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A "defeated" rice resistance gene acts as a QTL against a virulent strain of *Xanthomonas oryzae* pv. *oryzae*

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Abstract The genetic components responsible for qualitative and quantitative resistance of rice plants to three strains (CR4, CXO8, and CR6) of *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) were investigated using a set of 315 recombinant inbred lines (RILs) from the cross Lemont (*japonica*) × Teqing (*indica*) and a complete linkage map with 182 well distributed RFLP markers. We mapped a major gene (*Xa4*) and ten quantitative trait loci (QTLs) which were largely responsible for segregation of the resistance phenotype in the RILs. The Teqing allele at the *Xa4* locus, $Xa4^T$, acted as a dominant resistance gene against CR4 and CXO8. The breakdown of $Xa4^T$-associated resistance mediated by the mutant allele at the *avrXa4* locus in the virulent strain CR6 results from significant changes in both gene action (lose of dominance) and the magnitude of gene effect (≈50% reduction). Nevertheless, $Xa4^T$ still acted as a recessive QTL with a significant residual effect against CR6. The mutant alleles at the *avrXa4* locus in CXO8 and CR6 that lead to a reduction in effect, or "breakdown", of $Xa4^T$ were apparently accompanied by corresponding penalties for their fitness. The quantitative component of resistance to *Xoo* in the RILs was largely due to a number of resistance QTLs. Most resistance QTLs mapped to genomic locations where major resistance genes and/or QTLs for resistance to *Xoo*, blast and sheath blight were identified in the same cross. Most QTLs showed consistent levels of resistance against all three *Xoo* strains. Our results suggest that a high level of durable resistance to *Xoo* may be achieved by the cumulative effects of multiple QTLs, including the residual effects of "defeated" major resistance genes.

Key words *Oryza sativa* L. · Gene mapping · Quantitative trait loci (QTLs) · Host-pathogen co-evolution · Stabilizing selection

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Introduction

The presence of two major types of disease resistance to plant pathogens – vertical resistance and horizontal resistance – has long been recognized in interactions between plant hosts and their pathogens (Van der Plank 1968; Nelson 1972; Simmonds 1979). Vertical resistance in many plant host-pathogen relationships is hypersensitive, race-specific, and governed by interactions between avirulence genes in pathogens and resistance genes in plant hosts (Van der Plank 1968). In contrast, horizontal resistance is quantitative, presumably non-race specific, and controlled by polygenes (Van der Plank 1968; Nelson 1972), though these assumptions have not been actively tested. In the genetics of host-pathogen interactions, a long-standing controversial issue is the nature of the genetic basis of "stabilizing selection" which largely determines the co-evolution of many plant host-pathogen relationships (Van der Plank 1963, 1968; Leonard and Czochor 1980). This theory states that new pathogenic race(s) will suffer a loss in general fitness when they acquire new virulence genes by mutation. Genetically, this implies that conversion of

avirulence genes into corresponding virulence genes by mutation is expected to result in lower fitness of the pathogen. Unfortunately, direct evidence to support this theory has been difficult to obtain.

Rice (*Oryza sativa* L.) bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) is a devastating disease in Asia. Two types of resistance to *Xoo*, vertical (VR, complete whole-life resistance) and horizontal (HR, quantitative resistance), have been recognized in rice (Zhang and Mew 1985). VR to *Xoo* is controlled by at least 20 major genes that are usually race specific (Zhang 1991; Causse et al. 1994; G. Zhang et al. 1996; Q. Zhang et al. 1996; Lin et al. 1996). However, resistance of a segregating rice population to specific *Xoo* strains often shows both qualitative and quantitative components (Koch and Parlevliet 1991). These characteristics of the relationship between *O. sativa* and *Xoo* offer a unique opportunity to study the genetics of the interaction between host plants and their pathogens.

We report here that a "defeated" dominant resistance gene acts as a quantitative trait locus (QTL) against a virulent strain of *Xoo*, and quantify the interaction between mapped resistance QTLs and different *Xoo* strains in rice.

Materials and methods

Plant materials

The materials used in the study included two rice cultivars, Lemont (a commercial rice cultivar from the Southern US, ssp. *japonica*) and Teqing (a Chinese commercial cultivar, *ssp. indica*), their F_1 progeny, and a set of 315 F_{10} recombinant inbred lines (RILs), each derived (by single seed descent) from a cross between Lemont and Teqing.

Inoculation of plants and evaluation of disease symptoms

In the summer of 1995, the RILs, the parental lines, and F_1s were seeded in the nursery, and 25-day-old seedlings were transplanted into the field at the Chinese National Rice Research Institute in Hangzhou. Each of the RILs, parents, and F_1 plants was planted in single 7-row plots with 6 plants in each row. Three *Xoo* strains – CR4, CR6, and CXO8 – were selected for the study. CR4 and CR6 belong to *Xoo* races 4 and 6 and were collected in Southern China, while CXO8 is a local *Xoo* strain collected from the fields of the Chinese National Rice Research Institute. The three strains were cultured on a standard semisynthetic potato-agar medium for 72 h and then prepared as a suspension (in sterile water) of approximately 3×10^8 bacteria/ml (Ou 1972). Three newly expanded leaves on different tillers of five plants in each plot were simultaneously inoculated with the three *Xoo* strains 60 days after transplanting, using the standard leaf clipping method (Kauffman et al. 1973). Lesion length (LL) on each inoculated leaf was measured 21 days after inoculation.

Genotyping of the RILs

The RILs were genotyped using 179 well-distributed RFLP markers following standard procedures (Li et al. 1995), as described previously (Tabien et al. 1998). Three morphological markers, *gl-1* (glabrous leaf), *C* (purple apiculus), and *Ph* (reaction to phenol), were also included. These markers were used to construct a complete linkage map covering all 12 rice chromosomes (Tabien et al. 1998).

Data analysis

Log-transformed data of LL were used to identify resistance genes and QTLs using both composite interval mapping and multiple regression models (Zeng 1994; Li et al. 1997). This was carried out in two steps. First, each putative QTL was identified using stepwise regression-based single-marker genotypes with a threshold of $P \leq 0.002$. Then, all putative QTLs identified in the first step were re-evaluated using composite interval mapping, with all identified QTLs in the first step fixed in the model to control for background genetic effects, as described (Li et al. 1997). In this method, all intervals (QTLs) are sequentially fitted starting with the interval (QTL) having the largest LOD value. Marker intervals (QTLs) with the next largest LOD were added to the model if the interval (QTL) increased the overall LOD of the model by 2.0 or greater.

Results

Parental difference and segregation of LL in the Lemont/Teqing RILs

Teqing was resistant (LL = 3.5 and 2.1 cm, respectively) to CR4 and CXO8, but susceptible (LL = 14.7 cm) to CR6. Lemont was susceptible (LL = 22.9, 14.1 and 10.1 cm, respectively) to all three strains. On infection with CR4 and CXO8, the F_1 plants had a mean LL of 5.9 and 5.6 cm, respectively, indicating that resistance was largely dominant. The mean LL on the F_1 plants caused by CR6 was 16.5 cm, significantly longer than that of Lemont or Teqing. LL on the RILs caused by CR4 and CXO8 showed a bimodal distribution (Fig. 1), suggesting involvement of major resistance gene(s). On infection with CR6, LL of the RILs exhibited continuous variation, and transgressive segregation was present in both directions, showing typical polygenic inheritance. A significant portion (30%) of the RILs had shorter LLs than either parent, and 27 RILs had LL ≤ 3 cm. The resistance of the RILs to the three *Xoo* strains was highly correlated (r = 0.90 between CR4 and CXO8, 0.83 between CR4 and CR6, and 0.88 between CXO8 and CR6).

Mapping of a major resistance gene

Xa4, a major gene conferring resistance to all three *Xoo* strains was mapped between RZ536 and G2132b on chromosome 11 (Table 1 and Fig. 2) with extremely high LOD values (LOD = 61.1 for CR4, 43.1 for CXO8, and 36.1 for CR6). This gene was inferred to be *Xa4*, based on its map position (Causse et al. 1994) and previous studies on the resistance of several lines related to Teqing (Lin and Ming 1990; Wu et al. 1991). This gene explained 65.2%, 55.2%, and 52.1% of the total variation in LL caused by CR4, CXO8, and CR6. The Teqing allele at this locus had an additive effect for reducing LL caused by CR4, CXO8, and CR6 by 9.0 cm,

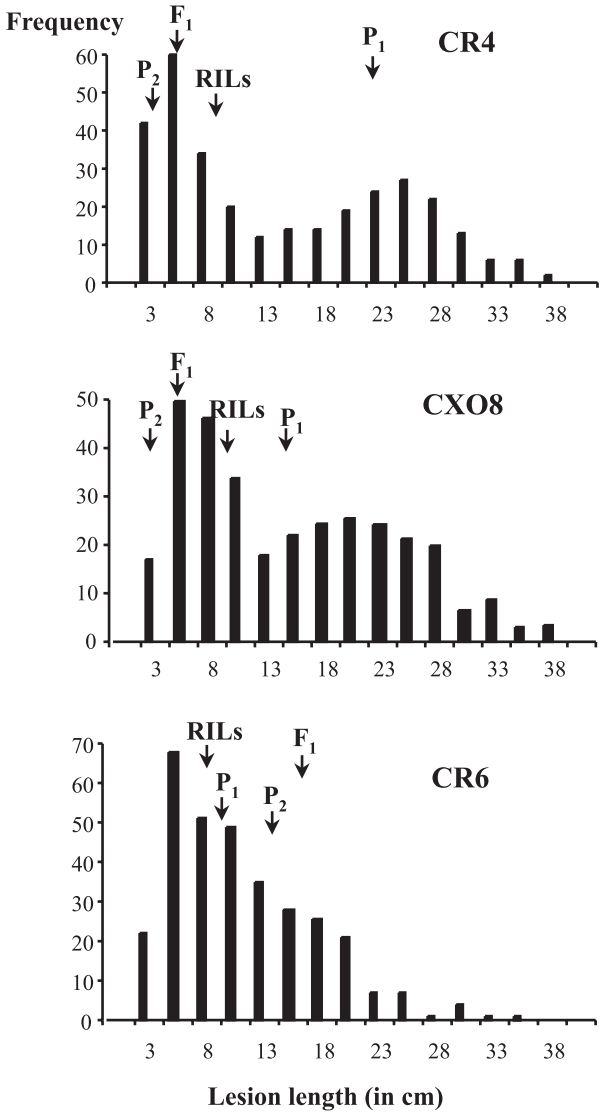

Fig. 1 Histograms of lesion length on the 315 Lemont/Teqing RILs caused by infection with each of the three *Xoo* strains CR4, CXO8, and CR6. The *arrows* indicated the mean lesion lengths for the parental lines (P1, Lemont; P2, Teqing) and the F1 plants

7.0 cm and 4.8 cm , respectively (Table 1). The Teqing allele(s) present at the *Xa4* locus, $Xa4^T$, thus acted as a dominant resistance gene against CR4 and CXO8 but as a recessive factor against CR6.

Mapping of resistance QTLs

Since the large effect of *Xa4* is expected to have an impact on the detection of other QTLs with smaller effects (cf. Li et al. 1997), composite interval mapping was conducted using the full data set with *Xa4* fixed in the model to control its effect. Furthermore, the RILs were grouped into two approximately equal subpopulations based on their genotypes at *Xa4* (inferred from the phenotypes caused by CR4 and CXO8 and two flanking markers). The identified putative QTLs detected using the full data set were confirmed using the two subsets of the data from susceptible (*xa4*/*xa4*) and resistant (*Xa4*/*Xa4*) subpopulations.

Ten putative QTLs were identified using both the whole data set and two data subsets (Table 1). These QTLs fell on eight of the 12 rice chromosomes (Fig. 2) and collectively explained more than 65% of the residual variation in LL unexplained by *Xa4*. The resistance alleles at seven of the QTLs were from Teqing, and three were from Lemont.

Race specificity of resistance QTLs

The identified QTLs showed some degree of race specificity. For two of the ten detected QTLs, phenotypic effects were significant against only one or two of the three *Xoo* strains (Table 1). The remaining eight QTLs showed consistent resistance to the three *Xoo* strains, though their effects varied in magnitude. To quantify the interactions between the ten identified QTLs and the three *Xoo* strains, we performed two-way ANOVA on the gene effects of the ten QTLs against the three *Xoo* strains. The result indicated that 64.0% of the total variation in QTL effect was due to variation among QTLs, 11.5% to variation among the *Xoo* strains, and 24.5% to the QTL × strain interaction.

Differences in the fitness of the *Xoo* strains and their associations with the effectiveness of *Xa4* and resistance QTLs

The differences between the mean LL caused by the three *Xoo* strains on the RILs were highly significant, particularly on the susceptible RILs which did not carry $Xa4^T$ (Table 2). CR6, which was virulent to both parents, caused the shortest LL (15.1 cm) on the susceptible RILs, 37.7% and 53.6% shorter, respectively, than those caused by CXO8 (20.8 cm) and CR4 (23.2 cm). There was highly significant correlation ($r = 0.99$, $P < 0.05$) between the gene effects of *Xa4* against the three *Xoo* strains and the mean fitness (LL) of the susceptible RILs caused by the *Xoo* strains. The correlation between the cumulative effect of the ten detected QTLs against the three *Xoo* strains and their fitness was not significant ($r = -0.38$, $P > 0.50$). Because the cumulative effects of the detected QTLs may serve roughly as the genetic background effects of the RILs on the fitness of the *Xoo* strains, this result implies that the differences in general fitness among the *Xoo* strains are largely due to interactions between the two alleles at the *Xa4* locus in the RILs and the three alleles at the corresponding avirulence gene locus (*avrXa4*) in the *Xoo* strains.

Discussion

Although a high level of resistance (hypersensitivity) to *Xoo* in rice is race specific and controlled by single major

Table 1. Additive gene effects (*a*) of Teqing alleles at *Xa4* and ten QTLs on lesion length (in cm) caused by infection with three *Xoo* strains (CR4, CXO8, and CR6), detected in the Lemont × Teqing RILs

Marker interval[a]	Chromosome	CR4		CXO8		CR6	
		a	*P*	*a*	*P*	*a*	*P*
RG520/RZ476a[b]	*2*	–	–	–	–	*−0.8*	*0.0359*
C515/RG348	*3*	2.2	0.0009	2.1	0.0010	1.6	0.0035
RG482/CDO795	*3*	−0.8	0.0006	−1.6	0.0010	−1.2	0.0020
RG214/*Ph*	*4*	−0.9	0.0338	−1.8	0.0074	−1.4	0.0012
RZ69/RG190	4	1.9	0.0060	1.8	0.0005	2.1	0.0008
G104/G1314a	8	−1.6	0.0022	−2.7	0.0004	−2.0	0.0001
RG451/RZ404	*9*	−0.7	0.0013	−2.4	0.0008	−1.8	0.0005
RG1094f/C16	*10*	1.8	0.0105	2.2	0.0180	2.2	0.0001
Xa4 (RZ536/G2132b)	*11*	−9.0	< 0.0001	−7.0	< 0.0001	−4.8	< 0.0001
RG1022/RZ525	*11*	−2.8	< 0.0001	−2.6	< 0.0001	−1.7	0.0004
RG91Q/RG341[b]	*12*	–	–	−1.7	0.0004	−1.5	0.0007

[a] *Underlined* markers are those in the multiple regression model with all significant QTLs included, and *P* is the probability, expressed as partial sums of squares, associated with individual QTLs

[b] *Dashes* indicate no significant marker effects associated with corresponding pathogen strains

genes, the reactions of a segregating rice population to different *Xoo* strains virtually always show both qualitative and quantitative components. Both qualitative and quantitative genetic determinants contribute to race specificity. The ''division'' between ''major genes'' and ''QTLs'' for resistance is obscured further by the fact that ''defeated'' major genes can act as QTLs conferring horizontal resistance against virulent pathogen races. Furthermore, our data indicate that stabilizing selection may be acting on the rice-*Xoo* relationship. Finally, we propose that a high level of durable resistance to *Xoo* (and perhaps other diseases) may be achieved by cumulative effects of multiple QTLs, including the residual effects of ''defeated'' major genes.

Fig. 2 Chromosomal locations of *Xa4* (*filled oval*, chromosome *11*) and ten QTLs (*filled squares*) conferring horizontal resistance to three *Xoo* strains (CR4, CXO8, and CR6) identified in the Lemont × Teqing RILs. Mapped major resistance genes (*Xa* for resistance to *Xoo*, *Pi* for resistance to blast) and QTLs (*QSbr* for resistance to sheath blight) detected in the same cross or reported in different crosses (Li et al. 1995; Tabien et al. 1998; Causse et al. 1994) are indicated in *bold italics*

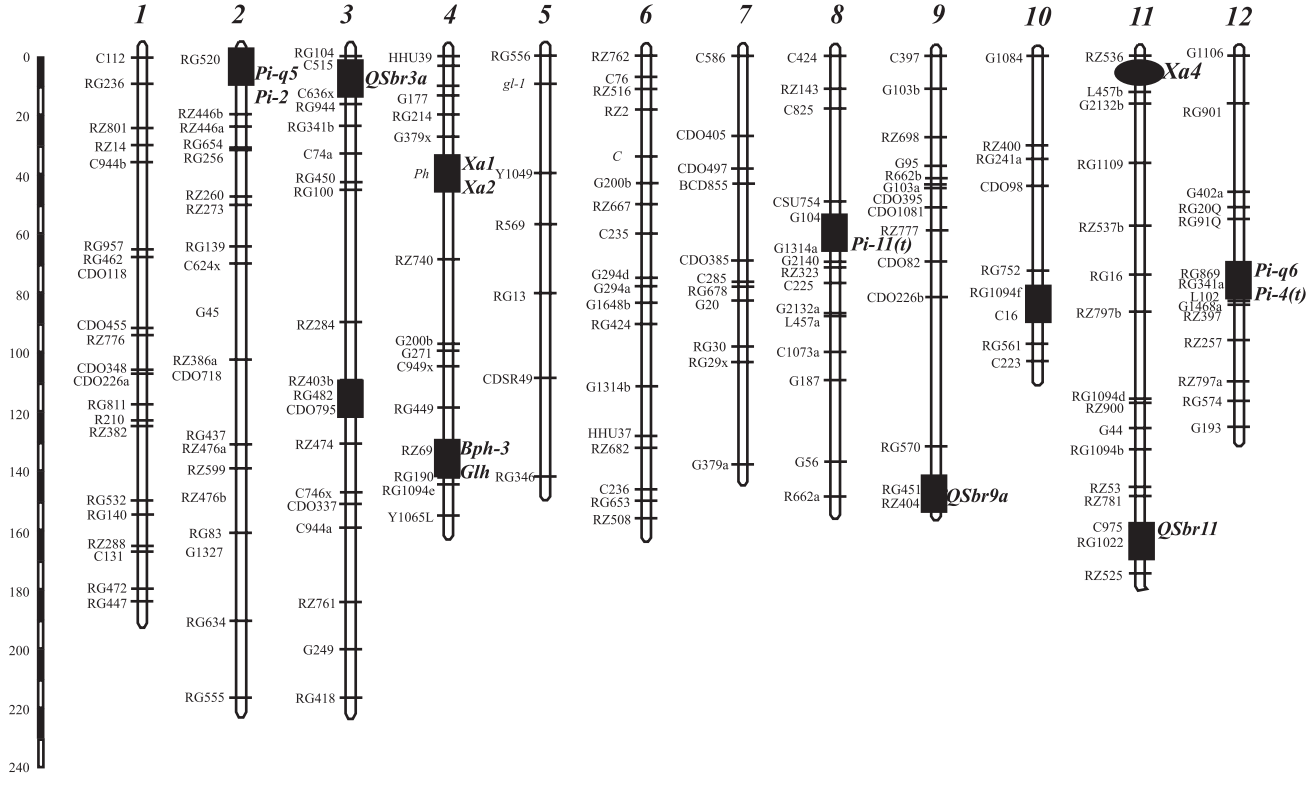

Table 2 Comparison of the average lesion length (in cm) caused by three *Xoo* strains on the resistant RILs (*Xa4*/*Xa4*) and the susceptible RILs (*xa4*/*xa4*)

Xoo strains	Inferred genotype at *Xa4* locus[a]	
	xa4/*xa4* (susceptible)	*Xa4*/*Xa4* (resistant)
CR4	23.2a	5.2a
CXO8	20.8b	7.0b
CR6	15.1c	5.7a

[a] The differences in mean lesion length between the *Xoo* strains indicated by different letters in the same column are statistically significant at $P = < 0.05$

Relationships between host resistance genes/QTLs and *Xoo* strains

The race specificity of rice resistance to different *Xoo* strains has both qualitative and quantitative components. *Xa4* is the most widely used resistance gene in many Asian rice breeding programs and conferred durable resistance in many commercial rice cultivars for more than a decade before being overcome by new virulent *Xoo* strains (Mew et al. 1992). Our results indicate that the breakdown of $Xa4^T$-mediated resistance by the mutation at the *avrXa4* locus in CR6 has both qualitative (lose of its dominance for resistance) and quantitative (an approximately 47% reduction in gene effect) components. As a major dominant gene, $Xa4^T$ has a reduced effect (by 22%) against CXO8 without losing its dominance as compared to CR4. In other words, mutations at the *avrXa4* locus in *Xoo* may result in both qualitative and quantitative changes in resistance phenotype when they interact with alleles at resistance loci in the host plants. This contrasts with the extreme cases of race specificity seen in many other plant host-pathogen relationships including rice blast, where race specificity is associated with the presence or absence of hypersensitivity.

While consistent with previously reported results (Koch and Parlevliet 1991), DNA markers further allow us to distinguish the residual effect of $Xa4^T$ against virulent *Xoo* strains from minor effects of other resistance QTLs. The fact that $Xa4^T$ acts as a QTL with a significant residual effect against the virulent strain CR6 explains, at least in part, its durability. Most resistance QTLs identified in this study do show some degree of strain specificity, but this specificity accounts for only a small portion (24.5%) of the total variation in resistance. Because the number of identified resistance QTLs is fairly large and none of the three *Xoo* strains interacts consistently with all these QTLs, it is expected that cumulative effects of multiple resistance QTLs may result in a significant level of durable quantitative resistance to *Xoo*.

The relationship between major resistance genes and QTLs

Our finding that $Xa4^T$ at the *Xa4* locus acts as a major gene against CR4 and CXO8, but as a recessive QTL against CR6, suggests that major genes and QTLs for resistance may be the same genes. We further note that one QTL has been mapped in very close proximity to *Xa1* (or *Xa2*) on chromosome *4* (Causse et al. 1994), suggesting that this QTL may be due to allelic difference at this locus. Furthermore, three additional QTLs were mapped near the major blast resistance genes *Pi-2* (*Pi-9* and *Pi-z*), *Pi-11* (t), and *Pi-q6* (*Pi-4* and *6*) on chromosomes 2 and 3 identified in the same RILs or reported previously (Tabien et al. 1998; Causse et al. 1994) (Fig. 2). Four QTLs (on chromosomes *3, 9*, *11* and *12*) were mapped to similar genomic positions to resistance QTLs against sheath blight (*R. solani*) and blast in the same cross (Li et al. 1995; Tabien et al. 1998). In all these cases, resistance alleles in the same genomic regions were from Teqing. QTLs for partial resistance to blast are also detected in the vicinity of major genes conferring hypersensitivity in this RIL population (Tabien 1998) and another rice RIL population (Wang et al. 1994). Such close correspondences in both genomic location and gene action between genes/QTLs conferring resistance to different pathogens is unlikely to be due to chance (Fig. 2). Thus, all these results appear to lend support to the postulate that "defeated" major resistance genes may have residual effects against different races of the same pathogens, or different pathogens (*cf*. Martin and Ellingboe 1976; Royer et al. 1984). Recent studies have revealed that disease resistance loci in tomato, rice and soybean tend to exist in clustered multigene families (Thilmony et al 1995; Song et al. 1996; White et al. 1995; Yu et al. 1996; Kanazin et al. 1996). Thus, an alternative explanation is that major resistance genes or QTLs at similar genomic locations may reflect functional differences in member genes within clusters of resistance gene families. Some QTLs not associated with known major resistance genes may represent loci of unidentified resistance gene families or their regulatory loci. Detailed molecular characterization of these resistance gene families will certainly shed light on this important issue.

Evidence for "stabilizing selection"

The co-evolution of many plant host-pathogen relationships may be governed by stabilizing selection (Van der Plank 1963, 1968; Leonard and Czochor 1980); these authors state that new pathogenic race(s) will suffer a loss in general fitness when they acquire new virulence genes by mutation. As a result, pathogenic strains with more virulence genes tend to have lower fitness. However, it has been difficult to obtain hard evidence to prove or disprove this theory (Leonard and Czochor 1980). We have found that the effectiveness of $Xa4^T$ against the three *Xoo* strains is highly correlated with the general fitness of these strains. This suggests that the genetic changes at the *avrXa4* locus in CXO8 and CR6 leading to a reduced effect or "breakdown" of $Xa4^T$ are

accompanied by corresponding penalties to their fitness. This, together with the similar results for the avirulence genes *avrXa7* and *avrXa10* in *Xoo* reported by White et al. (1995), appears to lend support to Van der Plank's theory and suggest that "stabilizing selection" may be operating on the rice-*Xoo* relationship.

Implications for breeding for disease resistance

Our observation that many RILs that are highly resistant to the virulent strain CR6 can be recovered from the cross between two susceptible parents shows that a high level of durable resistance may be achieved by the cumulative effect of multiple QTLs plus the residual effects of "defeated" major genes. In practice, this means that a breeding program for resistance to *Xoo* does not necessarily have to involve parents with a high level (hypersensitivity) of resistance to *Xoo*, as long as the resistance genes/QTLs in the parental lines are complementary to one another.

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