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Chapter 7

Current Advances on Genetic Resistance to Rice Blast Disease

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Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/56824

1. Introduction

1.1. The historical and contemporary aspects of rice blast disease

Rice (*Oryza sativa L.*) is one of the most important staple foods that feed more than half of the world's population, with Asia and Africa as the largest consuming regions [1]. Blast disease caused by *Magnaporthe oryzae* (Hebert) Barr is one of the most damaging diseases of rice. This disease was first known as rice fever disease in China as early as 1637 [2]. Blast disease was first reported in the United States in 1876, and has been identified in 85 rice-producing countries or regions worldwide (Figure 1).

Blast severely affects lowland rice in temperate and subtropical areas of Asia, and is highly destructive to upland rice in tropical areas of Asia, Latin America, and Africa [3]. Although blast is considered the most destructive rice disease due to the favorable environmental conditions for disease occurrence and worldwide distribution, little information about annual yield losses are available. Table 1 summarizes reported blast outbreaks with annual yield losses from five countries. In China, 40-50% yield losses were observed under severe rice blast infection; in some cases, 100% yield losses were found in severely infected fields [4]. Yield losses of 5-10%, 8%, and 14% were reported in India from 1960 to 1961, Korea from the mid-1970s, and China from 1980 to 1981, respectively [3]. The highest yield losses were recorded in the Philippines; ranging from 50% to 85% in 1963 [3]. It was estimated that 1.6 billion dollars were lost from 1975-1990 due to blast disease worldwide [5]. The estimated annual loss of rice was enough to feed 60 million people for one year [6] (Table 1).

Figure 1. Worldwide distribution of rice blast disease. Red dots show the countries or regions where blast disease has been reported.

Yield loss (%)	Country	Year
5-10	India	1960-61
50-60	Philippines	1963
70-85	Philippines	1969-70
8	Korea	mid-70s
14	China	1980-81
60	Thailand	1982

Source: http://www.knowledgebank.irri.org/ipm/rice-blast.html

Table 1. Yield losses due to blast.

1.2. The biology of *M. oryzae*

The most common symptoms in commercial rice fields induced by *M. oryzae* can be found on all the above ground parts of the rice plant at all growth stages. Seeds display brown spots after infection, which may have resulted from the infection of the florets as they mature into seeds. Infected roots have also been observed; however, lesions on the sheaths were relatively rare. Infections on young seedlings are initiated when the conidia are deposited on the surface of the leaves. Water is essential for spores to germinate and attach to the leaf surface [7, 8]. Under optimal conditions, spore germination occurs rapidly and the polarized germ tubes are

http://dx.doi.org/10.5772/56824

formed within hours after landing on the leaf [9]. The secondary cycles are initiated by the spores produced by the lesions on the young seedlings, which can be repeated many times through the growing season. Thousands of spores can be produced from a single lesion in 15 days after infection. Typically blast lesions are diamond shaped (Figure 2A). Initial lesions appear dark green or grey with brown borders; while, older lesions are light tan with necrotic borders. Under favorable conditions, lesions can merge together and rapidly enlarge to several centimeters in length, eventually killing the leaf, and ultimately resulting in plant death. On resistant cultivars, lesions induced by *M. oryzae* usually remain small in size (1-2mm) and appear brown to dark brown in color. Disease severity of rice blast and the amount of spores produced on single lesion depends on temperature, field conditions, relative humidity, fertilization levels, and genotype of rice cultivars. In general, moderate temperatures (~24°C), high relative humidity (90-92%), and high moisture with at least a 12-hour period are advantageous for rice blast. The disease severity of the vegetative phase during the growing season highly influences the amount of disease during the reproductive phase. Spores produced at the end of the growing season may result in collar blast and neck blast; neck blast often causes direct crop loss (Figure 2B).

Existence of physiological races of *M. oryzae* complicates the identification of resistance *(R)* genes. Physiological races of *M. oryzae* were first reported by Sasaki in Japan as early as 1922 [10]. From 1950s to 60s differential rice lines resistant to races of *M. oryzae* were identified in Japan, the United States, India, the Philippines, and South Korea. In 1961, 18 physiological races of *M. oryzae* were identified with 12 differential rice varieties in Japan. During that time, an international differential system using 8 rice varieties was established [11]. In China, the identification of *M. oryzae* races was initiated in late 1970s. Seven rice varieties, Tebo, Zhenlong13, Sifeng43, Dongnong363, Kanto51, Hejiang18, and Lijiangxintuanheigu (LTH), and 43 isolates of *M. oryzae* were used. In 1976, Yamada and his colleagues identified 23 races of *M. oryzae* from 2245 isolates with 9 differential rice varieties. Duan et al [12] used Yuyun 1 (with *Pia*), Gaoliangdao (with *Pii*), Kanto51 (with *Pik*), Chugeng1 (with *Pikm*), Dianyu1 × Fook Kam (with *Piz*), Dali782 (with *Pita*), Dan83-3 (with *Pita2*), and Chengbao1 (with*Pizt*) as differential varieties to characterize races of *M. oryzae* in China. These blast *R* genes are described in more details in the part II of this chapter. Near-isogenic lines (NILs) were chosen to better identify races of *M. oryzae* in a gene-for-gene specific manner. The NILs with *indica* high-susceptible variety CO39 background was developed at the International Rice Research Institute (IRRI), the Philippines [13]. In the United States, Marchetti [14] reported that the races IB-54, ID-13, IG-1, and IH-1 of *M. oryzae* were the most common. Most recently, monogenic lines with 24 major blast *R* genes in BC1 of LTH were developed by scientists at IRRI and Japan [15].

Extensive analysis of rice germplasm with physiological races in the past century reveals that complete genetic resistance (vertical resistance) is conferred by major blast *R* genes named as *Piricularia* genes or *Pi*-genes. These genes are often specific in preventing infections by strains of *M. oryzae* that contain the corresponding avirulence genes; whereas, incomplete resistance (slow-blasting components or horizontal resistance, field resistance, or dilatory resistance) is often conditioned by more than one gene on different chromosomal regions. These genes are

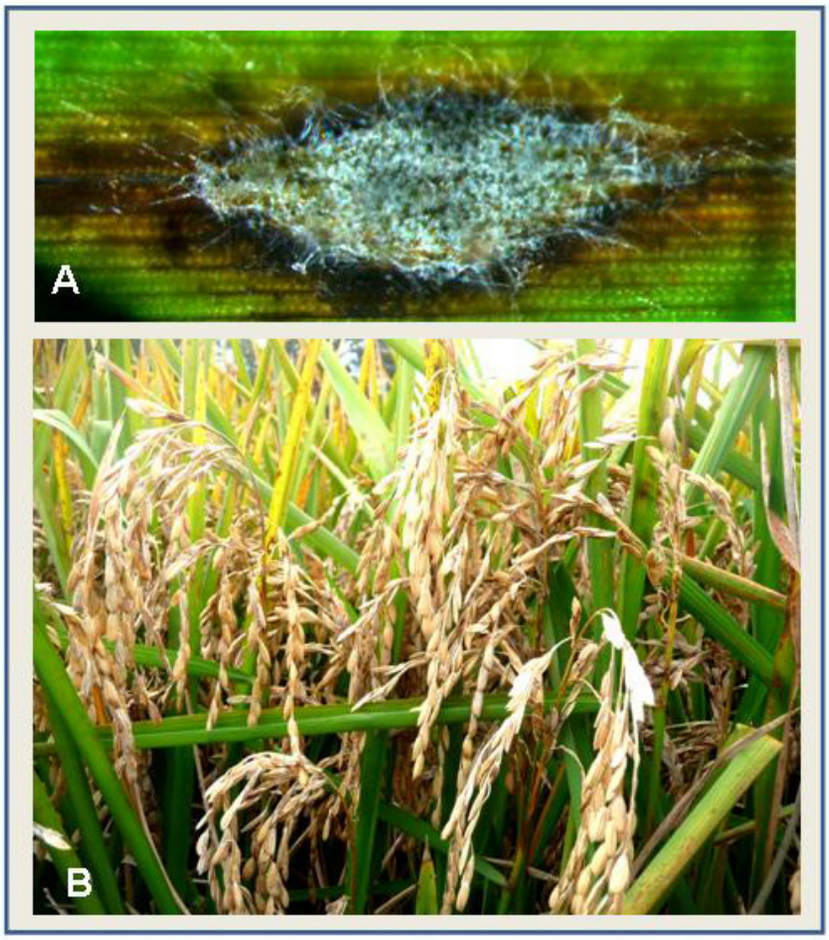

Figure 2. Symptoms of leaf (A) and neck blast diseases (B) in commercial rice fields.

referred to as quantitative resistant loci (QTLs). Resistant germpalsms carrying both major and minor *R* genes and are extremely important genetic resources that rice breeders can use to improve blast resistance in elite rice varieties.

2. Mapped blast *R* genes

Blast *R* genes are predicted to play important roles in the frontier of rice defense responses. During interactions between rice and blast pathogens, products of the *R* gene can specifically recognize the corresponding elicitors of *M. oryzae*. Since the *Pia* gene, indentified in 1967 by Kiyosawa as the first blast *R* gene from the *japonica* variety Aichi Asahi [16], 99 blast *R* genes have been identified; in which 45% were found in *japonica* cultivars, 51% in *indica* cultivars, and the rest 4% in wild rice species (Table 2 to 5). Most deployed *R* genes have often been identified in Asian cultivated rice, specially rice cultivars from Japan and China, with the exception of *Pi9*, *Pi54rh*, *Pi40(t)*, and *Pirf2-1(t)*, which were domesticated from *O. minuta*, *O.*

rhizomatis, *O. australiensis*, and *O. rufipogon*, respectively. All *R* genes have been mapped on all rice chromosomes except for chromosome 3 (Tables 2 to 4; Fig 3). Host genotypes, chromosomal loci, and molecular markers that are tightly linked to blast *R* genes are summarized in Figure 2 and Table 2 (60 major *R* gene) and Table 3 (17 minor *R* gene). Among them, three major *R* gene clusters have been well characeterized: the *Piz* locus on Chromosome 6, the *Pik* locus on Chromosome 11, and the *Pita* locus on Chromosome 12 (Figure 3). More detailed imformation of mapped blast *R* genes can be found at http://www.ricedata.cn/gene/, http://www.shigen.nig.ac.jp/rice/oryzabaseV4/, and http://www.gramene.com.

Chromosome	Major R genes mapped*	Minor R genes mapped*	*R gene cloned**	Total*
1	2	2	3	7
2	7	3	1	11
3	0	0	0	0
4	1	2	1	4
5	2	1	0	3
6	12	1	6	19
7	1	0	0	1
8	4	2	1	7
9	3	0	1	3
10	1	1	0	2
11	12	3	8	23
12	15	2	1	18
Total	60	17	22	99

* refers to number of the genes on the chromosome.

Table 2. Summary of blast *R* (major and minor, mapped and cloned) genes on rice chromosomes.

Chr.	Name of *R* gene	Name of germplasm	Map position (cM) >,#	Markers	Name of pathogenic Strains	Ref.
1	*Pi27(t)**	Q14	28.4-38.8	RM151, RM259	CHL0335, CHL0888, CHL0918	[17]
1	*Pitp(t)*	Tetep	114.1	RM246	IC9	[18]
2	*Pi14(t)*	Maowangu		Amp-1		[19]
2	*Pi16(t)*	Aus373		Amp-1	Hoko1, Ina72, TH67-22, Ai75-61	[20]
2	*PiDa(t)*	Dacca6	10.8-14.4	RM211, RM5529		[21]

Chr.	Name of *R* gene	Name of germplasm	Map position (cM) >#	Markers	Name of pathogenic Strains	Ref.
2	*Pid1(t)*	Digu	87.5-89.9	RM262	ZB13	[22]
2	*Pitq-5*	Teqing	150.5-157.5	RG520, RZ446b	IC-17, IB-49, IE-1, IG-1	[23]
2	*Pig(t)*	Guangchangzhan	142.0-154.1	RM166, RM208	Ken53-33	[24]
2	*Piy(t)*	Yanxian No.1	153.2-154.1	RM3284, RM208	97-27-2, Zhong10-8-14	[25]
4	*Pi39(t)**	Chubu 111	107.4-108.2	RM3743, RM5473		[26]
5	*Pi10(t)*	Tongil	88.5-102.8	RG13	IB46	[27]
5	*Pi23(t)*	Suweon 365	59.3-99.5			[28]
6	*Pi2-1*	Tianjingyeshengdao	Allilic to Pi2/9	AP4791, AP4007	CHL477, CHL473, P06-6, IC-17, 87-4	[29]
6	*Pi2-2*	Jefferson	58.7	RM19817, AP5659-5	HN318-2, CHL438, KJ201, ROR1, PO6-6	[30]
6	*Pi8(t)*	Kasalath	74.6-78.2	Amp-3	Race 447.1	[31]
6	*Pi13(t)**	Maowangu	74.6-78.2	Amp-3		[19]
6	*Pi13(t)**	Kasalath	67.7-68.5	RM2123, RM20155	Ken54-04, 95Mu-29, Ina86-137	[32]
6	*Pi22(t)*	Suweon 365	38.4-41.9		KJ-201	[28]
6	*Pi26(t)**	Gumei 2	51.0-61.6	B10, R674	Ca89	[33]
6	*Pi40(t)*	IR65482-4-136-2-2 *O. australiensis*	54.1-61.6	RM527, RM3330	KJ105, Ca89, PO6-6, M101-1-29-1, M64-1-3-9	[34]
6	*Pi50(t)*	Er-Ba-Zhan	46.8	GDAP51, GDAP16	09-3041a, SC0602, SCRB14, HN0102, W06-18a	[35]
6	*Pigm(t)*	Gumei 4	65.8	C5483, C0428	CH109 (ZC13), CH147 (ZB25), CH131 (ZA1)	[36]
6	*Piz*	Zenith	58.7	z4792, z60510, z5765		[37]
6	*Pitq-1*	Teqing	103.0-124.4	C236, RG653	IC-17, IB-49, IE-1	[23]
7	*Pi17(t)*	DJ123	94.0-104.0	Est9		[38]
8	*Pi42(t)*	Zhe733	58.5	RM72	IE1K	[39]
8	*Pi33(t)*	IR64	45.4	RM72, C483	Guy11	[40]
8	*Pi55(t)*	Yuejingsimiao 2	99.1-102.1	RM1345, RM3452	CHL688	[41]
8	*PiGD-1(t)*	Sanhuangzhan 2	53.7	RG1034	GD RFDW-I	[42]
9	*Pi3(t)*	C104PKT,	31.3-33.0	40N23r	PO6-6	[43]

Chr.	Name of *R* gene	Name of germplasm	Map position (cM) >#	Markers	Name of pathogenic Strains	Ref.
9	*Pi15(t)*	GA25	31.3-34.9	CRG3, CRG4	CHL0416 , Hoku 1	[44]
9	*Pi56(t)*	Sanhuangzhan 2	31.3	RM24022	PO6-6	[42]
10	*PiGD-2(t)*	Sanhuangzhan 2		R16, R14B	PO6-6	[42]
11	*Pi18(t)*	Suweon 365	117.9	RZ536	KI-313	[45]
11	*Pi38(t)*	Tadukan	79.1-88.7	RM206, RM21	B157	[46]
11	*Pi44(t)*	Moroberekan	91.4-117.9	AF349	C9240-1	[47]
11	*PiCO39(t)*	CO39	49.1	S2712	6082	[48]
11	*Pilm-2*	Lemont	56.2-117.9	R4, RZ536	IB54, IG1	[23]
11	*Pi7(t)*	Moroberekan	71.4-84.3	RG103, RG16	PO6-6	[49]
11	*Pi47(t)*	Xiangzi 3150	104.2-120.1	RM206, RM224		[50]
11	*Pi43(t)*	Zhe733	109.5	RM1233	IE1K	[39]
11	*Piks*	Bengal, M201	115.1-117.3	RM224, RM1233		[51]
11	*Pikg(t)*	GA20	119.9-120.3			[19]
11	*Piy(t)*	Yunyin	54	RM202	Sichuang-43	[52]
11	*Pizy(t)*	Ziyu44	102.9	RM206	ZB13, ZE1	[53]
12	*Pi19(t)*	Aichi Asahi	50.4-51.5	RM27937, RM1337	CHNO58-3-1, IRBL19-A	[54]
12	*Pita-2*	Tetep, Pi No.4	50.4			‡
12	*Pi6(t)*	Apura	12.2-47.9	RG457, RG869		[55]
12	*Pi62(t)*	Yashiro-mochi	12.2-26.0	RG9, RZ816	4360-R-62	[56]
12	*Pi24(t)**	Zhong 156	51.5	RG241A	92-183 (ZC15)	[57]
12	*Pi12(t)*	Moroberekan	47.9	RG869		[58]
12	*Pi20(t)*	IR24	51.5-51.8	RM1337, RM5364, RM7102	BN111	[58]
12	*PiGD-3*	Sanhuangzhan 2	55.8	RM179	GD RFDW-IV	[42]
12	*Pi51(t)*	Tianjingyeshengdao		RM5364, RM27990	CHL447, RB5, CHL473, PO6-6	[29]
12	*Pi39(t)**	Q15	50.4	RM27933, RM27940	CHL724	[59]
12	*Pi41(t)*	93-11		RM28130	CHL1789, CHL347, CHL688	[60]
12	*Pi157(t)*	Moroberekan	49.5-62.2	RG341, RG9		[61]

Chr.	Name of *R* gene	Name of germplasm	Map position (cM) >#	Markers	Name of pathogenic Strains	Ref.
12	*Pi48(t)*	Xiangzi 3150		RM5364, RM7102		[50]
12	*Pitq-6*	Teqing	47.9	RG869, RZ397	IC-17, IB-49, IE-1, IB-54	[23]
12	*Pih1(t)*	Hongjiaozhan	47.9	RG869, RG81	ZB1	[62]

* This *R* gene shares the same name with another *R* gene.

The map positions were integrated into IRGSP map according to marker information. Detail information can be found on http://rgp.dna.affrc.go.jp/E/IRGSP/index.html.

‡ Information is known, but has not been published.

Table 3. Summary of major blast *R* genes including their resistance specificity, chromosomal location, map position, and tightly linked DNA markers.

Chr.	Name of *R* gene	Donor	Map position (cM) >#	DNA Markers	Avirulent race/isolate	Ref.
1	*Pi24(t)**	Azucena	64.4	K5	CL6	[63]
1	*Pi35(t)*	Hokkai 188	132.0-136.6	RM1216, RM1003		[64]
2	*Pir2-3*	IR64	141.7	RM263, RM250	Race 173	[65]
2	*Pi25(t)**	IR64	157.9	RG520	BR26, CH66, CH72	[63]
2	*Pirf2-1(t)*	*O. rufipogon*	172.3	RM206, RM266	Race 001	[65]
4	*Pikur1*	Kuroka	86.0			[66]
4	*Pikahei1(t)*	Kahei	108.2	RM17496, RM6629		[67]
5	*Pi26(t)**	Azucena	22.5-24.7	RG313	PH68	[63]
6	*Pi27(t)**	IR64	51.9	Est-2	CH66	[63]
8	*Pi11(t)*	Zhaiyeqing8	53.2-84.8	BP127A, RZ617	18-2, ZH7-2, Zhong10-2-4,	[68]
8	*Pi29(t)*	IR64	69	RZ617, RGA-IR86	CL6	[63]
10	*Pi28(t)*	Azucena	114.7	RZ500	PH68	[63]
11	*Pi30(t)*	IR64	59.4-60.4	OpZ11-f, RGA-IR14	CH66, CH72	[63]
11	*Pi34(t)*	Chubu32	79.1-91.4	Z77, Z150-5		[69]
11	*Pif*	St No. 1	119-120			[70]
12	*Pi31(t)*	IR64	44.3	O10-800	PH68, CD69	[63]
12	*Pi32(t)*	IR64	47.5	AF6	BR26	[63]

* This R gene shares the same name with another R gene.

The map positions were integrated into IRGSP map according to marker information. Detail information can be found on http://rgp.dna.affrc.go.jp/E/IRGSP/index.html.

Table 4. Summary of minor blast *R* genes, donors, map position, tightly linked DNA markers, and associated blast races.

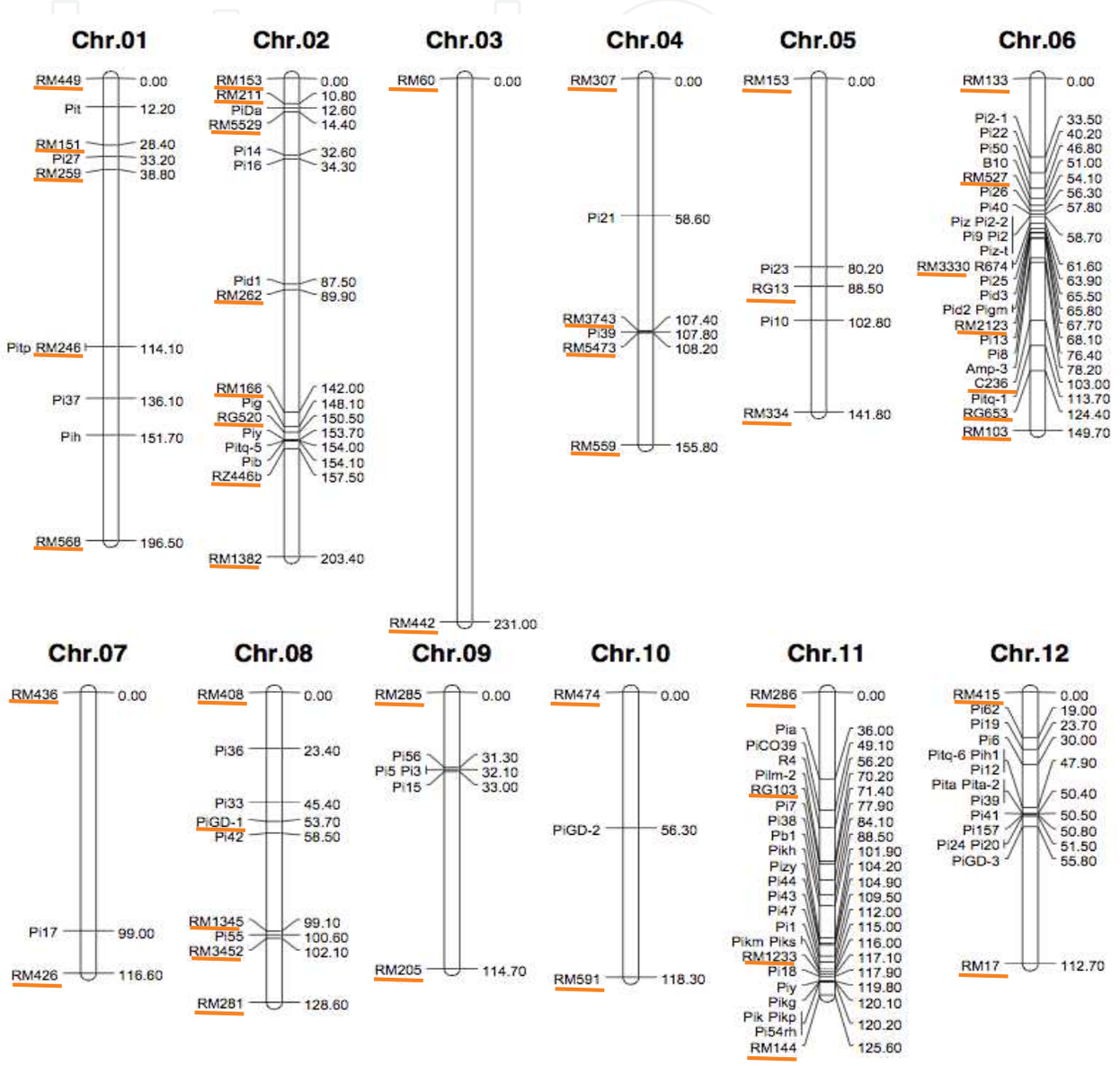

Figure 3. Location of cloned and mapped *R* genes on rice chromosomes. The locations of *R* genes have been integrated into IRGSP map according to marker information, then the map was built using Mapmaker software. Centimorgan was used to measure the map positions showing in the right column of the choromosome. The underlined words indicate either SSR or RFLP markers (see additional resources: http://www.shigen.nig.ac.jp/rice/oryzabaseV4/insd/detail/3554).

Chr.	*R* gene cloned	Donor and cultivar or line carrying the gene	Map position (cM)>#	Markers	Locus structure	Protein type	Subcellular localization	FNPs	Expression	Ref.
1	*Pit*	K59, Tjahaja	12.2	T256	Multiple	CC-NBS-LRR			Repressible	[71]
1	*Pi37*	St. No1	136.1	RM302, RM212	Multiple	NBS-LRR	Cytoplasm	V 239 A, I 247 M	Constitutive	[72]
1	*Pish*	Shin 2, Norin 22	148.7-154.8		Multiple	CC-NBS-LRR			Constitutive	[73]
2	*Pib*	Engkatek, Tohoku IL9, Teqing, Tjinam, BL1	154.1	RM208	Multiple	NBS-LRR			Inducible	[74]
4	*pi21*	Owarihatamochi	58.6	P702D03	Multiple	NBS-LRR	Cytoplasm			[75]
6	*Pid2*	Digu (I)	65.8		Single	Receptor kinase	Membrane	I 441 M	Constitutive	[76]
6	*Pi9*	*O.minuta,* 75-1-127	58.7		Multiple	NBS-LRR			Constitutive	[77]
6	*Pi2*	C101A51	58.7		Multiple	NBS-LRR		R 838 S	Constitutive	[78]
6	*Piz-t*	TKM, Toride 1	58.7	zt56591	Multiple	NBS-LRR		S 839 R	Constitutive	[79]
6	*Pid3*	Digu	65.2-65.8		Single	NBS-LRR		Q 737 Stop		[79]
6	*Pi25**	Gumei 2	63.2-64.6		Multiple	CC-NBS-LRR				[80]
8	*Pi36*	Q61, Kasalath	21.6-25.2	CRG3	Single	NBS-LRR		S 590 D	Constitutive	[81]
9	*Pi5*	Tetep, RIL 260	31.3-33.0	76B14r, 40N23r	Multiple	CC-NBS-LRR	Cytoplasm		Pi5-1 is inducible, Pi5-2 is constitutive	[82]
11	*Pi1*	LAC23, C101LAC	112.1-117.9		Multiple	NBS-LRR				[83]
11	*Pik*	To-To, Kusabue, Kanto 51, K60, Chugoku 31, Shin 2-1, K2, K3, , Minehikari, GA 20	119.9-120.3	k8823, k8824, k3951, k39512	Multiple	CC-NBS-LRR			Constitutive	[84], [85]
11	*Pikm*	Hokushi,Tsuyuake , IRBLkm-Ts	115.1-117.0	k2167, k6441	Multiple	NBS-LRR			Constitutive	[86]
11	*Pikp*	Tetep K60	119.9-120.3	k3957	Multiple	CC-NBS-LRR			Constitutive	[87]
11	*Pikh*	Tetep, K3, Kaybonnet, Lemont, Lebonnet	101.9	RM224	Multiple	NBS-LRR			Inducible	[88]
11	*Pi54rh*	*O. rhizomatis*	119.9-120.3		Multiple	CC-NBS-LRR	Extracellular		Inducible	[89]
11	*Pia*	Aichi Asahi	36.0	Yca72	Multiple	NBS-LRR				[90]
11	*Pb1*	Modan, Tsukinohikari, St NO. 1	85.7-91.4		Single	CC-NBS-LRR			Age-dependent	[91]
12	*Pita*	Tetep, Katy, Teqing	50.4		Single	NBS-LRR	Cytoplasm	A 918 S	Constitutive	[92]

* This R gene shares the same name with another R gene. # The map positions were integrated into IRGSP map according to marker information. Detail information can be found on http://rgp.dna.affrc.go.jp/E/IR.

Table 5. Summary of cloned *R* genes, map position, closely linked DNA markers, and their expression.

http://dx.doi.org/10.5772/56824

3. Structure and function of blast *R* genes

Among the mapped *R* genes (Table 3 and 4), 22 genes including 20 major and 2 minor *R* genes (*Pb1* and *pi21*) have been molecularly characterized (Table 5). Noticeably, *Pid2*, *Pid3*, *Pi36*, *Pb1*, and *Pita* are single copy genes; while others are members of small gene families. A total of eight *R* genes have been identified on chromosome 11, with six at the *Pik* locus; six *R* genes on chromosome 6, four of which are at the *Piz* locus. Most cloned blast *R* genes are adequate in providing complete resistance to strains of *M. oryzae* that contain the corresponding avirulence genes. Interestingly, two different members of each of *Pi5*, *Pik*, *Pikp*, *Pikm*, and *Pia* are required for complete resistance to some avirulent races.

Similar to other plant *R* genes, all cloned blast *R* genes to date encode predicted proteins with centrally located nucleotide binding sites (NBS) and leucine rich repeat (LRR) at the carboxyl terminus (Figure 4), with the exception of *Pid2* and *pi21* encoding a B-lectin kinase protein and a proline containing protein, respectively. Plant NBS-LRR proteins can be divided into two subgroups based on whether they contain a Toll-interleukin receptor (TIR)-like domain (TIR-

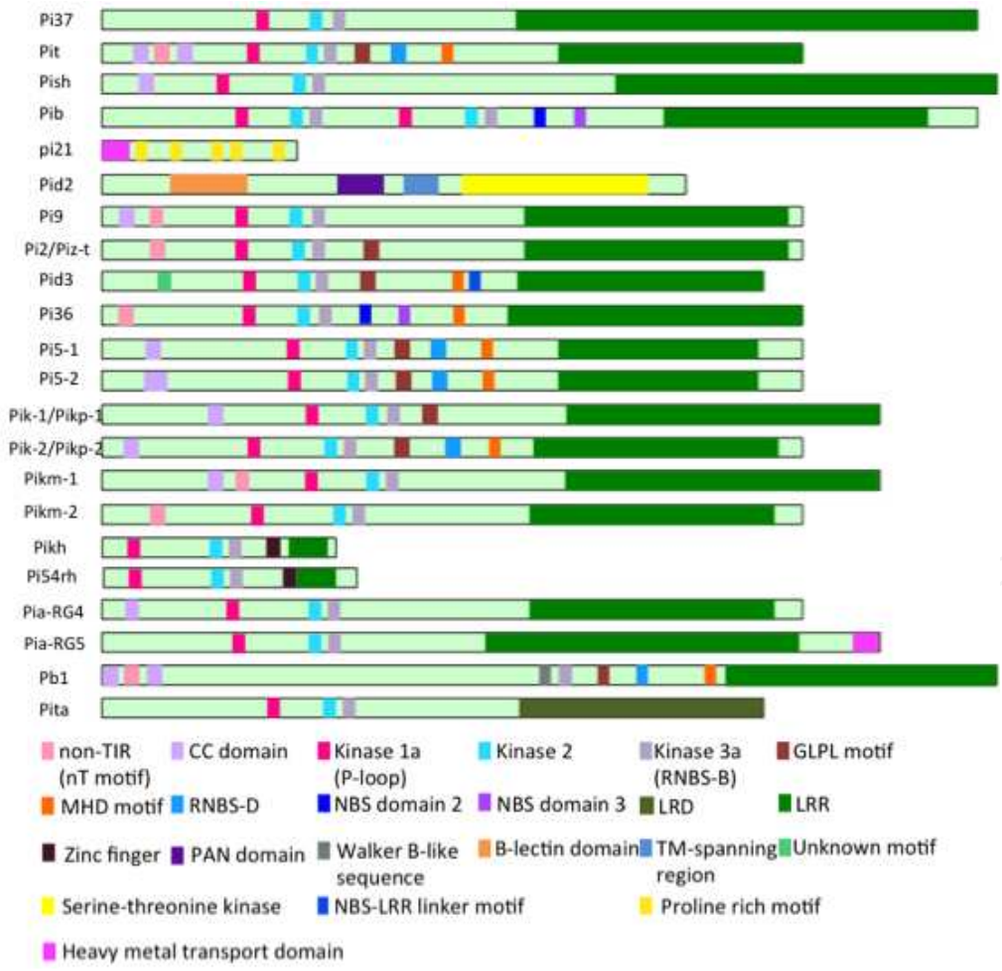

Figure 4. Structure of all cloned *R* genes. The light green bar represents the length of the *R* genes. The highlighted bars represent the different domains of the *R* genes.

NBS-LRR) or a putative coiled-coil (CC) structure (CC-NBS-LRR) in their amino-terminal region. The rice genome has 500 NBS-LRR gene families, and most of them belong to the CC-NBS-LRR family. The NBS domain contains kinase 1a (p-loop), kinase 2 and 3a (RNBS-B) motif, which presumably bind to ATP and trigger downstream signal transduction; whereas, the LRR is predicted to recognize pathogen effectors, either directly or indirectly. Other noticeable protein domains of plant *R* proteins were also summarized in Figure 4.

The observed structural similarities of blast R proteins might imply that their predicted conserved regions are associated with functional roles in triggering resistance to *M. oryzae*. Cloned blast *R* genes can be separated into two clades, I and II (Figure 5). Clade I consists of all NBS-LRR genes and clade II contains both NBS-LRR and non-NBS-LRR gene, such as *Pid2* and *pi21.* Among them, *Pi1-5, Pik-1, Pikp-1*, and *Pikm1-TS* on chromosome 11 share substantial homology to the *Pi9* locus on Chromosome 6; whereas, *Pi1-6, Pik-2, Pikp-2*, and *Pikm2-T* are more similar to *Pid2* and *pi21*, which are not NBS-LRR *R* genes. Homologous sequences of blast *R* genes can be found in the diverse germplasm of cultivated species including domesticated landrace varieties and wild relatives of rice. These observations suggest that genetics of rice immunity is ancient and may have been evolved during speciation and domestication.

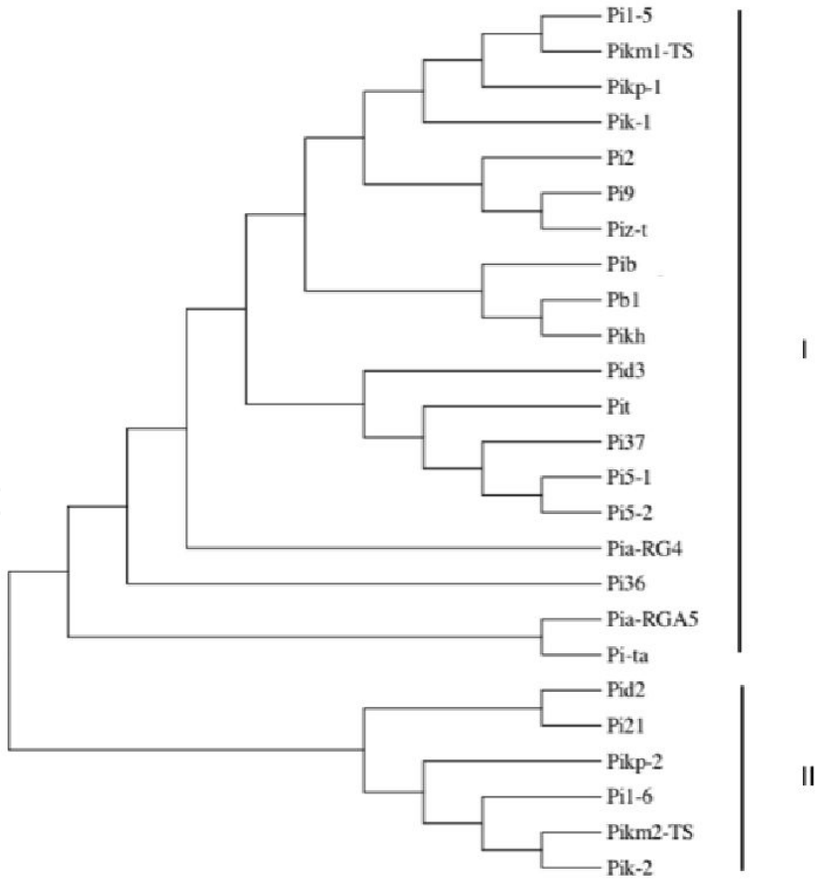

Figure 5. Phylogenetic tree of all cloned blast *R* genes. The tree was constructed using protein sequences by software Mega 5.0 NJ method.

http://dx.doi.org/10.5772/56824

4. *R* gene-mediated signaling transduction pathways

It is now commonly accepted that products of *R* genes in plants can specifically recognize avirulence genes from the pathogen directly or indirectly to initiate innate immunity system responses. Direct recognition of the putative product of the avirulence gene, *AVR-Pita1* by the *Pita* protein was first reported in 2000 [93]. Over a decade later, in 2012, another blast *R* protein, *Pik*, with similar structure to *Pita*, was found to directly recognize the corresponding avirulence gene *AvrPik* [94]. Direct interactions between other blast *R* and avirulence genes have not been reported; suggesting that indirect interactions may be responsible in triggering effective signal transduction pathways. Other plant genes involved in signal transduction have been investigated by the use of *R* proteins as bait in the yeast two-hybrid system (Y2H). While Y2H is a highly effective tool, it is limited in indentifying immediate plant components of *R* proteins. Molecular basis of blast *R* gene-mediated signaling has been a subject of intensive investigation worldwide. Abundant genes that may be involved in *R* gene-mediated signaling have been identified with DNA microarray [95, 96], and most of them were pathogenicity related genes. Genetic analysis using mutagenesis has been another commonly used alternative to identify downstream components. However, most mutants identified, thus far, are lesion mimic mutants [97]. A major effort to identify *Pita* mediated signaling was accomplished by treating 20,000 Katy seeds with *Pita*/*Pita*^{2}/*Pik*^{s} using fast neutrons, ethyl methyl sulfate (EMS), and gamma irradiation [98]. A total of 142 rice seedlings, with altered disease reactions, were identified from independent M2. The susceptibility of M2 individuals was verified in subsequent generations, and 20 of them were confirmed to be derived from Katy using 20 diagnostic single sequence repeat (SSR) markers. Consequently, the *Ptr(t)* gene in rice was identified to be essential for *Pita* mediated signal transudation [99]. Molecular cloning of *Ptr(t)* will shed light on the interaction mechanism of *Pita* and *Ptr(t)*, and subsequent plant genes involved in defense responses.

5. The management of blast disease-marker assisted selection

Blast disease has been effectively managed by a combination of fungicides and *R* genes integrated into diverse cultural practices. These include seed treatment with fungicide; preventive application of fungicide before heading; crop rotation; balanced application of fertilizers with nitrogen, potassium, and phosphate; and maintaining a sufficient water level during tillering and flowering stages. However, the most effective way to manage rice blast is by the utilization of resistant cultivars due to its environmental and economic sustainability. Incorporating major blast *R* genes have been traditionally accomplished by classical breeding methods and can be accelerated by the use of marker assisted selection (MAS) [100]. MAS has become a practical tool in cultivar improvement by selecting important traits at the early growth stages based on DNA markers, thus breeders can screen for resistance without having to maintain pathogen culture. MAS is efficient and consistent in the field and greenhouse [101]. MAS is also reliable in dealing with traits whose phenotype is affected by the environment. To date, 99 blast *R* genes have been mapped with closely linked DNA markers; and some of

them can be used for MAS. DNA markers were also developed from portions of cloned *R* genes, such as *Pi-ta* and *Pi-b*, for their introduction into elite rice cultivars using MAS. Markers for *Pita*, one of the most important *R* genes for blast in the United States, were developed [102]; while, linked markers for 4 blast *R* genes (*Pik*, *Pib*, *Pita2*, and *Pii*) are effective against eight to ten races of *M. oryzae* were identified [103]. Using MAS, *R* genes like *Pi1*, *Pi5*, *Piz-5*, and *Pita* have been established in different rice genotypes [82, 100, 104, 105]

6. Future prospects

Blast disease is a moving target where the fungus can rapidly adapt to the host. The major difficulty in controlling rice blast is the durability of genetic resistance. Rice cultivars containing only a single *R* gene to a specific pathogen race often become susceptible over time due to the emergence of new virulent races. In theory, *R* genes can be found in rice germplasm in different rice production areas. Stacking *R* genes with overlapped resistance spectra can lead to long lasting resistance. Knowledge of genetic identity of contemporary *M. oryzae* is crucial for precise deployment of rice cultivars with different *R* genes [104]. Effective blast management also requires unprecedented international cooperation. IRRI and research institutions worldwide have been coordinating their resources for both genotyping using next generation of DNA sequencing and phenotyping at different geographic locations. The knowledge gained by this massive collaborative effort ought to lead to more effective methods to reduce crop loss due to blast disease worldwide.

Acknowledgements

We thank the Arkansas Rice Research and Promotion Board, and the US National Science Foundation (Plant Research Program no. 0701745), Natural Science Foundation of China (Program no. 31000847), Zhejiang Natural Science Foundation (Program no. Y3100577), and Qianjiang Talents Project supported by Science Technology Department of Zhejiang Province (Program no. 2011R10038) for their partial financial supports. USDA is an equal opportunity provider and employer.

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