Are the Dominant and Recessive Plant Disease Resistance Genes Similar?: A Case Study of Rice R Genes and *Xanthomonas oryzae* pv. *oryzae* Races

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ABSTRACT

The resistance of rice to its bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) has both qualitative and quantitative components that were investigated using three near-isogenic line sets for four resistance (R) genes (*Xa4*, *xa5*, *xa13*, and *Xa21)* and 12 *Xoo* races. Our results indicate that these two resistance components of rice plants were associated with the properties of the R genes. The qualitative component of the R genes was reflected by their large effects against corresponding avirulent *Xoo* races. The quantitative component of the R genes was their residual effects against corresponding virulent races and their epistatic effects, which together could lead to high-level resistance in a race-specific manner. Our results revealed important differences between the different types of R genes. Two R genes, *Xa4* and *Xa21*, showed complete dominance against the avirulent *Xoo* races and had large residual effects against virulent ones. They acted independently and cumulatively, suggesting they are involved in different pathways of the rice defensive system. The third R gene, *xa5*, showed partial dominance or additivity to the avirulent *Xoo* races and had relatively small but significant residual effects against the virulent races. In contrast, *xa13* was completely recessive, had no residual effects against the virulent races, and showed more pronounced race specificity. There was a strong interaction leading to increased resistance between *xa13* and *xa5* and between either of them and *Xa4* or *Xa21*, suggesting their regulatory roles in the rice defensive pathway(s). Our results indicated that high-level and durable resistance to *Xoo* should be more efficiently achieved by pyramiding different types of R genes.

PLANT disease resistance is often controlled by Mendelian genes and follows a gene-for-gene relationship in many plant species and their pathogens (Flor 1971). According to this theory, there are many resistance (R) genes in a plant species against each of its pathogens and there is a corresponding avirulence gene in the pathogen population for every R gene in the host plant. This theory has been well demonstrated in cases where plant resistance is associated with hypersensitivity. However, a clear-cut resistant phenotype like hypersensitivity does not always exist in many other cases and plant resistance often shows both qualitative and quantitative components. The qualitative resistance in many plant-pathogen relationships is hypersensitive, race specific, and governed by interactions between avirulence genes in pathogens and resistance genes in hosts, while the quantitative resistance is nonhypersensitive, presumably nonrace specific, and controlled by polygenes (Nelson 1972).

Recent advances in DNA marker technology and genomic research have provided powerful tools for addressing many questions about genetics of interactions between plants and their pathogens. Large numbers of R genes in several plant species have been accurately mapped to their corresponding genomic locations (Michelmore and Meyers 1998). To date, at least 18 R genes including 17 dominant and 1 recessive one in nine plant species have been cloned (*cf.* Bent 1996). A general model of plant defense responses to pathogen invasion involving complex biochemical pathways has been proposed, explaining the molecular basis of the gene-for-gene theory (Dangl 1998). Several hypotheses in this regard can be summarized below. First, R genes in a plant species are distributed in clusters in many locations of its genome (Crute and Pink 1996; Botella *et al.* 1997). Second, R genes from different species appear to share sequence similarities in certain domains of their gene products such as leucine-rich repeats (LRR) and nucleotide-binding sites (NBS; Bent 1996). Third, different R genes from a particular plant species against one of its pathogens may not show high homology in DNA sequence. Fourth, quantitative trait loci (QTL) for partial resistance tend to map at genomic regions in the vicinity of clustered R genes (Wang *et al.* 1994; Li *et al.* 1999). However, many important questions remain to be answered regarding various R genes. For instance, do

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different R genes act independently or synergistically? Are genes conferring qualitative effects different from genes conferring quantitative effects? What are the differences between dominant and recessive R genes?

Rice (*Oryza sativa* L.) and its bacterial blight (BB) pathogen *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) present an excellent opportunity for addressing many of the above questions. Resistance in rice to *Xoo* is known to have both qualitative and quantitative components (Mew 1987; Zhang and Mew 1988; Koch and Parlevliet 1991; Li *et al.* 1999). There are at least 20 major genes that confer resistance to *Xoo* and follow a gene-for-gene relationship in rice (Ogawa and Khush 1988; Lin *et al.* 1996; Ogawa 1996; Zhang *et al.* 1996). Most of these R genes are dominant and have been recently mapped to respective genomic locations (Ronald *et al.* 1992; Yoshimura *et al.* 1992; Causse *et al.* 1994; Lin *et al.* 1996; Ogawa 1996; Zhang *et al.* 1996). Two of these genes (*Xa21* and *Xa1*) have been cloned and molecularly characterized (Song *et al.* 1995; Yoshimura *et al.* 1998). Recently, near-isogenic lines (NILs) with pyramids of the R genes have been developed through molecular marker-aided backcross breeding, providing valuable materials for detailed characterization of the R genes (Huang *et al.* 1997; Sanchez *et al.* 2000).

In this study, we address the questions mentioned above on the basis of quantitative analyses of responses of three isogenic line sets carrying three and four R genes and gene combinations to 12 *Xoo* races from the Philippines and India.

MATERIALS AND METHODS

Plant materials: Three sets of rice NILs carrying genes for resistance to BB were used in the study (Table 1). The first set included 16 NILs carrying four R genes (*Xa4*, *xa5*, *xa13*, and *Xa21*) in all possible combinations in the IR24 (*indica*) genetic background and four F_1's between IR24 and their single-gene NILs. The single-R-gene IR24 NILs were developed by more than six to seven generations of backcrossing plus phenotypic selection and these lines are phenotypically identical except for their reactions to the differential *Xoo* races (Yoshimura *et al.* 1992, 1996). The multi-R-gene NILs were developed by crossing the single-R-gene NILs and performing marker-aided selection (Huang *et al.* 1997). The second and third sets each contained 8 BC_3F_3 NILs carrying three R genes (*xa5*, *xa13*, and *Xa21*) and their possible combinations in the genetic backgrounds of two new plant type (NPT) lines, IR65598-112 (NPT2) and IR65600-96 (NPT3), developed by marker-aided selection (Sanchez *et al.* 2000). NPT lines are tropical japonicas.

Bacterial blight inoculation and evaluation: The three sets of NILs were evaluated separately for their resistance to six Philippine *Xoo* races in the screenhouse during 1995 and 1997 at International Rice Research Institute (IRRI), as described previously (Huang *et al.* 1997; Sanchez *et al.* 2000). In experiment 1, the IR24 NILs were evaluated twice in the 1995 dry and wet seasons. In each of the two seasons, plants were sown in the screenhouse conditions at IRRI with three replications for each of the NILs and managed using standard cultural practices. Insecticides were used regularly to keep the plants from damage from insects. Six Philippine *Xoo* races (race 1, PXO61; race 2, PXO86; race 3, PXO79; race 4, PXO71; race 5, PXO112; and race 6, PXO99), which could clearly differentiate *Xa4*, *xa5*, and *xa13*, were used to inoculate the NILs (Mew 1987). At the maximum tillering stage, five to six leaves of each of the four plants in a plot were inoculated with the *Xoo* races using the leaf clipping method (Kauffman *et al.* 1973). The inoculum of each of the *Xoo* races was prepared by suspending the bacterial mass in sterile water to a concentration of $\sim 10^9$ cells/ml. For the susceptible check (IR24), a total of 24 plants (12 plants of two plots) in each replication were inoculated. The lesion lengths of the inoculated leaves were measured 18 days after inoculation. In experiment 2, the partial set of the IR24 NILs and the two NPT NIL sets containing three BB resistance genes (*xa5*, *xa13*, and *Xa21*) and their combinations were planted in two-row plots in the screenhouse with three replications for each of the plots in the 1997–1998 dry season. Nine leaves of 3 different plants (three leaves/plant) in each of the NILs were clip inoculated with the same six *Xoo* races at the maximum tillering stage. Three plots of the susceptible parents (IR24, NPT2, and NPT3) in each replication were inoculated as checks. Inoculum was prepared by suspending the bacterial mass in sterile water to a concentration of $\sim 10^9$ cells/ml. Lesion length was measured on each of the inoculated leaves 18 days after inoculation. In experiment 3, the IR24 NILs containing *xa5*, *xa13*, *Xa21* and their combinations were evaluated in the greenhouse of Punjab Agricultural University, Ludhiana (Northern India) in 1997 with six representative *Xoo* isolates of Punjab (Table 2). Four plants per NIL were grown in pots and the three youngest fully expanded leaves on each of the plants were clip inoculated the same way as above. The preparation of the inoculum was done in the same way except that lesion length was measured on each of the inoculated leaves 16 days after inoculation.

Data analyses: $\log_e$-transformed lesion length data were used for analyses. ANOVA was performed to partition variance components of each of the experiments due to (1) NIL genotype, (2) *Xoo* races, (3) NIL × race interaction, (4) season, etc., using SAS GLM. Pairwise *t*-tests were performed for all possible comparisons between different NILs for lesion length differences. The effects, measured as $\log_e$-transformed lesion length (LL), of individual BB resistance genes were estimated as deviations due to substitution of the susceptible allele by the resistant one at each of the resistance loci against the six *Xoo* races in different NIL genotypes. Standard *t*-tests were performed to determine if the estimated gene effects were equal to zero. The epistatic effects between two, three, and four resistance genes were estimated from the mean values of the $\log_e$-transformed lesion length measurements of the NILs on the basis of the genetic expectations formulated by Mather and Jinks (1982).

RESULTS

Table 2 shows that in experiment 1, 68.3% of the total variation for lesion length was due to differences among the 16 IR24 NILs. Of this variance component, 8.0% was due to differences among the 4 one-gene NILs, 10.9% to differences among the 6 two-gene NILs, and only 2.9% to differences among the 4 three-gene NILs. There was a general pattern regarding the resistance levels of the NILs, *i.e.*, the four-gene NIL ≥ three-gene NILs ≥ two-gene NILs ≥ one-gene NILs ≥ IR24. The differences among the *Xoo* races and NIL × race interac-

TABLE 1

Near-isogenic lines (NILs) with different resistance genes to bacterial blight developed in the genetic backgrounds of IR24 (indica type), new plant type (NPT2), and NPT3 (japonica type)

IR24 NILs	Gene(s)	NPT NILs [a]	Gene(s)
IR24		NPT3	
IRBB4	*Xa4*	NPT31	*xa5*
IRBB5	*xa5*	NPT32	*xa13*
IRBB13	*xa13*	NPT33	*Xa21*
IRBB21	*Xa21*	NPT34	*xa5/xa13*
IRBB50	*Xa4/xa5*	NPT35	*xa5/Xa21*
IRBB51	*Xa4/xa13*	NPT36	*xa13/Xa21*
IRBB52	*Xa4/Xa21*	NPT37	*xa5/xa13/Xa21*
IRBB53	*xa5/xa13*	NPT2	
IRBB54	*xa5/Xa21*	NPT21	*xa5*
IRBB55	*xa13/Xa21*	NPT22	*xa13*
IRBB56	*Xa4/xa5/xa13*	NPT23	*Xa21*
IRBB57	*Xa4/xa5/Xa21*	NPT24	*xa5/xa13*
IRBB58	*Xa4/xa13/Xa21*	NPT25	*xa5/Xa21*
IRBB59	*xa5/xa13/Xa21*	NPT26	*xa13/Xa21*
IRBB60	*Xa4/xa5/xa13/Xa21*	NPT27	*xa5/xa13/Xa21*
IR24/IRBB4 F_1	*Xa4/xa4*		
IR24/IRBB5 F_1	*Xa5/xa5*		
IR24/IRBB13 F_1	*Xa13/xa13*		
IR24/IRBB21 F_1	*Xa21/xa21*		

[a] NPT is new plant type, and NPT2 and NPT3 are IR65598-112 and IR65606-96.

tion explained 8.3 and 16.2% of the total variation, respectively. Interestingly, the one-gene and two-gene NILs showed greater interactions with race as compared with the other NILs. The residual including differences between seasons, season × NILs, and season × race, etc. accounted for only 7.1% of the total variation.

Table 3 shows that in experiment 2 the among-NIL variation explained 66.7, 75.2, and 57.3% of the total variances in the IR24, NPT2, and NPT3 NIL sets, respectively. The maximum difference was detected between one-gene NILs and the susceptible parents (IR24, NPT2, and NPT3). Differences between one-gene and two-gene NILs and between two-gene and three-gene NILs were less pronounced. Variances due to interactions between NILs and *Xoo* races accounted for 14.3, 12.2, and 20.1% of the total variation for the IR24, NPT2, and NPT3 NIL sets, respectively. The variation between the *Xoo* races explained only 2.5, 1.6, and 4.4% of the total variation, respectively. A similar pattern was observed for IR24 NILs against six Indian *Xoo* races in experiment 3 in which >85% of total variation was due to differences among the NILs and the NIL × race interactions.

Table 4 shows the main effects of the R genes against the 12 *Xoo* races in the IR24 genetic background. *Xa4* was effective against Philippine *Xoo* races 1 and 5 with a mean effect of −1.24. It also showed a strong residual effect (−0.66, −0.67, and −0.69) against the virulent Philippine *Xoo* races 2, 3, and 4 and a weak effect (−0.20) against race 6. *Xa21* conferred resistance against all 6 Philippine and 5 of the 6 Indian *Xoo* races. Its effect was consistent against all Philippine races but varied considerably against the Indian races. It had a large residual effect of −0.47 against the only virulent Indian race, PXO4. On the other hand, *xa5* conferred resistance to Philippine races 1, 2, 3, and 5 and the Indian race PXO1. It conferred moderate resistance to Indian races PXO13 and PXO17 and had relatively small but consistent residual effects (−0.22 and −0.25) against Philippine races 4 and 6 and Indian races 4, 6, and 8. However, the F_1 plants between IR24 and its *xa5* NIL indicated that *xa5* showed partial dominance to Philippine races 1 and 3 but additivity to races 2 and 5 (data not shown). The only recessive gene, *xa13*, was effective only to Philippine race 6 and Indian race PXO8 and had a small insignificant mean residual effect of −0.06 against the 10 virulent *Xoo* races (Table 2).

Tables 5 and 6 show the epistatic effects between or among the R genes estimated in the IR24 and NPT genetic backgrounds. Two types of interactions were recognized: one leading to reduced lesion length and the other to increased lesion length. Highly significant negative epistatic effects (for increased resistance) were detected only between the recessive R genes (*xa5* and *xa13*) or between a dominant R gene and a recessive one and occurred more often against their common virulent races. For example, together, *xa5* and *xa13* conferred a high level of resistance to three of their common virulent *Xoo* races (Indian races PXO4, PXO6, and Philippine race 4). Similarly, when in pair, *xa13/Xa4*

TABLE 2

ANOVA results of $\log_e$-transformed lesion length data for IR24 NILs caused by six Philippine *Xanthomonas oryzae* pv. *Oryzae* races

Source [a]	d.f.	Sum of squares	Mean square	*F*	R^2 (%)
NILs	15	4058.5	270.6	5279.3	68.3
One-gene NILs	3	324.4	108.1	2110.2	
Two-gene NILs	5	441.9	88.4	1724.4	
Three-gene NILs	3	117.7	39.2	765.5	
0 *vs.* 1	1	201.4	201.4	3929.2	
1 *vs.* 2	1	527.2	527.2	10287.5	
2 *vs.* 3	1	624.9	624.9	12192.7	
3 *vs.* 4	1	50.9	50.9	993.8	
Races	5	494.4	98.9	1929.6	8.3
IR24	5	1.9	0.4	7.5	
One-gene NILs	5	49.4	9.9	192.9	
Two-gene NILs	5	181.4	36.3	708.0	
Three-gene NILs	5	385.6	77.1	1504.8	
Four-gene NILs	5	130.5	26.1	509.1	
0 *vs.* 1	5	3.0	0.6	11.7	
1 *vs.* 2	5	166.6	33.3	650.1	
2 *vs.* 3	5	40.9	8.2	159.5	
3 *vs.* 4	5	39.8	8.0	155.3	
NILs × races	75	962.7	12.8	250.5	16.2
One-gene NILs	15	226.2	15.1	294.2	
Two-gene NILs	25	419.8	16.8	327.6	
Three-gene NILs	15	107.5	7.2	139.8	
0 *vs.* 1	1	8.5	8.5	165.7	
1 *vs.* 2	5	19.1	3.8	74.7	
2 *vs.* 3	5	77.4	15.5	302.0	
3 *vs.* 4	5	37.0	7.4	144.5	
Replication	2	0.0	0.0	0.3	0.0
Residual	3646	424.2	0.12		7.1

[a] One-gene, two-gene, and three-gene NILs represent the differences among NILs with only one R gene, among those with two R genes, and among those with three R genes. 0 *vs.* 1, 1 *vs.* 2, 2 *vs.* 3, and 3 *vs.* 4 represent the differences between IR24 and the mean one-gene NILs, between the mean one-gene NILs and mean two-gene NILs, between the mean two-gene NILs and mean three-gene NILs, and between the mean three-gene NILs and the four-gene NIL, respectively.

and *xa5/Xa4* resulted in a high level of resistance against their common virulent *Xoo* race (Philippine race 4), and so did *xa5/Xa21* and *xa13/Xa21* against Indian race PXO4. In contrast, the positive epistatic effects for increased lesion length were detected almost exclusively in the incompatible cases where at least one of the interacting R genes was resistant to the involved *Xoo* races (Tables 3 and 5). It should be pointed out that because of data transformation these significant effects did not reflect the true interactions between the R genes but rather they indicated that, when challenged against avirulent races, these R genes acted in an additive manner.

ANOVA (Tables 2 and 3) also indicated that variances in lesion length among the different susceptible recurrent parents of the NILs, variation in aggressiveness among the *Xoo* races, and the parent × race interactions were highly significant. The three components explained 17.8, 11.4, and 8.3% of the total variation, respectively. On average, NPT2 had longer lesions than NPT3 and IR24 (Table 7). The Philippine *Xoo* races 5 and 6 were most aggressive and apparently adapted well to *japonica* (NPT2 and 3) and less so to the *indica* type (Table 7).

The NIL genetic backgrounds had significant impacts on the estimated main and epistatic effects of the R genes. In the incompatible cases, *xa13* had a consistent effect against race 6 in all three genetic backgrounds. However, *Xa21* and *xa5* had greater effects in the indica genetic background (IR24) than in the japonica ones (NPT2 and 3), and so did *xa5* regarding its residual effects in the compatible cases (Table 7). Similarly, highly significant epistatic effects between *xa13* and *Xa21* leading to increased resistance against Philippine race 4, and that between *xa5* and *Xa21* against Philippine race 6, were detected only in the NPT genetic backgrounds but not in the IR24 NILs (Table 5). The epistatic effect of *xa5* and *xa13* leading to resistance against race 4 was much greater in IR24 and NPT2 genetic backgrounds than in NPT3.

TABLE 3

ANOVA results of $\log_e$-transformed lesion length data for NPT2 (IR65598-112), NPT3 (IR65600-96), and IR24 NILs caused by six Philippine *Xanthomonas oryzae* pv. *Oryzae* races

		Recurrent parent								
		NPT2			NPT3			IR24		
Source[a]	d.f.	MS	*F*	R^2 (%)	MS	*F*	R^2 (%)	MS	*F*	R^2 (%)
NILs	7	88.6	323.9	75.2	65.5	239.5	57.3	383.7	1402.1	66.7
One-gene NILs	2	26.0	94.9		25.3	92.5		164.7	601.8	
Two-gene NILs	2	11.6	42.5		10.4	38.0		33.0	120.6	
0 *vs.* 1	1	111.3	406.8		102.7	375.3		530.3	1938.1	
1 *vs.* 2	1	63.8	233.1		76.4	279.3		249.0	909.8	
2 *vs.* 3	1	8.2	29.9		0.2	0.9		95.5	348.8	
Races	5	2.6	9.4	1.6	7.0	25.6	4.4	17.4	63.5	2.5
NPT2	5	2.4	8.7		4.4	16.1		0.5	2.0	
One-gene NILs	5	0.9	3.4		2.9	10.6		16.5	60.4	
Two-gene NILs	5	5.1	18.5		2.6	9.4		8.8	32.0	
Three-gene NILs	5	1.4	5.1		1.9	6.9				
0 *vs.* 1	5	3.2	11.5		5.9	21.6		12.6	46.2	
1 *vs.* 2	5	2.4	8.6		2.9	10.4		20.2	73.9	
2 *vs.* 3	5	3.3	11.9		3.8	14.0		9.6	35.2	
NILs × races	35	2.9	10.5	12.2	4.6	16.8	20.1	16.4	60.0	14.3
One-gene NILs	10	6.3	22.9		12.5	45.5		38.6	140.9	
Two-gene NILs	10	0.6	2.2		1.3	4.7		8.2	30.1	
0 *vs.* 1	5	0.1	0.3		1.5	5.4		4.2	15.5	
1 *vs.* 2	5	3.6	13.1		2.7	9.7		5.0	18.1	
2 *vs.* 3	5	3.2	11.7		0.6	2.3		3.9	14.1	
Replication	2	0.2	0.7	0.0	0.1	0.4	0.0	0.0	0.1	0.0
Residual		0.1		10.5	0.2		18.2	0.3		16.5

MS, mean square.

[a] One-gene, two-gene, and three-gene NILs represent the differences among NILs with only one R gene, among those with two R genes, and among those with three R genes. 0 *vs.* 1, 1 *vs.* 2, and 2 *vs.* 3 represent the differences between the recurrent parent and the mean one-gene NILs, between the mean one-gene NILs and mean two-gene NILs, and between the mean two-gene NILs and three-gene NIL, respectively.

DISCUSSION

The resistance of rice plants to *Xoo* measured by leaf-clipping inoculation should refer strictly to inhibition of pathogen growth, which has been known to have both qualitative and quantitative components. Our results indicated that the two components of rice resistance to *Xoo* are associated with the properties of all the R genes studied. The qualitative component of the R genes was reflected by their large effects against the corresponding avirulent *Xoo* races. While these R genes appeared to follow the gene-for-gene theory, as reported previously (Mew 1987), their effects may vary to some extent depending on the pathogen races and the host genetic backgrounds, even in the incompatible cases.

The quantitative component of the R genes (*Xa4*, *xa5*, and *xa13*) appeared to have two elements, the main effects and epistatic effects. In the compatible cases, the main effects of the R genes were reflected as their residual effects against virulent races. Li *et al.* (1999) reported that the breakdown of *Xa4* caused by a Chinese *Xoo* race, CR6, involves loss of its dominance and a 50% reduction in gene effect. In this study, *Xa4* also showed approximately the same residual effect against virulent Philippine races 3 and 4 but a much weaker effect against race 6. Similarly, *Xa21* had a strong residual effect against its virulent Indian race, PXO4. This property appears to be due to the nature of *Xa21*, which has been reported to be a gene family consisting of several member genes (Wang *et al.* 1996). In fact, large residual effects against virulent *Xoo* races of several other dominant R genes including *Xa1*, *Xa7*, and *Xa10* have been reported (Yoshimura *et al.* 1996). In contrast, *xa5* should be considered as a conditional recessive gene because the F_1 plants had lesion lengths between the two homozygotes for all incompatible *Xoo* races except Philippine race 4. It also had significant residual effects against the virulent races, though the magnitude was much smaller than those of *Xa4* and *Xa21*. In this respect, *xa13* was a typical recessive gene with no residual effect against all tested virulent *Xoo* races.

The second quantitative feature of the R genes was their synergistic effects. In our study, we found that a

TABLE 4

Estimated main effects (in $\log_e$-transformed lesion length) of the bacterial blight resistance genes *Xa4*, *Xa21*, *xa5*, and *xa13* against six Philippine and six Indian races of *Xanthomonas oryzae* pv. *Oryzae* races in the rice IR24 (indica) genetic background

	Xa4		*Xa21*		*xa5*		*xa13*	
	Reaction	Effect	Reaction	Effect	Reaction	Effect	Reaction	Effect
Philippine races								
Race 1	*R*	-1.41^{D}	*R*	-1.18^{D}	*R*	-1.22^{A}	*S*	0.01
Race 2	*S*	−0.67	*R*	-1.16^{D}	*R*	-1.15^{A}	*S*	0.05
Race 3	*S*	−0.66	*R*	-1.16^{D}	*R*	-1.22^{PD}	*S*	0.06
Race 4	*S*	-0.69^{D}	*R*	-1.11^{D}	*S*	-0.33^{R}	*S*	0.13
Race 5	*R*	-1.06^{D}	*R*	-1.09^{D}	*R*	-1.21^{A}	*S*	−0.08
Race 6	*S*	−0.20	*R*	-1.19^{D}	*S*	−0.16	*R*	-1.13^{R}
Mean (*R*)		−1.24		-1.15^{D}		−1.20		−1.13
Mean (*S*)		−0.56				−0.25		0.03
Indian races								
PXO1			*R*	−0.88	*R*	−0.68	*S*	0.02
PXO4			MS	−0.47	*S*	−0.33	*S*	−0.13
PXO6			*R*	−0.79	*S*	−0.08	*S*	0.09
PXO8			*R*	−1.29	*S*	−0.25	*R*	−0.86
PXO13			*R*	−1.23	MR	−0.77	*S*	−0.11
PXO17			*R*	−1.69	MR	−0.54	*S*	−0.10
Mean (*R*)				−1.18		−0.66		−0.86
Mean (*S*)				−0.47		−0.22		−0.06

The reaction was based on the standard disease rating system of lesion length (LL): R^{+} = LL < 1 cm; *R* = LL ~1–3 cm; MR = LL ~3.1–6 cm; MS = LL ~6.1–10 cm; *S* = LL > 10 cm (IRRI 1996). (*R*) and (*S*) are the mean values of gene effects for the incompatible (*R*) and compatible (*S*) cases, respectively. The estimated parameters >0.14 are statistically significant (different from zero) based on *t*-tests. The superscripts, A, D, PD, and R indicate additivity (F_1 mean = mid-parental value), complete dominance (F_1 = the resistant parent), partial dominance (resistant parent < F_1 mean < parental mean), and recessiveness (F_1 mean = the susceptible parent) of the resistance genes, on the basis of *t*-tests of lesion length differences between the F_1 plants and the NIL parents. MR, moderately resistant.

high level of resistance against common virulent pathogen races resulted frequently from the defeated gene pairs such as *xa13/xa5*, *xa13/Xa4*, *xa13/Xa21*, *xa5/Xa4*, and *xa5/Xa21*. This has been referred to as "quantitative complementation" (Ogawa and Khush 1988; Yoshimura *et al.* 1996; Huang *et al.* 1997). Our results indicated that this quantitative complementation resulted partially from the residual effects of the defeated dominant R genes and partially from their epistatic effects with the recessive R genes. Specifically, the residual effects of the defeated R genes played a more important role in cases of *xa5/Xa4* and *xa5/Xa21*, and epistasis was much more important in the cases of *xa5/xa13*, *xa13/Xa4*, and *xa13/Xa21*.

Although it has been well known that most plant R genes are dominant, the presence of recessive ones has been recognized in many plant and pathogen relationships. In this respect, the rice-*Xoo* relationship is unique in that 6 out of the 20 R genes are recessive (Ogawa and Khush 1988). Of these, 3 (*xa5*, *xa8*, and *xa13*) occurred naturally and confer race-specific resistance. The other 3 (*xa15*, *xa19*, and *xa20*) were induced by mutagenesis and each confers a wide spectrum of resistance to *Xoo* races (Ogawa 1996). In this study, the two interesting results regarding the behaviors of different R genes were observed. First, there appeared to be a close correspondence between the dominance and residual effects of the R genes. When against virulent *Xoo* races, completely dominant *Xa21* and *Xa4* had fairly large residual effects (~50% of that against avirulent races), the partially dominant *xa5* had a smaller but significant residual effect (~25% of that against avirulent races), and the recessive gene *xa13* had no residual effect and showed more pronounced race specificity. Second, epistasis leading to increased resistance occurred exclusively between *xa5* and *xa13* or between these two genes and a dominant one.

These results suggest some important differences among the R genes. For instance, the fact that the two dominant R genes act independently and additively implies that they might function in different pathways of the rice defensive system. Their larger residual effects against the virulent pathogen races suggest a more complex structure of these dominant R genes. This is consistent with the recent findings that most dominant plant R genes (including *Xa21* and *Xa1*) represent complex

TABLE 5

Estimated epistatic effects (in $\log_e$ transformed lesion length) between or among four bacterial blight resistance genes against six Philippine and six Indian *Xanthomonas oryzae* pv. *Oryzae* races, in the rice IR24 (indica) genetic background

	Philippine *Xoo* races					
R genes	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
Xa4/xa5	0.23	0.30	0.30	(−0.29)	0.26	0.05
Xa4/xa13	0.21	0.31	0.29	(−0.17)	0.24	0.06
Xa4/Xa21	0.41	0.28	0.27	0.26	0.49	0.05
xa5/xa13	−0.04	0.05	0.07	(−0.37)	0.01	0.10
xa5/Xa21	0.41	0.39	0.39	0.11	0.43	0.04
xa13/Xa21	0.07	0.05	0.06	0.05	0.06	0.57
Xa4/xa5/xa13	0.17	−0.07	−0.07	0.37	−0.06	0.52
Xa4/xa5/Xa21	0.13	0.10	0.08	0.28	0.05	0.55
Xa4/xa13/Xa21	0.38	0.43	0.43	0.50	0.26	0.59
xa5/xa13/Xa21	0.46	0.51	0.50	0.60	0.48	0.54
Xa4/xa5/xa13/Xa21	0.23	0.28	0.27	0.30	0.35	0.25
	Indian *Xoo* races					
R genes	PXO1	PXO4	PXO6	PXO8	PXO13	PXO17
xa5/xa13	R^a −0.09	*R* (−0.33)	MR (−0.30)	*R* 0.08	MR 0.11	*R* −0.09
xa5/Xa21	R^+ 0.03	*R* (−0.68)	R^+ (−0.21)	R^+ (−0.17)	R^+ 0.08	R^+ 0.32
xa13/Xa21	*R* (−0.14)	*R* (−0.38)	R^+ (−0.57)	R^+ 0.25	R^+ (−0.46)	R^+ −0.09
xa5/xa13/Xa21	R^+ −0.07	R^+ (−0.27)	R^+ (−0.46)	R^+ 0.02	R^+ (−0.21)	R^+ (−0.11)

The numbers in parentheses are highly significant epistatic effects between R genes that resulted in increased resistance (large negative values).

[a] The estimated parameters >0.14 are statistically significant (different from zero), based on *t*-tests.

gene families, each consisting of several member genes of potentially different functions (Song *et al.* 1995; Wang *et al.* 1996; Yu *et al.* 1996; Yoshimura *et al.* 1998). For instance, member D (*Xa21D*) of the *Xa21* gene family is known to confer partial resistance to *Xoo* (Wang *et al.* 1996), which provides a perfect explanation for the residual effect of *Xa21* against the virulent Indian race POX4 observed in this study. In contrast, the more

TABLE 6

Estimated epistatic effects (in $\log_e$-transformed lesion length) between or among bacterial blight resistance genes against six Philippine *Xanthomonas oryzae* pv. *Oryzae* races in the rice IR24 (indica), NPT2, and NPT3 (japonica) genetic backgrounds

Genetic backgrounds	R gene combination	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
IR24	*xa5/xa13*	−0.04	0.05	0.07	(−0.37)	0.01	0.10
	xa5/Xa21	0.41	0.39	0.39	0.11	0.43	0.04
	xa13/Xa21	0.07	0.05	0.06	0.05	0.06	0.57
	xa5/xa13/Xa21	0.46	0.51	0.50	0.60	0.48	0.54
NPT2	*xa5/xa13*	−0.01	0.15	0.11	(−0.32)	0.10	0.08
	xa5/Xa21	0.38	0.39	0.26	0.08	0.14	(−0.35)
	xa13/Xa21	(−0.10)	0.07	(−0.09)	(−0.34)	(−0.15)	0.20
	xa5/xa13/Xa21	0.41	0.19	0.35	0.86	0.36	0.48
NPT3	*xa5/xa13*	(−0.12)	0.15	(−0.15)	(−0.10)	0.04	0.34
	xa5/Xa21	0.15	0.58	0.09	0.06	0.00	(−0.34)
	xa13/Xa21	0.08	−0.02	0.04	(−0.33)	0.10	0.32
	xa5/xa13/Xa21	0.43	0.32	0.46	0.67	0.44	0.30

The estimated parameters >0.06 are statistically significant (different from zero), based on *t*-tests. The numbers in parentheses are highly significant epistatic effects between R genes that resulted in increased resistance (large negative values).

TABLE 7

Estimated main effects (in $\log_e$-transformed lesion length) of bacterial blight resistance genes against six Philippine *Xanthomonas oryzae* pv. *Oryzae* races in the rice IR24 (indica), NPT2, and NPT3 (japonica) genetic backgrounds

Genetic background	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6	Mean (*R*)	Mean (*S*)
	S	*S*	*S*	*S*	*S*	*S*		
IR24	2.09 bc	2.24 a	2.21 ab	2.26 a	2.01 c	2.31 a		2.19 a
NPT2	2.32 c	2.42 c	2.38 c	2.60 b	2.84 a	2.74 a		2.55 b
NPT3	1.87 d	1.99 c	2.03 c	2.22 b	2.52 a	2.48 a		2.19 a
Mean	2.09 d	2.22 c	2.21 c	2.36 b	2.46 a	2.51 a		
	R	*R*	*R*	*R*	*R*	*R*		
Xa21 (IR24)	−1.19	−1.21	−1.11	−1.23	−0.52	−0.76	−1.00	
Xa21 (NPT2)	−1.00	−0.84	−0.80	−0.94	−0.65	−0.64	−0.81	
Xa21 (NPT3)	−0.73	−0.95	−0.82	−0.86	−0.67	−0.48	−0.75	
	R	*R*	*R*	*S*	*R*	*S*		
xa5 (IR24)	−1.61	−1.18	−1.08	−0.63	−0.93	−0.24	−1.20	−0.44
xa5 (NPT2)	−0.77	−0.92	−0.64	−0.56	−0.80	0.08	−0.78	−0.24
xa5 (NPT3)	−0.54	−0.98	−0.37	−0.63	−0.48	0.01	−0.59	−0.31
	S	*S*	*S*	*S*	*S*	*R*		
xa13 (IR24)	0.14	0.07	0.12	0.14	−0.01	−1.03	−1.03	0.09
xa13 (NPT2)	0.18	0.19	0.02	0.04	−0.14	−1.04	−1.04	0.06
xa13 (NPT3)	0.08	0.09	0.15	0.10	−0.36	−1.18	−1.18	0.01

In the first four rows, different letters within a row indicate statistically significant differences in aggressiveness between *Xoo* races while different letters in the mean indicate the significant differences in quantitative resistance between the susceptible parents. The estimated parameters >0.06 are statistically significant (different from zero), based on *t*-tests.

pronounced epistatic role and race specificity of *xa13* may suggest a relatively simple structure and possible regulatory role of this recessive R gene in the more upstream of the rice defensive system. Thus, molecular cloning of *xa5* and *xa13* would greatly enhance our understanding of the defensive system of rice plants.

Our results reveal several genetic aspects of the rice-*Xoo* interactions. First, the reaction of a plant to a race of its pathogen reflects the outcome of the interactions between alleles at all avirulence loci in the pathogen and alleles at all R loci of the plant, even if there is a one-to-one relationship between each of the interacting R-avirulence gene pairs. Also, the reaction is determined by the rate in which the plant defensive responses are triggered through interactions between alleles at plant R loci and alleles at the corresponding avirulence loci in the pathogen. The faster the response rate is, the more likely a resistant phenotype is to arise. Thus, it was not surprising that more pronounced genotype (rice) × race (*Xoo*) interactions were observed at digenic or trigenic levels than that at the monogenic level in the present study. Second, the observation that all dominant R genes had residual effects against their corresponding virulent races indicated that suppression of individual R genes by their corresponding virulent genes in the pathogen may or may not be complete, at least in the cases of rice-*Xoo* interaction. In other words, the quantitative resistance, which resulted partially from the residual effects of major R genes and partially from epistasis between defeated R genes, is not strictly nonrace specific. Finally, the significant effects of the NIL genetic backgrounds on the estimated main and epistatic effects of the R genes could be attributed to the differences between the IR24 and NPT NILs. IR24 is highly susceptible to all *Xoo* races, and all single R-gene NILs were developed by more than nine generations of backcrossing, while the NILs with R-gene pyramids were developed by crossing the single R-gene NILs and marker-aided selection (Huang *et al.* 1997). Thus, the IR24 NILs are expected to be largely identical except for the small genomic regions of the R genes. On the other hand, the two sets of NPT NILs are actually BC_3F_3 lines developed by crossing the NPT parents and IRBB59 (the IR24 NIL with all four R genes), by marker-assisted selection and selection for the phenotype of the recurrent parent (Sanchez *et al.* 2000). Thus, according to Young and Tanksley (1989), large introgressed segments in the target R gene regions and random nontarget IR24 genomic segments are expected to be present in the NPT NILs, which might have contributed to part of the genetic background effects on the estimated main and epistatic effects of the R genes. In application, our results suggest that the choice of individual R genes and their combinations for high-level and durable resistance

to *Xoo* should vary depending on the host genetic backgrounds (indica or japonica) and on specific breeding programs for inbred or hybrid cultivars.

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