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IDENTIFICATION OF GENES CONFFERING RESISTANCE TO SOME PHILIPPINE AND VIETNAMESE RACES OF BLAST

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ABSTRACT

The F_{14} recombinant inbred population derived from a cross between japonica rice variety, Lemont and indica variety, Teqing was inoculated by ten Philippine and Vietnamese blast races (M36-1-3-10-1, M64-1-3-9-1, BN111, M39-1-2-21-2, V86010, 142A, 101A, 110A, RB and 138A). The three major resistance genes were identified on chromosomes 2, 11 and 12 as previously reported. The other two resistance loci on chromosomes 6 and 10 were new blast resistance genes. By QTL mapping, out of 48 detected QTLs, fourteen main-effect QTLs (M-QTLs) were identified, nine of which with the resistance allele coming from Teqing, M-QTL bracketed by J01100-AJ13060 on chromosome 11 that was derived from Lemont was mapped to the same genomic region of previously identified qualitative resistance genes; 34 were epistatic QTLs (E-QTLs). Most of partial resitance genes may be race specific in effect and the interactions between QTLs were also important for overall phenotype.

Keywords: Rice Blast, Major genes, QTLs, Mapping.

INTRODUCTION

Blast caused by *Pyricularia grisea* Sacc. has been a major factor limiting rice production worldwide. Rice blast epidemics have been reported in several countries, particularly in Asia, typically resulting in 10-50% yield losses. Breeding for blast resistance is limited by the high degree of pathogenic variability that can overcome resistance in a very short period of time. Breeding and utilization of resistant varieties are still considered the most economic and effective ways for controlling rice blast.

Extensive classical genetic studies on blast resistance have been conducted, resulting in the identification of 13 major genes at eight loci conferring complete resistance (Kiyosawa, 1981). Moreover, many resistance genes have been found, a total of 38 genes were registered and some have been located on rice genetic maps (Nagato and Yoshimora, 1998). In the past few years, more than 15 major genes and 13 quantitative trait loci (QTLs) associated with blast resistance have been localized through the use of molecular marker technology (Wang et al 1994, Yu et al 1991,1996, McCouch et al 1988, 1994, Causse et al 1994, Pan et al 1996, 1999, Naqvi et al 1995, Naqvi and Chattoo 1996, Tabien et al 1998, Chen et al 1999). Three blast resistance genes, *Pi-b*, *Pi-ta*[2], and *Pi-ta*, have been cloned through approaches of positional cloning (Miyamoto et al 1996, Wang et al 1999, Nakamura et al 1997). Sallaud et al (2003) showed that additional resistance genes can be identified in crosses between different rice cultivars after exposure to a large set of isolates from diverse geographic or genetic origins. The identity and behavior of resistance genes in rice are highly dependent on the resistance genotypes of all R genes, the test materials, the pathogen isolate(s) used, and the particular phenological and environment conditions under which plants are inoculated and evaluated (McCouch et al 1994).

The objective of this study was to study the number of major resistance genes present in Lemont and Teqing and to locate main-effect QTLs and epistatic QTLs controlling blast resistance.

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This study was conducted at the Genome Mapping Laboratory, International Rice Research Institute, Philippines and the Plant Pathology Department, Cuu Long Delta Rice Research Institute, Vietnam.

MATERIALS AND METHODS

Phenotyping of recombinant inbred line (RIL)

Reaction to rice blast isolates

The materials included a set of 292 F_{14} RILs (at International Rice Research Institute, Philippines) and a subset of 174 F_{14} RILs (at Cuu Long Delta Rice Research Institute, Vietnam) derived by single seed descent from a cross between a *japonica* variety, Lemont, and an *indica* rice variety, Teqing (Li et al 1995a, 1995b); the parents (Lemont and Teqing); and a susceptible check, Li-Jiang-Xin-Tuan-Hei-Gu (LTH).

Ten *Pyricularia grisea* races were used to evaluate the reaction of plant materials to blast, namely, the Philippine races M36-1-3-10-1, M64-1-3-9-1, BN111, M39-1-2-21-2, and V86010 and the Vietnamese races 142A, 101A, 110A, RB, and 138A . Plants were grown in the greenhouse and inoculated at 21 days after sowing. Disease reaction of each RI line was scored seven days after inoculation using the Standard Evaluation System (SES) rating scale for leaf blast (INGER, 1996). Scores of 0-3 were considered resistant (R) and 4-9 were grouped as susceptible (S).

Genotyping of Recombinant Inbred Lines

A linkage map using 292 F_{13} RILs with a total of 230 markers (2 RFLPs, 121 SSRs, 99 RAPDs, 7 isozyme markers, and 1 morphological markers) was constructed by Yu, Loan and co-workers at the Genome Mapping Laboratory, IRRI, using the software Mapmaker/exp Version 3.0 (Lincoln et al. 1993).

For microsatellite (SSR) analysis, young leaf tissues from a single plant of each RIL was harvested. DNA was extracted by the method of Zheng et al (1995). PCR amplification was performed and PCR products were run on 5% polyacrylamide gel.

Statistical Analysis

Likelihood ratio chi-square tests were used to analyze the discrete greenhouse phenotypic data on resistance or susceptibility to the races and the genotypic data using SAS PROC FREQ (SAS Institute 1996) to detect the association between resistance phenotypes and the marker genotypes, which allowed mapping the major gene involved. To make sure of the association, two rounds of analysis were done. Very large likelihood chi-square statistic values (G^2) would indicate the strong marker-phenotype (resistance) association and suggest the presence of a major gene for vertical resistance. To further evaluate the location of resistance QTLs, the average ratings were treated as quantitative data and were analyzed using QTLMapper v. 1.0 (Wang et al 1999). The threshold of 2.0 LOD or greater was chosen for claiming putative main effect and epistatic QTLs. A relative contribution was calculated as the portion of variance caused by a specific genetic source in the total phenotypic variance, taken as a heritability contributed by that genetic source.

RESULTS

Genotyping of Lemont/Teqing Recombinant

Inbred Population

The linkage map of 230 markers included one morphological trait locus, 7 isozyme loci, 2 RFLP, 121 SSR, and 99 RAPD loci spanning 2314.4 cM and covering 12 rice chromosomes with an average distance of 10.1 cM between adjacent markers. The number of markers per chromosome ranged from 10 to 30. The smallest genetic distance was between OSR26 and RM48 (1.2 cM) on chromosome 2 and the largest, between RM258 and RM333 (33.6 cM) on chromosome 10. The orders of most markers on individual chromosomes were identical to the existing map based on the IR64/Azucena doubled haploid population published by Temnykh et al (2001). On the average, alleles from the indica parent, Teqing, accounted for 51.5%, (ranging from 25 to 78%), slightly higher than the expected 50%. However, individual markers in the RILs favored either the Lemont allele or the Teqing allele. Based on χ^2 tests, segregation

at 145 marker loci (63%) had a good fit with the expected 1:1 ratio and segregation distortion was observed at the remaining loci on all 12 chromosomes (Table 1). The Lemont allele was favored in loci on chromosomes 5 and 12 (17%) while the Teqing allele was in excess at the remaining loci (83%).

Table 1. Linkage map of LemontxTeqing F_{13} recombinant inbred population

PARAMETERS	LEMONT x TEQING
Total length	2314.4 cM
Average distance between markers	10.1 cM
Smallest genetic distance	1.2 cM
Largest genetic distance	33.6 cM
Mean allele frequency ofTeqing	51.5 %
No. of markers with the good fit with the expected segregation 1:1 ratio	154.0

Parental Reactions to Blast Races

Table 2 showed the disease scores of the parents (Lemont and Teqing) for the five Philippine races and five Vietnamese races of blast. Quantitatively, none of the parents were highly susceptible to any of the blast races used as compared to the susceptible check, LJXTHG (rating = 9.0), though race 8 appeared to be most virulent and had average ratings of 7.2 and 6.6 on Lemont and Teqing, respectively. Race 6 was also compatible to both parents.

Phenotypic Variation of the RILs for Blast Resistance

The RILs showed considerable variation for disease ratings (Table 2 and Fig. 1). The frequency distributions of the 292 RILs for disease ratings were continuous except for races 2 and 5 which showed bimodal distribution suggesting the involvement of major resistance gene(s). The segregation ratio of resistant (rating ≤ 3.0) vs. susceptible (rating > 3.0) plants of the RILs varied considerably across different races and in most cases, there was an excess of susceptible plants except for race 10 with a 1:1 ratio, race 4 with an approximate ratio of 15R: 1S, and race 3 with an approximate ratio of 24R: 1S, respectively. Transgressive segregation was detected in the RILs with all levels of resistance, ranging from highly resistant to highly susceptible. These results indicated that the genetics of resistance to leaf blast of the RILs was very complex and depended largely on the races of the pathogen.

Table 2. Leaf blast rating indices for the parental lines and Lemont/Teqing recombinant inbred population to ten races of *P. grisea*

RACE	RATINGS*					
	Recombinant Inbred Lines				Parents	
	mean	SD	min.	max.	Lemont mean	Teqing mean
M36-1-3-10-1 (Race1)	4.7	1.5	1.0	9.0	3.0 (R)	5.4 (S)
M64-1-3-9-1 (Race 2)	4.2	1.9	0.0	8.8	4.8 (S)	3.0 (R)
BN111 (Race 3)	1.4	1.0	0.0	6.5	0.7 (R)	1.9 (R)
M39-1-2-21-2 (Race 4)	1.8	1.0	0.0	9.0	1.7 (R)	2.6 (R)
V86010 (Race 5)	2.3	1.9	0.0	6.8	1.0 (R)	2.8 (R)
142A (Race 6)	4.6	1.2	2.3	8.0	4.0 (S)	4.4 (S)
101A (Race 7)	5.4	2.0	1.6	9.0	6.2 (S)	2.9 (R)
110A (Race 8)	6.6	1.6	3.0	9.0	7.2 (S)	6.6 (S)
RB (Race 9)	4.1	1.8	0.3	8.8	4.0 (S)	2.5 (R)
138A (Race 10)	3.7	1.1	1.0	9.0	3.0 (R)	2.9 (R)

* R: resistant (score of 0-3), S: susceptible (score of 4-9)

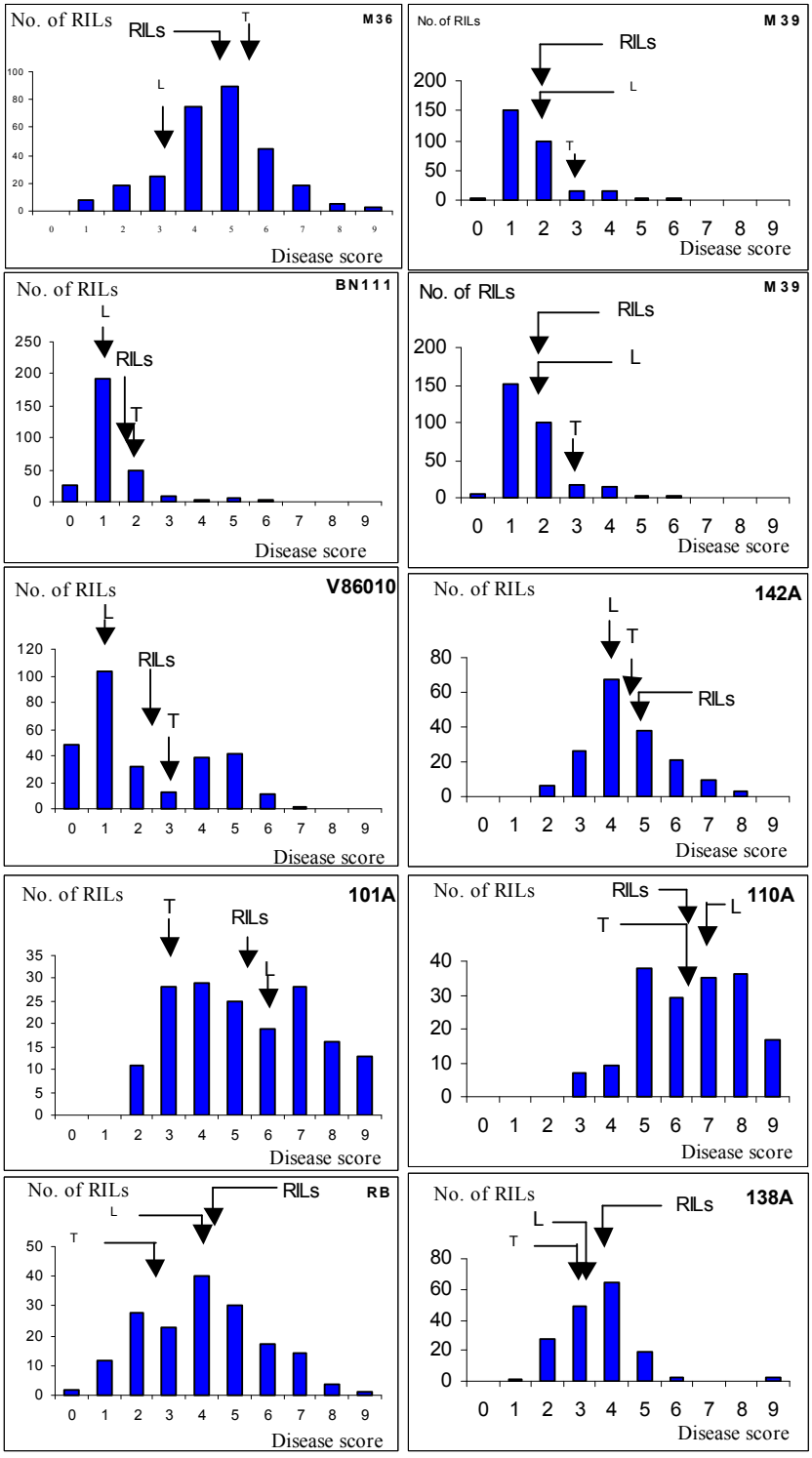

Fig. 1. Frequency distribution of mean scores for resistance to five Philippine rice blast races (M36--1-3-10-1, M64-1-3-9-1, BN111, M39-1-2-21-2, V86010) and five races from Vietnam (142A, 101A, 110A, RB, 138A) in the RI population. Arrows show the mean values of disease score of the two parents and RILs.

Genomic Regions Associated with Complete Resistance to Blast

When the RILs were classified qualitatively into resistant (rating ≤ 3.0) and susceptible ones (rating > 3.0) and subjected to association analyses, 27 genomic regions were found to be associated significantly with resistance to at least one of the ten races based on both unconditional and conditional likelihood chi-square statistics G^2 (Table 3). Out of these, there were 15, 6, 3, 2, 8, 4, 0, 2, 2, and 3 genomic regions associated with resistance to races 1-10, respectively, 12 genomic regions associated with resistance to 2 of the races, and 3 regions with resistance to 3 of the races. The Lemont allele at 16 of these regions and the Teqing allele at 6 loci were associated with resistance. At four loci on chromosomes 6, 7, 8, and 11, resistance was associated with either Lemont or Teqing allele depending on the race. Three loci showing the strongest association with resistance were A10100, J01100, and OSR32 on chromosomes 2, 11, and 12, respectively, which mapped to the same regions of *Pi-tq5*, *Pi-lm2*, and *Pi-tq6* identified in the same population by Tabien et al (2000). Two additional loci near AG15040 on chromosome 6 (G^2 = 19.94) and AK10150 on chromosome 10 (G^2 = 15.49) also showed strong associations with resistance. The Lemont allele at both loci conferred this resistance. These two loci were not reported previously.

The genomic region identified with the highest G^2 (91.2) was near OSR32 on chromosome 12 where the Teqing allele conferred a high level of resistance to race 2 and four US races of blast (Tabien et al, 2000). This region was reportedly harboring a cluster of R genes including *Pi-4(t)* and *Pi-6(t)* (Yu et al, 1991), *Pi-ta* (Wu et al, 1995), *Pi-12(t)* (Zheng et al, 1995), *Pi-62(t)* (Wu et al, 1995), *Pi-20(t)* (Imbe et al, 1997), *Pi-21(t)* (Ahn et al, 1997), and *Pi-tq6* (Tabien et al, 2000).

The genomic region near J01100 in chromosome 11 where the Lemont allele was effective against races 1, 5, and 6 was also of interest because this region was also reported to harbor a cluster of resistance genes including *Pi-1(t)* (Yu et al 1991, Mew et al 1994), *Pi-k* (Inukai et al 1994), *Pi-7(t)* (Wang et al 1994), *Pi-44(t)* (Chen et al 1999), *Pi-lm2* (Tabien et al 2000), and *Pi-18(t)* (Ahn et al 2000). It is, however, difficult to determine the correspondence between the R gene identified in this study to any of the previously reported R genes based on the available data. This gene showed a high level of race specificity. For instance, the Lemont allele had a strong association with resistance to race 5 detected by a G^2 of 49.7, much weaker associations with resistance to races 1 and 6 (G^2 = 9.6 and 16.3), and no association with resistance to the remaining 7 races.

Except for the three loci mentioned above, the remaining 24 genomic regions had relatively weak associations with resistance, though some of these loci (AD14030, AL08120, RM217 and RM254 on chromosomes 2, 4, 6, and 11, respectively) appeared also to be located near previously reported R genes such as *Pi-14(t)* (Pan et al 1998), *Pi-5(t)* (Wang et al 1994), *Pi-10(t)* (Naqvi et al 1995), *Pi-23(t)* (Ahn et al 1997), *Pi-8(t)* (Pan et al 1996), *Pi13(t)* (Pan et al 1998), *Pi-2(t)* (Yu et al 1991), *Pi-z* (Inukai et al 1994), *Pi-22(t)* Ahn et al 1997), and *Pi-7(t)* (Wang et al 1994).

Table 3. Genomic regions showing significant association with blast resistance detected in the Lemont x Teqing (LxT) recombinant inbred population.

Race	Chr.	Marker[a]	G^2 uncon.	G^2 con.	R ALLELE	Race	Chr.	Marker[a]	G^2 uncon.	G^2 con.	R ALLELE
R1	1	RM220	10.63	7.82	L	R1	7	G06080	8.40	6.81	L
R1	1	RM294b		8.57	T	R1	7	Q05150	10.18	9.43	L
R2			7.98	7.29	T	R5			23.28	7.11	L
R2	1	**RM212***	7.21	8.90	T	R1	8	AJ5055	9.20	12.75	L
R9			9.57		T	R1	8	G10080	10.10	10.14	L
R1	2	RM211	7.90	9.39	L	R6	8	**RM210**		10.08	L
R10	2	AD14030	6.04	8.56	T	R9			4.17	7.13	T
R5	2	A10100	16.25	37.84	T	R1	10	AK10150*	15.49		L
R1	4	RM307	10.18	7.91	L	R1	10	RM228	13.44	9.25	L
R5			11.61	8.60	L	R6			13.10	12.00	L
R2	4	D15040	3.86	11.78	L	R5	11	RM20	10.79	11.02	L
R3			5.20	7.40	L	R10			5.41	7.71	L
R1	4	H19075*	5.38	14.70	L	R1	11	A12055	9.26	9.28	L
R4			10.36		L	R8			5.41	8.39	T
R6			6.86	7.54	L	R2	11	RM254		9.40	L
R2	4	AL08120	10.45	13.19	L	R5			21.16	8.46	L
R3	6	RM217	7.46	8.01	T	R6			11.13	12.00	L
R5			10.28	7.63	L	R1	11	**J01100***	8.76	9.58	L
R8	6	K09070	8.91	8.74	T	R5			49.66		L
R1	6	AG15040	8.49	19.94	L	R6			16.26		L
R1	7	**AL08025**		7.25	L	R2	12	OSR32*	91.18		T
R10				8.98	T	R4	12	P01065	13.65	9.53	L
						R5				7.33	L

$G^2_{uncon.}$ is the likelihood ratio chi square statistic in unconditional test.
$G^2_{con.}$ is the likelihood ratio chi square statistic in conditional test. The significant values of G^2 at P = 0.01, 0.001, and 0.0001are 6.64, 10.83, and 15.12, respectively.
*Markers with highest G^2 value are used in conditional tests.
Shaded values are the highest G^2 values at P < 0.0001.
[a] Bold markers are associated with the detected main effect QTLs affecting blast resistance.

QTLs Affecting Blast Resistance

Table 4 and Fig. 2 showed the 14 main-effect QTLs (M-QTLs) affecting resistance to 10 blast races. All these QTLs were detected by LOD scores greater than 2.5 except one (LOD = 1.71). Of the 14 M-QTLs, four were associated with markers for vertical resistance (Table 3). Each of three M-QTLs (*QBr3*, *QBr5,* and *QBr8*) located in the intervals between RM251 and RM282 on chromosome 3, between RM163 and RM161 on chromosome 5, and between RM223 and RM210 on chromosome 8 was effective against two of the races. The Lemont allele at *QBr8* was associated with resistance to races 6 and 10 while the Teqing allele at *QBr3* resulted in resistance to both races 4 and 8. Interestingly, the Lemont allele at *QBr5* was associated with resistance to race 10 while the Teqing allele at this locus resulted in resistance to race 2. The remaining M-QTLs were each effective to one single race. Associated with resistance was the Teqing allele at nine of the loci (*QBr1a*, *QBr1b*, *QBr2a*, *QBr2b*, *QBr2c*, *QBr3, QBr7*, *QBr10*, and *QBr11a*) and the Lemont allele at the remaining 3 loci (*QBr4*, *QBr9,* and *QBr11b*).

Table 4. Main effect QTLs affecting SES ratings detected in Lemont/Teqing RILs inoculated with ten rice blast races.

M-QTL	CHR.	MARKER INTERVAL	RACE	LOD	A	R^2 (%)
QBr1a	1	E11075 – RM84	9	2.61	0.46	6.4
QBr1b	1	**G04035 – RM212**	8	6.01	0.60	14.9
QBr2a	2	RM27 – RM324	7	2.88	0.40	7.4
QBr2b	2	RM221 – RM6	6	2.45	0.23	4.2
QBr2c	2	RM138 – OSR26	9	3.05	0.48	7.0
QBr3	3	RM251-RM282	4	2.26	0.19	3.5
			8	2.52	0.38	6.9
QBr4	4	**O07080 – AB11070**	9	1.71	-0.38	4.4
QBr5	5	RM163 – RM161	2	2.62	0.33	4.7
			10	2.92	-0.32	7.1
QBr7	7	**AL08025 – Q05070**	10	2.45	0.30	5.6
QBr8	8	**RM223 – RM210**	6	6.11	-0.45	15.6
			10	4.23	-0.42	12.6
QBr9	9	RM278 – OSR28	8	2.93	-0.37	6.4
QBr10	10	RM239 – D02090	6	4.61	0.45	16.1
QBr11a	11	RM229 – RM21	3	2.68	0.20	4.1
QBr11b	11	**J01100 – AJ13060**	6	6.95	-0.39	12.1

A: additive genetic effect of QTLs due to substitution of Lemont allele by Teqing allele. Positive and negative values show that allele resulting in an increase in resistance is from Teqing and Lemont, respectively.
R^2 (%): proportion of phenotypic variation explained by a single QTL. Bold markers are also associated with vertical resistance to one or more races by association analyses (Table 3).

Table 5 and Fig. 2 showed 34 pairs of digenic epistatic QTLs (E-QTLs) for resistance to 10 blast races. All these QTLs were detected by LOD scores greater than 2.5. Of the E-QTLs, 10 were associated with markers for vertical resistance (Table 3) and 9 were associated with markers for M-QTLs for resistance (Table 4). Positive epistatic effects indicated that the recombinant types of alleles at the interaction loci were expected to result in resistance, and negative epistatic effect showed that parental-type interactions tended to result in resistance.

The contributions of E-QTLs were higher (14.6-51.0%) than those of main effect QTLs (M-QTLs) (3.5-16.1%). M-QTL bracketed by J01100-AJ13060 mapped to the same genomic region of previously identified qualitative resistance gene. This result is in agreement with other studies indicating that this QTL was a "defeated" version of qualitative resistance gene, though only more precise mapping and gene cloning can resolve this definitely (Young 1996, Li et al 1999). Eleven resistance QTLs detected by the race 142A highly contributed to phenotypic variation (total R^2 = 99%).

Table 5. Digenic epistatic QTLs affecting SES ratings detected in Lemont/Teqing RILs inoculated with ten rice blast races.

Race	Chr.	Marker interval *I*	Chr.	Marker interval *J*	LOD	A_I	A_J	AA_{IJ}	R^2 (%)
Race 1	1	RM23-W17065	4	RM317-AP13060	4.80	-	-	0.46***	8.7
	2	RM27-RM324	12	**OSR20-OSR32**	4.13	-	-	-0.30**	3.6
	2	RM29-K02050	5	RM163-RM161	3.62	-	-0.21*	-0.44**	8.0
	3	RM148-RM85	12	**RM17-P01065**	4.08	-	-	0.37**	5.5
	4	AP13060-RM255	12	Sdh1-A19080	5.24	-	-	0.45***	8.0
	10	RM216-G03030	11	**J01100-AJ13060**	4.80	-	-	-0.34**	4.7
Race 2	1	F04025-RM9	3	Q05075-A10050	4.43	-	-	-0.59***	7.6
	2	K02050-RM263	4	**F05050-RM307**	3.35	-	-	0.52**	5.8
	5	AP05170-RM249	6	A07020-G15110	6.59	-	-0.33*	0.74***	12.2
	7	AL09170-Q120100	8	A12100-AP20120	4.97	-	-	-0.72***	11.4
Race 4	2	RM221-RM6	4	O07080-AB11070	3.99	-	-	0.27***	8.1
	3	Q05075-A10050	5	RM161-OSR49	3.33	-	-	0.24**	6.5
Race 5	2	RM221-RM6	7	AL08025-Q05070	3.75	-	-	-0.46***	5.9
	4	O07080-AB11070	10	**RM222-AK10150**	4.84	-	-	-0.48**	6.4
	6	D06095-AB18130	9	RM316-RM219	4.05	-	-	-0.44**	5.3
	6	D06095-AB18130	11	RM209-RM229	3.74	-	-	-0.50**	6.9
Race 6	1	RM246-AL08055	2	RM6-RM250	7.29	-	-	0.66***	17.7
	2	K02050-RM263	2	RM208-RM138	3.04	-	-	-0.29**	3.3
	2	RM221-RM6	8	**RM223-RM210**	4.88	0.25**	-0.19*	-0.17*	1.9
	6	RM30-RM340	11	AA18045-RM202	5.34	-	-	-0.47***	9.0
	8	**RM25-AJ5055**	9	RM278-OSR28	5.44	-	-	-0.42***	7.0
	8	**G10080-D06045**	12	RM277-RM260	7.64	-0.17*	-	-0.49***	9.7
	9	RM257-RM242	11	**J01100-AJ13060**	6.77	-	-0.37***	0.19*	2.4
Race 7	2	RM29-K02050	3	A10050-AF07160	3.57	-	-	-0.59**	8.6
	5	RM249-RM163	11	AA18045-RM202	6.21	-	-	0.80***	16.2
Race 8	2	RM29-K02050	7	J14110-G06080	7.13	-	-	-0.71***	17.6
	3	G06075-RM168	12	G193-RM19	4.72	0.30*	-	0.50***	8.6
	3	RM55-O07130	6	**RM225-RM217**	5.04	-	-	-0.57***	11.2
Race 9	1	RM9-RM5	11	**OSR6-J01100**	4.08	-	-	0.58***	9.2
	1	AL08055-AP13110	2	RM6-RM250	5.21	-	-	0.70***	13.4
	2	OSR14-OSR17	12	AB17100-A19080	3.81	-	-	0.62***	10.7
	2	RM138-OSR26	9	RM205-AG08050	7.61	0.37*	-	-0.61***	10.4
Race 10	1	OSR3-RM104	4	RM137-AP13060	2.84	-	-	-0.31**	8.2
	6	**K09070-RM50**	6	K09070-O02075	3.54	-	-	0.37**	11.2

A_I and A_J: the main effects of the loci i and j, arising from the substitution of the Lemont allele by the Teqing allele
A_{IJ}: the epistatic effect between loci i and j.
*, **, ***: significant levels of P<0.05, 0.001, 0.0001, respectively
The underlined markers are associated with M-QTLs affecting blast resistance (Table 4) and the bold markers are associated with vertical resistance (Table 3).

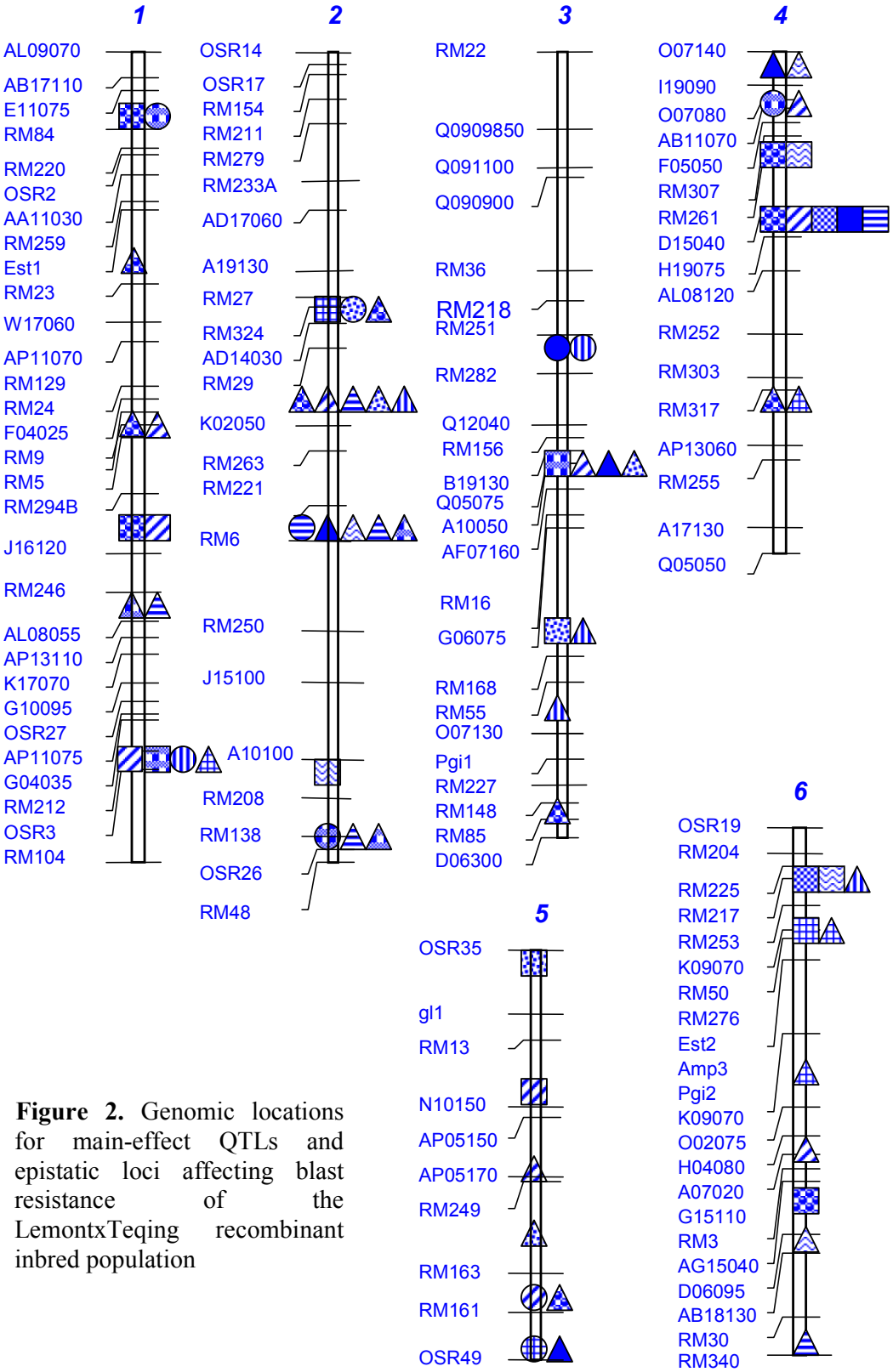

Figure 2. Genomic locations for main-effect QTLs and epistatic loci affecting blast resistance of the LemontxTeqing recombinant inbred population

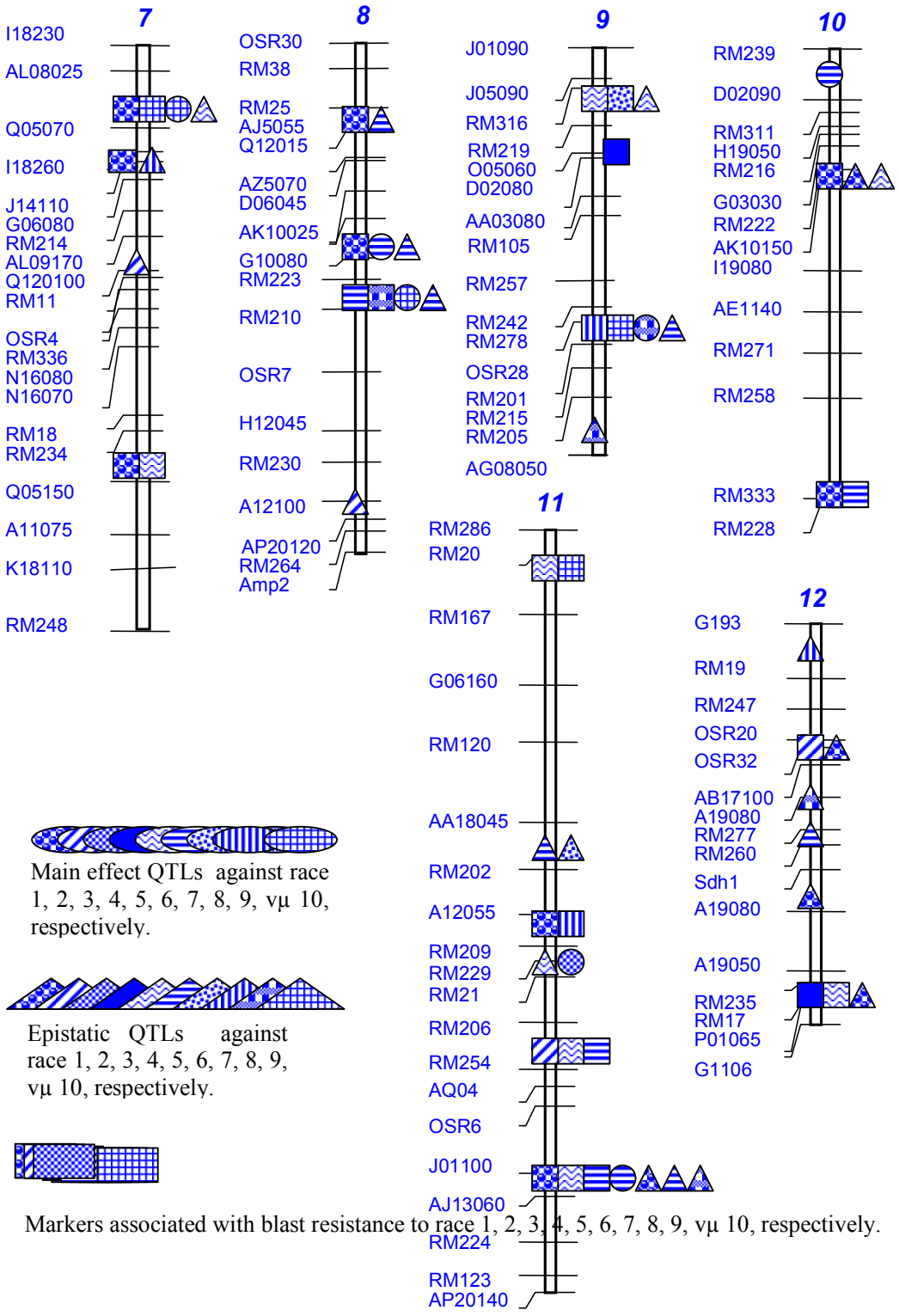

Figure 2. Continued...

DISCUSSION

Two types of resistance of rice plants to blast, the true or vertical resistance and field or horizontal resistance, have long been recognized (Kiyosawa 1970, van der Plank 1963). The former is known to be associated with hypersensitivity and controlled by single major genes and the latter is quantitative and presumably controlled by polygenes. The ten-scale evaluation system of 0-9 used in the present study could actually measure both types of resistance, in which ratings 0-3 were considered resistant (plants showing the hypersensitive reaction) and scores 4-9 were considered susceptible and measure the severity of the disease. The most striking observation in this experiment was that, when the disease score data were treated qualitatively, there were excessive susceptible lines in the RI population to most races, which could not be explained by any simple genetic models. For the only 2 cases (races 10 and 4) where the reactions of RILs segregated in the expected 1-gene and 4-gene models, the mapping results by association analyses did not reveal involvement of any major genes. The only three major R genes discovered in this study were each associated strongly with hypersensitivity to races 2 and 5, consistent with the bimodal distributions of the RILs for their reactions to these races. According to their genomic locations and resistance alleles, the three genes were apparently *Pi-tq5*, *Pi-lm2,* and *Pi-tq6* on chromosomes 2, 11, and 12 identified in the same population by Tabien et al (2000). The strength of the associations, however, of the three R genes with hypersensitivity was much lower than the perfect 100% penetrance which would produce a $G^2 > 200.00$ (Zhikang Li, personal communication). The hypersensitive reactions of some RILs to the remaining 8 races of blast appeared to be due to several genes with much lower penetrance as suggested by relatively low values of G^2 statistic. Thus, the differential and quantitative variation in penetrance of R genes to different races of blast appears to provide an adequate explanation for the "abnormal" segregation ratios for resistance of the RILs. This explanation was also consistent with the presence of resistant lines to races 6 and 8 when both parents were compatible and with the observations that many of the identified QTLs fell in the genomic regions that were also associated with hypersensitivity.

The identification of many QTLs affecting quantitative resistance to the ten blast races was expected given the compatibility of the parents to races 6 and 8, and large variation for susceptibility (score > 3.0) of the RILs to most races. In this study, 11 of the 14 M-QTLs and all E-QTL pairs were each effective against only one race and the remaining three were each effective against two of the races. This high level of race-specificity of the identified QTLs was unexpected from the common view of "non-race specificity" presumed for the field or horizontal resistance to blast (Van der Plank 1968). It is not surprising if the observed race specificity of QTLs for partial resistance is in part due to the partial penetrance of many race specific R genes that were segregating in the RILs. Thus, results from this study and from many previous studies (Leonard-Schipper et al 1994, Young, 1996, Lespinasse et al 2000) suggest that quantitative or field resistance may not be non-race specific.

The frequent breakdown of high level complete resistance (hypersensitivity) due to the appearance of virulent races of the pathogen has imposed the greatest challenge to plant breeders to develop durably resistant rice cultivars. The observed presence of a large number of resistance loci and the high level of race specificity of these loci suggest that accumulation (pyramiding) of effective resistance alleles at as many as possible resistance loci should be one of the effective ways to achieve durable resistance to blast. The striking differentiation of alleles at many of the resistance loci for their reactions to blast races from geographically different regions suggests that effective alleles at resistance loci are more likely present in the germplasm from different geographic or ecological regions where the virulence of the pathogen races have become differentiated as a result of co-evolution in adaptation to the local races of the pathogen.

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SUMMARY IN VIETNAMESE

Xác định gen kháng bệnh cháy lá đối với các nòi chính ở Việt nam và Philippines

Quần thể F14 các dòng cận giao tái tổ hợp có nguồn gốc từ cặp lai Japonica Lemont và Indica Teqing được đánh giá phản ứng đối với 10 nòi nấm cháy lá từ Philippines và Việt nam. 3 gen chủ lực đã được xác định trên nhiễm sắc thể 2, 11, 12, nằm trên cùng một vị trí đối với 3 gen chủ lực đã được công bố trong nghiên cứu trước đây. Hai gen kháng bệnh cháy lá mới đã được tìm thấy trên nhiễm sắc thể số 6 và 10. Trong 48 QTLs, có 14 QTL chính và 34 QTL có tính tương tác. Trong 14 QTL chính có 9 QTLs có nguồn gốc từ Teqing, QTL ở giữa 2 marker J01100 và AJ13060 có nguồn gốc từ Lemont và trên cùng một vị trí với gen chủ lực. Đa số các gen kháng ngang có sự chuyên tính về nòi và mối tương tác giữa các QTL đã giữ vai trò quan trọng trong sự biểu hiện của tính kháng.