingam *et al*. used resistance gene analogs (RGAs) and putative DR genes as molecular markers to test for the association with resistance to bacterial blight in the double-haploid population derived from a cross between IR64 and Azucena (Ramalingam et al 2003). They found that six QTLs from Azucena are located on chromosomes 2, 3, 5, 7, and 8 and that each explains at least 8.4% of the phenotypic variation. Similarly, Hu et al (2008) and Kou et al (2010) localized four functionally characterized DR genes (OsWRKY13, OsDR8, OsGH3.8, and OsMAPK6) into regions with previously mapped bacterial blight QTLs from the Minghui63 and Zhenshan97 QTL mapping population.

3.3. Mapping of sheath blight QTLs

Sheath blight, caused by the semisaprophytic fungus *Rhizoctonia solani* Kühn, is a major rice disease responsible for severe reductions in rice grain yield and grain quality worldwide (Lee et al 1983, Rush et al 1996, Xiang et al 2005; see Part II, Section 1, Chapter 3). Because of its semisaprophytic nature, *R. solani* has a wide host range. No rice germplasm with high resistance to *R. solani* has been found (Pinson et al 2005). It is generally believed that resistance to *R. solani* is a typical quantitative trait controlled by QTLs (Sha and Zhu 1989) and that only partially resistant or tolerant varieties exist in rice germplasm (Lee et al 1999, Raina et al 1999, Chen et al 2000, Meena et al 2000, Li et al 2000). Several studies, however, have shown that sheath blight resistance can be controlled by several major genes (Xie et al 1992; Pan et al 1999b,c; Che et al 2003). So far, 37 resistance QTLs have been reported in different mapping populations and are listed

MB Table 3. Mapping of rice bacterial blight resistance QTLs.

QTL	Chr.	Markers	Populaiton	References
QBr2	2	RG520/RZ446b	Lemont and Teqing RIL	Luo et al (1998)
QBr3a	3	C515/RG348		
QBr3b	3	RG482/CDO795		
QBr4a	4	RG214/Ph		
QBr4b	4	RZ69/RG190		
QBr8	8	G104/G1314a		
QBr9	9	RG451/RZ404		
QBr10	10	RG1094f/C16		
QBr11	11	RG1022/RZ525		
QBr12	12	RG91Q/RG341		
XR1a	1	RM488/MRG2143	Minghui63 and Teqing F2	Zhou et al (2009)
XR1b	1	RM472/RM104		
XR3a	3	MRG0338/RM218		
XR3b	3	RM554/MRG5959		
XR4	4	RM401/RM471		
XR5	5	RM509/RM430		
XR7	7	RM18/RM118		
XR9	9	RM215/RM245		
qBB2	2	XLRin12A1/RG437	IR64 and Azucena DH lines	Ramalingam et al (2003)
qBB3-1	3	PK1K2A3/RZ394		
qBB3-2[h]	3	CDO/Oxalate oxidase		
qBB5	5	XLRfrA1/RA70		
qBB7	7	RG477/NLRin1212		
qBB8	8	rNBS35/CDO99		

Note: Chr. is chromosome.

in MB Table 4 (www.gramene.org). Because of difficulties in phenotyping rice plants to *R. solani* in growth chambers and in the field, none of the identified QTLs has been fine-mapped or cloned.

3.4. Bacterial leaf streak

Bacterial leaf streak (see Part II, Section 2, Chapter 2), caused by *Xanthomonas oryzae pv. Oryzicola (Xoc)*, is another rice disease and may cause 10–20% yield reduction (Xu and Qian 1995, Han et al 2008). The disease was first observed in Guangdong, China, and is widely distributed in rice production areas of southern China including Guangdong, Fujian, Hunan, Jiangxi, and Guangxi provinces (Fan and Wu 1957, Han et al 2008). Resistance to bacterial leaf streak is generally controlled by QTLs. A total of 11 resistance QTLs have been mapped in the rice genome and the linked markers and mapping populations are listed in MB Table 5 (Tang et al 2000, Zheng et al 2005, Chen et al 2006, Han et al 2008). In the

MB Table 4. Mapping of rice sheath blight resistance QTLs.

QTL	Chr.	Markers	Populaiton	References
QSbr2a	2	RG654-RZ260	Teqing(R) and Lemont(S) F4	Li et al (1995)
QSbr3a	3	RG348-RG944		
QSbr4a	4	RG143-RG214		
QSbr8a	8	RG20-RG1034		
QSbr9a	9	RG910a-RZ777		
QSbr12a	12	RG214a-RZ397		
qSB-1	1	RG532x	Teqing(R) and Lemont(S) F2:10	Pinson et al (2005)
qSB-2	2	C624x		
qSB-3-1	3	RG348x		
qSB-3-2	3	RZ474		
qSB-4-1	4	RG1094e		
qSB-4-2	4	RZ590x		
qSB-5	5	Y1049		
qSB-6-1	6	C		
qSB-6-2	6	RZ508		
qSB-7	7	C285		
qSB-8-1 from Lemont	8	G104		
qSB-8-2 from Lemont	8	R662		
qSB-9	9	RZ404		
qSB-10	10	RG561		
qSB-12	12	G1106		
qSB-5	5	C624-C246; C246-RM26	Minghui63 and Zhenshan97B (RIL)	Han et al (2002)
qSB-9	9	C472-R2638; RM257-RM242		
Rsb1 Major gene	5	RM39200 OPN16 2000	4011(R) and Xiangzaoxian19 (S) F2	Che et al (2003)
qSB-3	3	RM3856	WSS2/Hinohikari/ Hinohikari BC1F1	Sato et al (2004)
qSB-12	12	RM1880		
qSB1	1	RM1339	Rosemont (semi-dwarf, SB susceptible) and Pecos (tall, SB resistant)	Sharma et al (2009)
qSB2	2	RM3685		
qSB3	3	RM7072		
qSB9	9	RM3823		

MB Table 4. Continued.

QTL	Chr.	Markers	Populaiton	References
qShB1	1	RM1361-RM104	Lemont and Jasmine 85 NIL	Liu et al (2009)
qShB2-1	2	RM424-RM5427		
qShB2-2	2	RM112-RM250		
qShB3-1	3	RM16-RM426		
qShB3-2	3	RM5626-RM426		
qShB3-3	3	RM514-RM85		
qShB5	5	RM507-RM7349		
qShB6	6	RM435-RM190		
qShB9-1	9	RM409-RM257		
qShB9-2	9	RM215-RM245		
Rh2/qSB-2	2	G243-RM29	Jasmine 85 (R) and Lemont (S) F2	Pan et al (1999c), Zou et al (2000)
Rh3/qSB-3	3	R250-C746		
Rh7/qSB-7	7	RG30-RG477		
qSB9-2	9	RG570–C356		
qSB9-1 from Lemont	9	C397–G103		
qSB-11 from Lemont	11	G44–RG118		

Note: Chr. is chromosome.

absence of resistance sources of rice to *Xoc*, attention has turned to nonhost resistance available in maize (Zhao et al 2004a). The maize line B73 possessing a single dominant gene *Rxo1* expressing resistance to its pathogen, *Burkholdeira andropogonis* also responds hypersensitively to *Xoc* (Zhao et al 2004b). An avirulence gene, avrRxo1 has been identified in the *Xoc* genomic library and the avrRxo1-Rxo1 interaction has been found to be dependent on type III secretion system (Zhao et al 2004 b). Rice lines transformed with the cloned *Rxo1* are resistant to bacterial leaf streak and the transgene has been found to be inherited as a dominant gene (Zhao et al 2005, Xie et al 2007).

4. Molecular dissection of defense signaling pathways

To prevent pathogen infections, plants have evolved with two innate immune pathways that rely on pattern-recognition receptors (PRRs) and R proteins (Chisholm et al 2006, Jones and Dangl 2006). The first line of defense depends on the recognition of PAMPs by plant transmembrane PRRs. This recognition activating defense response is called PAMP-triggered immunity (PTI). During the coevolution of plant and pathogen, pathogens have evolved effecter proteins that they can secrete into the cytoplasm of host cells to suppress plant PTI and cause disease. In response, plants have evolved the ability to recognize proteins secreted by the pathogens into the plant cell called effecter proteins (also called avirulence proteins) via a secretory as part of an effecter-triggered immunity (ETI) mechanism. In rice, remarkable progress has made in systems characterizing the components in PTI- and ETI-mediated defense signaling pathways through multiple strategies including forward and reverse genetics and genomic approaches (MB Table 6).

MB Table 5. Mapping of rice bacterial leaf streak resistance QTL mapping.

QTL	Chr	Markers	Populaiton	References
qBlsr1	1	C49 and Xpsr56	Acc8558(R) and H359(S) F2 and RIL	Tang et al (2000), Han et al (2008)
qBlsr2	2	C1419		
qBlsr3a	3	Xpsr145B and Xpsr575		
qBlsr3b	3	P76/M22-1		
qBlsr3c	3	C63A and P76/M22-16		
qBlsr3d	3	Xpsr301		
qBlsr4a	4	C975		
qBlsr4b	4	Xpsr488		
qBlsr5a(fine mapped)	5	R830 RM153 and RM159(290kb)		
qBlsr5b	5	C624 and R1553		
qBlsr11	11	P19/M76-12		
qBLS	2	RM179-RM154	Jiafuzha (R) and Minghui86 (S) F2	Zheng et al (2005)
qBLSR-11-1	11	RM120 and RM441	Dular(R)/Ballila(S) Dular(R)/IR24(S) F2	Chen et al (2006)

Note: Chr. is chromosome.

4.1. Forward genetic approach for dissecting the defense signaling pathways

Hypersensitive reaction (HR) can induce a rapid death of cells, more appropriately termed as programmed cell death (PCD), surrounding the infected area and thereby restrict pathogen invasion of neighboring cells (Greenberg and Yao 2004). HR usually activates a set of innate defense signals with a series of biochemical events such as ion fluxes (McDowell and Dangl 2000), generation of reactive oxygen species (ROS) (Van Breusegem and Dat 2006), release of nitric oxide (NO) (Zago et al 2006), and cross talking of hormone pathways (jasmonic acid, salicylic acid, ethylene, and sphingolipid) (Love et al 2008). Therefore, HR is used as an important visible phenotype marker for identifying critical components in plant defense signaling pathways (Lorrain et al 2003). Mutants that display a spontaneous HR-like cell death and often constitutively promote plant defense responses are collectively called lesion mimic (LM) mutants. In the last 10 to 20 years, many such mutants have been identified in *Arabidopsis* (Lorrain et al 2003), maize (Gray et al 1997), barley (Wolter et al 1993), and rice (Takahashi et al 1999, Yin et al 2000, Wu et al 2008a) and these mutants have provided insight into the molecular basis of HR-associated cell death in plants.

In rice, many LM mutants have been generated by the use of different mutagenesis methods such as chemical, physical or T-DNA insertion methods (Mori et al 2007, Takahashi et al 2007, Wu et al 2008a). Five genes controlling cell death have been recently cloned in rice. The *spl7* (*spotted leaf 7*) mutant was determined to be susceptible to both *M. oryzae* and *Xoo* and to exhibit a strong suppression of several defense-related genes (Yamanouchi et al 2002). Map-based cloning revealed that the *spl7* gene encodes a heat-stress transcription factor (HSF) (Yamanouchi et al 2002). One base substitution causing a change from tryptophan to cysteine in the HSF DNA-binding domain of the mutant

MB Table 6. Defense related signaling components involved in rice disease resistance.

Gene	Protein encoding	Biological function	References
Spl7	Heat stress transcription factor	Negative regulator of rice cell death	Yamanouchi et al (2002)
Spl11	U-Box E3 ligase	Negative regulator of rice cell death and defenese	Zeng et al (2004)
Spl18	An acyltransferase-like protein	Negative regulator of rice cell death and defenese	Mori et al (2007)
Spl28	Golgi apparatus localizing clathrin-associated adaptor protein complex 1, medium subunit micro 1 (AP1M1)	Negative regulator of rice cell death and defenese	Qiao et al (2010)
OsPti1a	Phosphorylation/ dephosphorylation protein, a homolog of tomato *Pti1*	Negative regulator of rice cell death and rice RAR1-dependent defense responses	Takahashi et al (2007)
OsRac1	Small GTPase protein	Positive regulator to rice bacterial blight (BB) and rice blast fungus (RB)	Kawasaki et al (1999), Ono et al (2001)
OsSGT1	For suppressor of the G2 allele of skp1	Race-specific resistance to rice BB and RB; component of OsRac1 complex	Thao et al (2007), Wang et al (2008c)
OsRAR1	A novel eukaryotic CHORD-I and CHORD-II (Cys- and His-rich domains) domain containing zinc-binding protein	Race-specific resistance to rice BB and RB; component of OsRac1 complex	Thao et al (2007), Wang et al (2008c)
OsRACK1	For Receptor for Activated C-Kinase 1	Interact with OsRACK1 to positvely regulate ROS and rice basal resistance	Nakashima et al (2008)
OsRbohB	Homologs of the gp91phox subunit, for respiratory burst oxidase homolo	Interact with OsRac1 to regulate NADPH oxidase activity to controll ROS generation	Wong et al (2007)
Hsp70/ HSP90	Heat shock proteins	Form a complex with OsRac1, Hsp90 is essenstial for OsRac1 complex formation	Thao et al (2007)
OsCCA1	Cinnamoyl-CoA reductase	Interacts with OsRac1 to control lignin synthesis through regulation of both NADPH oxidase and OsCCR1 activities during defense responses.	Kawasaki et al (2006)
OsCERK1	Rice chitin receptor kinase	Interact with Hti/Hop involved in OsRac1 mediated immune signaling	Chen et al (2010b)
Hti/Hop	Hsp70/90 cochaperone Hop	Positive regulator for RB resistance	Chen et al (2010a)

MB Table 6. Continued.

Gene	Protein encoding	Biological function	References
MAPK6	Mitogen-activated protein kinase	Interact with OsRac1 involved in rice basal resitance	Lieberherr et al (2005)
OsMPK6	Mitogen-activated protein kinase	Negative regulator for rice BB reistance	Yuan et al (2007)
XB3	Ankyrin repeat domain containing protein	E3 ligase, interact with Xa21 positively regulate resisatance to rice BB	Wang et al (2006)
XB15	Protein phosphatase 2C (PP2C)	Negative regulator for rice Xa21 mediated resistance	Park et al (2008)
XB24	ATPase protein	Negative regulator for rice Xa21 mediated resistance	Chen et al (2010c)
XB10	Transcprition factor WRKY62	Negative regulator for rice Xa21 mediated resistance	Peng et al (2008)
OsCOT1/ OsCOT5	Copper transproter protein	Interact with Xa13 to regulate plant copper distribution and resistance to rice BB	Yuan et al (2010b)
OsWRKY13	Transcription factor	Positive regulator to rice disease resistance	Qiu et al (2007)
WRKY45	Transcription factor	Positive regulator to rice blast resistance	Shimono et al (2007)
OsGH3.8/ 3.1/3.2	IAA–amino synthetase	Positve regulator for rice BB and RB	Ding et al (2008), Domingo et al (2009), Fu (2010)
OsNH1	Rice NPR1 homologue with a bipartite nuclear localization sequence and two potential protein–protein interaction domains: an ankyrin repeat domain and a BTB/POZ domain	Positive regulator for rice BB resistance	Chern et al (2005b)
OsNRR	Interacting protein of rice NH1, for negative regulator of resistance, no significant homology to known protein domains with a proline rich in C-terminal	Negative regulator of rice disease resitance	Chern et al (2005a)
OsGLP	Germin-like protein	Positive regulator of RB and sheath blight resistance	Manosalva et al (2009)
Os-11N3	NODULIN3 (N3) gene encoding protein	Negative regulator of rice BB resistance	Antony et al (2010)

allele leads to a significant decrease in the *spl7* transcription level. The second molecularly characterized LM mutant, *spl11*, displays a nonrace-specific resistance to both *M. oryzae* and *Xoo* with a spontaneous HR-like cell death (Yin et al 2000). Molecular cloning revealed that *spl11* encodes a U-Box and Armillo domain-containing protein with an E3 ligase, suggesting the involvement of ubiquitination-mediated protein modification in rice cell

death and defense responses (Zeng et al 2004). The third molecularly characterized LM mutant, *spl28*, also displays a phenotype of LM formation after flowering and enhanced resistance to both *M. oryzae* and *Xoo*. Map-based cloning revealed that *spl28* encodes a Golgi apparatus localizing clathrin-associated adaptor protein complex 1, medium subunit micro 1 (AP1M1), which is involved in the post-Golgi trafficking pathway (Qiao et al 2010).

The LM genes described in the previous paragraph were characterized by map-based cloning strategies, but two other LM genes, *Spl18* (Mori et al 2007) and *OsPti1a* (Takahashi et al 2007), were characterized by T-DNA insertional mutagenesis. The ***Spl18*** gene was identified in a set of rice activation-tagging mutants. The activation of ***Spl18*** causes a spontaneous cell death with enhanced resistance to both ***M. oryzae*** and ***Xoo***. It encodes an acyltransferase-like protein, designated OsAT1, which shows sequence similarity to an acyltransferase whose expression is induced by HR in tobacco. Knock-out of the ***OsPti1a*** gene by T-DNA insertion causes a constitutively lesion mimic phenotype and enhanced resistance to ***M. oryzae*** with activation of defense response-related genes. The ***OsPti1a*** gene encodes a phosphorylation / dephosphorylation protein and is a homolog of tomato ***Pti1*** (Takahashi et al 2007). It is noteworthy to mention that the rice ***OsRAR1*** can compromise the defense responses induced in the ***Ospti1a*** mutant, suggesting that OsPti1a is a negative regulator of rice RAR1-dependent defense responses.

Isolation of these five lesion mimic-controlling genes in rice has revealed that a diverse set of proteins is involved in rice defense signaling pathways. How these components function in rice PTI and ETI-mediated defense pathways and how they interact with PPRs and R proteins remain unclear.

A mutagenesis approach was used to identify ***Pi-ta***-expressing susceptible mutants (Jia and Martin 2008). The identified mutant ***Ptr(t)*** does not alter recognition specificity to another resistance gene ***Pi-k(s)***, suggesting that the ***Ptr(t)*** locus is more likely specific to ***Pi-ta***-mediated signal recognition. Genetic analysis revealed that ***Ptr(t)*** acts as a single dominant gene and is closely linked to the ***Pi-ta*** gene within a 9-Mb genomic region.

4.2. Reverse genetic approach for dissecting the defense signaling pathways

4.2.1. OsRac1 defensome-mediated PTI and ETI defense pathway. The small GTPase protein OsRac1 positively regulates rice programmed cell death through activating ROS generation (Kawasaki et al 1999). Constitutive activation of *OsRac1* leads to enhanced resistance to both *M. oryzae* and *Xoo* (Ono et al 2001), revealing the critical roles of OsRac1 in regulating cell death and defense responses in rice. Subsequent investigations indicated that OsRac1 functions as an important integrator of upstream receptors such as PRR- and R protein-mediated PTI and ETI defense signaling pathways, and diverse downstream defense response proteins. In the upstream, both R proteins and PRRs are required for the activation of OsRac1-mediated resistance. For example, OsRac1 is required for the both Pia- and Pit-mediated defense responses (Chen et al 2010a, Kawano et al 2010a,b). Further, OsRac1 interacts directly with the NBS domain of rice blast R protein Pit at the plasma membrane. OsRac1 also contributes to Pit-mediated ROS production as well as to the HR and is required for Pit-mediated disease resistance in rice (Kawano et al 2010a,b), suggesting a critical role of OsRac1 in rice R gene defense signaling. Additionally, Ono et al (2001) indicated that OsRac1 acts as a downstream molecule of the PRR RLK pathway, although no RLK directly interacts with OsRac1. Chen et al (2010a) determined that the rice chitin receptor OsCERK1 interacts with Hsp90 and its cochaperone Hop/Sti1 in the endoplasmic reticulum (ER). Hop/Sti1 and Hsp90 are required for efficient transport of OsCERK1 from the ER to the plasma membrane.

Genetic analysis showed that Hop/Sti1 is required for chitin-triggered immunity and resistance to the rice blast fungus. These results suggest that the Hop/Sti1-Hsp90

chaperone complex plays an essential role in the maturation and transport of PRRs and may link PRRs and Rac/Rop GTPases during defense activation. After receiving the upstream activating signaling, OsRac1 promotes several downstream defense pathways. For example, Wong et al (2007) found that OsRac1 directly targets the rice Rboh (respiratory burst oxidase homolog) proteins RbohB to activate the critical enzyme NADPH oxidase for ROS production. OsRac1 also targets the mitogen-activated protein kinase OsMAPK6 that is induced by sphingolipid elicitors and further promotes the bHLH transcription factor RAI1 to activate the expression of the defense-related genes PAL and OsWRKY19 (Lieberherr et al 2005, Kim et al 2012). The Cinnamoyl-CoA reductase OsCCR1 targeted by OsRac1 is involved in the lignin-mediated defense response (Kawasaki et al 2006). In addition, the OsRac1 complex-mediated resistance is also required for the collaboration of several molecular chaperones and cochaperones such as HSP90, Hop/Sti1, RAR1, and SGT1 (Kawano et al 2010a,b). Based on these results, Kawano et al (2010a,b,) proposed a model to decipher the OsRac1 complex-mediated defensome. In this model, the defensome is composed of four different groups of proteins, including two types of immune receptors (PRRs and R proteins), chaperones, and cochaperones (SGT1, RAR1, HSP90, HSP70, Hop/Sti1, and RACK1), the molecular switch OsRac1 and its activator RacGEF and downstream target proteins of OsRac1 (NADPH oxidase, CCR1, and MAPK6). The pathogen infection signaling is primarily perceived by PRR and R proteins and transducted to OsRac1, which activates OsRac1 to trigger a series of downstream defense responses.

4.2.2.* Xa21*-mediated PTI defense signaling. Recently, several signaling components have been identified in the *Xa21*-mediated defense pathway through yeast hybrid screening. The first characterized *XA21*-binding protein (XB), XB3, is an ankyrin repeat domain and a RING finger-containing protein with E3 ubiquitin ligase activity (Wang et al 2006). XB3 is phosphorylated by *XA21* and functions as a positive regulator in the *XA21*-mediated immune signaling because reduced expression of the *Xb3* gene is compromised in resistance to the avirulent *Xoo* strains. Three other XBs, i.e., XB10 (transcription factor OsWRKY62) (Peng et al 2008), XB15 (a PP2C phosphatase; Park et al 2008), and XB24 (ATPase enzyme) (Chen et al 2010c), are negative regulators in the *XA21*-mediated immune signaling. Over-expression of any of the three genes compromises resistance to *Xoo* strains.

XB15 and XB24 play opposite roles in the biochemical regulation of *XA21*. XB24 is a promoter for the autophosphorylation of *XA21* (Chen et al 2010c). In contrast, XB15 dephosphorylates the autophosphorylated *XA21* (Park et al 2008), suggesting that the phosphorylation state of *XA21* is critical for *XA21*-mediated signaling. Recently, *XA21* was found to be cleaved to release the intracellular kinase domain with a functional nuclear localization sequence, and the intracellular kinase domain is transported into the nucleus and interacts with OsWRKY62 (Park et al 2012). Further *in vivo* cleavage of *XA21* and translocalization of the intracellular kinase domain to the nucleus is required for the *XA21*-mediated immune responses. The cleavage stability of *XA21* is regulated by the endoplasmic reticulum (ER) chaperone BiP3 and this regulation is critical for the resistance of *XA21* to *Xoo* (Park et al 2010). These results revealed a novel insight into immune receptor function: upon receptor recognition of conserved microbial signatures, the associated kinase translocates to the nucleus where it directly interacts with transcriptional regulators (Park et al 2012).

4.2.3.* Xa13*-mediated novel susceptibility mechanism. Significant progress has been made in understanding the ***Xa13***-mediated susceptibility to *Xoo.* A cDNA fragment of the *Xa13* N-terminal was used to screen interacting proteins in a rice cDNA library by yeast two-hybrid assays. Among the putative interacting proteins, two are putative copper

(Cu) transporters and are named COPT1 and COPT5 (Yuan et al 2010b). Expression of the three proteins, Xa13, COPT1, and COPT5, complemented a yeast mutant deficient in Cu transport, indicating they act in cooperation to transport Cu. The expression level of *Xa13*, *COPT1*, and *COPT5* is increased under Cu deficiency and is repressed under Cu excess. Over expression of the three genes increases the Cu content of roots and shoots but decreases the Cu content of xylem sap. A bacterial growth assay showed that Cu inhibits the growth of *Xoo* strain PXO99 (avirulent to *xa13*) but does not affect the growth of the strains lacking of the avirulence gene. Moreover, extracted xylem sap from sensitive *Xa13* plants supported growth of *Xoo* PXO99, but sap from *xa13* plants reduced pathogen growth. These results demonstrate that the infection by *Xoo* strain PXO99 is associated with activation of *XA13* (COPT1, and COPT5, which modulate Cu redistribution in rice.

Another study found that the expression of rice gene *Os8N3* or *Xa13* is highly induced by infection of *Xoo* strain PXO99A and is dependent on the type III effecter gene *pthXo1* (Yang et al 2006). RNAi silencing plants of *Os8N3* were resistant to infection by strain PXO99A but remained susceptible to other strains of the pathogen, indicating that *Os8N3* is a host susceptibility gene for bacterial blight targeted by the type III effecter *PthXo1*. Additional inoculations showed that *xa13* is defeated by strains of the pathogen carrying any of the type III effecters *AvrXa7, PthXo2,* or *PthXo3*. Both *AvrXa7* and *PthXo3* induce the expression of a second member of the *Os8N3* gene family, named Os-*11N3*. Insertional mutagenesis or RNAi silencing of the gene resulted in plants with loss of susceptibility specifically to *Xoo* strains carrying *AvrXa7* or *PthXo3* for virulence (Antony et al 2010). AvrXa7 interacts and binds specifically to an effecter-binding element within the *Os-11N3* promoter, confirming the proposed model for TAL effector-binding specificity. These results demonstrated that Os-*8N3* and Os-*11N3*-like N3 proteins have a specific function in facilitating bacterial blight disease.

An exciting study reported that *Xa13* is involved in sugar transport during *Xoo* infection (Chen et al 2010b). The TAL effecter *PthXo1* directly interacts with the *OsSWEET11 (Xa13)* promoter as shown by chromatin immune precipitation and transient coexpression in *Nicotiana benthamiana* leaves. The activation of *OsSWEET11* by *PthXo1* may induce sugar efflux to feed bacteria in the xylem and/or apoplasm. Mutations in the promoter of *OsSWEET11* or RNAi silencing of the gene lead to limitation of *Xoo* growth. The study also confirmed that *AvrXa7* activates the paralog *OsSWEET14* (Os-*11N3*), which may function as a low-affinity sugar transporter. These results demonstrate that the *Xoo* TAL effecters not only inhibit plant innate immunity but also manipulate nutritional resources in the host.

***4.2.4. Function of auxin-responsive genes in disease resistance to* Xoo.** A role for the plant growth hormone auxin in disease resistance to *Xoo* has been recently revealed in rice (Ding et al 2008, Domingo et al 2009, Kou et al 2010). Bacterial infection of rice induces the accumulation of indole-3-acetic acid (IAA). The *GH3* genes are a group of auxin-responsive genes and encode an IAA-amino synthetase that prevents free IAA accumulation. Over expression of one member of the GH3 family, *OsGH3-8,* results in enhanced disease resistance to *Xoo*. Two other members, *OsGH3-1* and *OsGH3-2,* also positively regulate the resistance to rice blast and bacterial blight (Domingo et al 2009, Fu et al 2010). In addition, the *OsGH3-8* mediated defense response is independent of JA and SA signaling, which suggests a novel role of the phytohormone auxin in rice disease resistance.

4.2.5. Function of germin-like protein (GLP) genes in defense responses. High levels of blast resistance were observed in a backcross line carrying the major-effect QTL on chromosome 8, which included a cluster of 12 OsGLP gene members (Liu et al 2004b). Oralate oxidase-like genes are referred to as german-like protein (GLP) genes belonging to cup in the superfamily (Membre et al 2000). GLPs influence plant defense through

the generation of active oxygen species and exhibit superoxide dismitase activity and are related to the accumulation of H_2O_2 involved in hypersensitive cell death (Manosalva 2009). To verify the contribution of these genes to disease resistance, Manosalva et al (2009) used a highly conserved portion of the OsGLP coding region as an RNA interference trigger to silence one to all members of the expressed chromosome 8 OsGLP family. Inoculations with both *M. oryzae* and *Xoo* showed that as more OsGLP genes were suppressed, disease susceptibility increased. Because these genes are highly conserved in rice and other cereals, natural selection may have preserved a whole gene family to provide a basal, broad-spectrum defense response to pathogen invasion**.**

4.2.6. Function of the SA-mediated pathway in disease resistance. The *NPR1* gene is a key regulator of systemic acquired resistance (SAR) in *Arabidopsis* (Dong, 2004). Overexpression of the *NPR1* homolog 1 (*NH1*) in rice results in enhanced resistance to *Xoo,* suggesting the presence of a related defense pathway in rice (Chern et al 2005b). Using NH1 as the bait, a rice cDNA encoding a novel protein called NRR was identified (Chern et al 2005a). While the *NRR* RNAi plants did not affect resistance to *Xoo,* plants over expressing NRR showed enhanced susceptibility to *Xoo.* This phenotype was correlated with elevated *NRR* mRNA and protein levels, and increased *Xoo* growth. In addition, over expression of *NRR* compromises *Xa21*-mediated resistance, suggesting a cross-talk or overlap between NH1- and *Xa21*-mediated defense pathways.

A microarray study also demonstrated the involvement of the SA-mediated pathway in rice disease resistance (Shimono et al 2007). Several WRKY transcription factor (TF) genes were induced 3 hrs after BTH treatment. Overexpression of one of the TF genes, *WRKY45,* significantly enhanced resistance to *M. oryzae* in rice. RNAi knockdown of *WRKY45* compromised BTH-inducible resistance to *M. oryzae,* indicating that *WRKY45* is essential for BTH-induced defense responses. Two defense-related genes, which encode a glutathione S-transferase and a cytochrome P450, are regulated downstream of WRKY45 but are not regulated by NH1, suggesting that WRKY45-mediated SAR is independent of NH1.

4.3. Genomic approach for dissecting the defense signaling pathways

In addition to the genetic approach, a direct assessment of the biochemical and physiological changes during disease development can be used to identify genes that might be involved in the disease response pathway. Enhanced expression of genes during infection indicates their possible involvement in host defenses. Many large-scale transcriptome analysis techniques such as EST (expressed sequence tag), SAGE (serial analysis of gene expression), MPSS (massively parallel signature sequencing), microarray, and RNA-Seq have been applied to reveal the genome-wide defense responses after pathogen infection in rice.

To characterize the genes activated in the *xa13*-mediated defense pathway against *Xoo,* Chu et al (2004) made two pathogen-induced subtraction cDNA libraries using the infected leaves of an IR24 resistant line carrying an R gene, *xa13,* and the susceptible cultivar IR24. Clustering analysis identified 702 unique expressed sequences that are specifically induced in the *xa13* plants including 110 new rice ESTs. These sequences encode a wide range of products, including defense-responsive genes commonly involved in different host–pathogen interactions, genes that have not previously been reported to be associated with pathogen-induced defense responses, and genes (38%) with no homology to previously described functional genes. About 48% of the mapped ESTs are colocalized with QTLs for resistance to various rice diseases, including bacterial blight, rice blast, sheath blight, and yellow mottle virus.

Using the 22-K rice microarray, Kottapalli et al (2007) compared the transcriptional profile at 6 and 120 hours after *Xoo* infection in the susceptible cultivar IR24 and the

resistant cultivar IET8585, which carries multiple R genes including a recessive R gene. The high expression of ethylene response element binding protein (EREBP) TF and the reduced expression of the alcohol dehydrogenase gene in the resistant cultivar may be responsible for the HR upon bacterial infection. Induction of the genes in the glutathione-mediated detoxification and flavonoid biosynthetic pathways and many defense genes during infection may be responsible for the recessive gene-mediated bacterial blight resistance in rice.

To understand the molecular basis of the defense response against *M. oryzae,* Jantasuriyarat et al (2005) constructed eight cDNA libraries using infected leaf tissues from resistant, partially resistant, and susceptible reactions at 6 and 24 hours after blast inoculation. Among the 68,920 sequenced ESTs, 13,570 unique sequences were obtained. Comparison of the pathogen-challenged libraries with the uninoculated control library revealed an increase in the percentage of genes in the functional categories of defense and signal transduction mechanisms, cell cycle control, cell division, and chromosome partitioning. A total of 7,748 new and unique ESTs were identified compared with the KOME full-length cDNA collection. The large cataloged collection of rice ESTs provided rich genomic resources for dissecting the rice defense pathways to pathogens.

Robust-long-SAGE (RL-SAGE) is a tag-based transcriptome analysis technique that allows researchers to isolate lowly expressed transcripts on a large-scale. Using this method, Gowda et al (2007) identified 83,382 distinct 21-bp tags from 627,262 individual tags from blast-infected leaves of a japonica cv Nipponbare challenged with an avirulent and a virulent pathogen isolate. Sequence analysis revealed that the tags in the resistant and susceptible libraries had a significantly reduced matching rate with the rice genomic and expressed sequences in the control. The high level of one-nucleotide mismatches is due to nucleotide conversions in the infected plants, suggesting that RNA editing may occur in the rice transcriptome at early infection stages.

Using both RL-SAGE and microarray methods, Venu et al (2007) investigated the transcriptome changes in *R. solani*-infected rice plants. RL-SAGE sequence analysis identified 20,233 and 24,049 distinct tags from the control and inoculated libraries, respectively. About 60% of the library-specific (≥10 copies) and differentially expressed (>4.0-fold change) tags were novel transcripts. About 70% of the genes identified in the RL-SAGE libraries showed similar expression patterns (up or down-regulated) in the microarray data. Some candidate RL-SAGE tags and microarray genes were located in known sheath blight QTL regions. This study provided useful genomic materials for further elucidation of the molecular basis of the defense response to *R. solani* and for fine mapping of target sheath blight QTLs.

5. Challenges and opportunities

During recent times, the characterization of many R genes, QTLs, and genes involved in defense signaling and responses to pathogens in rice has greatly advanced our understanding about the molecular mechanisms of host-pathogen interactions and activation. As indicated in the following, however, major questions remain unanswered and should be the focus of future research.

5.1. Difference and relationship between R genes and QTLs

In-depth analyses of all the identified blast R and QTL genes have shown that many metaQTLs are colocalized in the complete R-gene regions (Ballini 2008). This raises a question about the differences and relationships between these two types of genes: does a QTL encode a defeated R protein or a defense-related protein? Consistent with this possibility is the finding that the cloned blast QTL *Pb1* is isolate-specific and also

encodes an NBS-LRR protein (Hayashi et al 2010), which is a common structure of most cloned R genes (Liu et al 2010). However, the other cloned recessive QTL, *pi21,* encodes a novel proline-containing protein (Fukuoka et al 2009). Future research should investigate whether QTLs recognize Avr genes in the pathogen just as R genes do. Future research should also determine the differences and similarities between R gene- and QTL-mediated resistance pathways at the molecular level.

5.2 Function of Avr proteins and their host targets during infection

Although about 12 Avr genes have been cloned from *M. oryzae* and *Xoo,* how they are secreted into rice cells and interact with host proteins is unclear. The finding that *Xoo* TAL effectors interact and bind the promoter of host defense genes is quite encouraging. Still, the function of most cloned Avr effectors during initial recognition processes, their secretion mechanism, and their host targets during infection remain unknown.

5.3. Molecular mechanism of ETI-mediated resistance

Although about 17 major blast R genes have been cloned, it is still unknown how these R proteins directly or indirectly perceive signals from Avr effectors and thereby activate a cascade of defense responses after infection. Because many conventional approaches (such as Y2H and mutagenesis) have failed to reveal how these signals are perceived, new methods should be used to find R protein interactors and chaperones such as protein affinity purification.

5.4. Molecular mechanism of PTI-mediated resistance

Only a few PRRs in rice and PAMP effectors from rice pathogens have been characterized. Cloning of AX21 has raised critical questions about the relationship between PAMP effectors and R proteins. Is the pair of AX21 and Xa21 in the bacterial blight pathosystem an exception or a common mechanism for rice diseases? Does any PRR protein recognize Avr proteins during pathogen infection?

5.5. Function of signaling chaperones and other defense proteins

Recent research shows that OsRac1 is an integrator of rice PTI- and ETI-mediated defense signaling (Kawano and Shimamoto 2013) and that several molecular chaperones and cochaperones play critical roles in the OsRac1-mediated defensomes (Chen et al 2010a). The Hop/Sti-Hsp90 and lectin chaperone systems are responsible for the maturation and transport of PRRs. Research is needed to determine whether these systems are also involved in the processing of other R proteins.

5.6. Relationship between innate immunity and plant hormones and nutrition regulation

A role for auxin and SA in disease resistance to *Xoo* has been revealed in rice (Chern et al 2005b, Ding et al 2008). Whether plant hormones affect R gene-mediated resistance and whether other plant hormones are also involved in resistance should be determined. The involvement of *Xoo* TAL effectors in targeting a host protein to manipulate sugar transport during *Xoo* infection is quite intriguing (Chen et al 2010b). Research is needed to elucidate how changes in sugar efflux caused by pathogens affect host resistance.

5.7. Application of ultra-fast sequencing technologies and systems biology for dissection of disease resistance pathways

Because the cost of sequencing will drop rapidly in the near future and more advanced programs suitable in handling large and complex datasets are developed, systems biology

will become a powerful approach for dissecting complex disease resistance pathways in rice. Resequencing of rice cultivars, development of high-density SNP maps, and application of whole-genome association mapping will undoubtedly facilitate our efforts to identify new genes that are important in host resistance.

5.8. Roles of epigenetic regulation and small RNA-mediated pathway in rice–pathogen interactions

Both epigenetic control and small RNA-mediated pathway play pivotal roles in plant-microbe interactions and host defenses in the model plant ***Arabidopsis*** (Padmanabhan et al 2009, Alvarez et al 2010). Investigating whether these host defense mechanisms have similar functions in rice will be fruitful.

5.9. Control of rice diseases is critical

Given that about half of the world population depends on rice, controlling pathogens and other pests of this staple crop is vital. Although substantial progress has been made in developing cultivars with resistance to blast, blight, and other serious diseases of rice, we clearly cannot assume victory. First, we still do not understand the mechanisms underlying resistance and this limits our ability to develop and deploy new kinds of resistance. Second, the pathogen populations are not static and their ability to overcome resistance is an ongoing arms race in the coevolution of host and the pathogen, and does not depend on obtaining grant money. We must try to minimize the selection of new resistance-breaking strains, and we must be prepared to respond when such strains emerge.

6. Acknowledgements

The authors thank the NSF-Plant Genome Research Program (#0605017) and USDA-CSREES (#2007-01667), OARDC Seeds, and USAID/IRRI Linkage Program for their financial support.

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