Genetics of Bacterial Blight Resistance in a Breeding Line of Rice

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ABSTRACT

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The genetics of resistance in rice breeding line IR1545-339 to strains of four races of *Xanthomonas campestris* pv. *oryzae* in the Philippines was studied. The analysis of F_1 and F_2 populations from the cross of IR1545-339 with susceptible cultivar TN1 demonstrated that recessive gene *xa*-5 conferred the high resistance of IR1545-339 to races 1, 2, and 3. The moderate resistance of IR1545-339 to a strain of race 4, which is characterized by slow lesion development, also appeared to be governed by *xa*-5. Trisomic analysis revealed that *xa*-5 is located on chromosome 2 as designated by Nishimura.

Additional key words: bacterial races.

The strains of *Xanthomonas campestris* pv. *oryzae* (Ishiyama) Dye in the Philippines are classified into four race groups based on the specific interactions among rice cultivars and the bacteria (6). Five rice cultivars (IR8, IR20, Cas 209, IR1545-339, and DV85) (7) are used as differentials: IR8 is susceptible to all Philippine races; IR20 is resistant only to race 1; Cas 209 is resistant only to race 2; IR1545-339 is resistant to races 1, 2, and 3; and DV85 is resistant to all four races. The inheritance of resistance of these differential cultivars to race 1 has been investigated for several years. IR20 has gene *Xa*-4 (11) and Cas 209 has gene *Xa*-10 (18). The resistance of IR1545-339 to race 1 is controlled by gene *xa*-5 (10). Two genes, *xa*-5 and *Xa*-7, were identified in DV85 when it was analyzed with a strain of race 1 (15).

This study was undertaken to determine the inheritance of resistance of IR1545-339 to races 2 and 3 and its moderate resistance to race 4 of the bacteria and to find the chromosomal location of gene *xa*-5 by trisomic analysis.

MATERIALS AND METHODS

IR1545-339 (hereafter referred to as IR1545) is a breeding line developed at the International Rice Research Institute (IRRI) from the cross of IR24 and DZ192. The latter parent was the donor of *xa*-5. IR1545 was crossed with Taichung Native 1 (TN1) and IR20. TN1 is susceptible to all races, whereas IR20 is resistant to race 1. F_1 and F_2 progenies from the crosses of TN1/IR1545 and IR20/IR1545 were tested for bacterial blight resistance by using races 1, 2, and 3. F_1, F_2, and F_3 progenies of TN1/IR1545 were also tested for disease reaction to race 4. Two sets of seeds of 65 F_3 families from this cross were planted separately and one set was tested for reactions to PX086 and the other for reactions to PX071. Seventeen to 20 plants of each family were grown. The other planting of the same F_3 families were inoculated with PX071.

The bacterial strains PX061 of race 1, PX086 of race 2, PX079 of race 3, and PX071 of race 4 were used for inoculating the segregating populations from the crosses of IR1545 with TN1 and IR20. Hereafter, these strains will be referred to by their race number. The inoculum of each strain was prepared by culturing the bacteria on potato semisynthetic agar medium (17) in slants and incubating at 30 C for 3 days. Inoculum was adjusted to a concentration of about 10^9 cells per milliliter.

For the inheritance study, hybrid populations and the parents were grown in the screenhouse or in the greenhouse under standard management. In the greenhouse experiments, 14-day-old seedlings raised in the seed boxes were transplanted to 20-cm-diameter clay pots at two seedlings per pot. The soil was fertilized with N-P-K at 90-60-60 kg/ha before transplanting and additional nitrogen at the rate of 30 kg N/ha was applied 1 wk before inoculation. In the screenhouse experiments, 14-day-old seedlings were transplanted with one seedling per hill at a spacing of 30 × 20 cm. The soil was fertilized with N-P-K at 60-30-30 kg/ha as basal application. Additional 30 kg N/ha was applied 1 wk before inoculation. Tillers of each plant were divided equally according to the number of bacterial strains used for inoculation. The plants were inoculated at maximum tillering stage (~50–55 days after seeding). Inoculation of fully expanded leaves was by the leaf clipping method (5). Three or more leaves of each plant of the parental, F_1, F_2, and F_3 plants were inoculated with each bacterial strain. Disease scores were taken 14 days after inoculation. Two inoculated leaves of each plant of the parental, F_1, F_2, and F_3 populations were taken

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randomly for lesion length measurement. The mean lesion length for each race was used to determine the reaction of each plant to each race.

For trisomic analysis, at least five leaves of each plant of each F_2 population were inoculated with bacterial strain PX086 at 60–70 days after seeding. Each plant was scored according to the Standard Evaluation Systems for Rice (SES) based on percent lesion area over leaf area, and classified as either resistant or susceptible.

RESULTS

Inheritance of resistance to PX061, PX086, and PX079. The disease scores of parental and F_1 plants and frequency distribution of lesion lengths of parental and F_2 populations from the cross TN1/IR1545 to three bacterial strains are shown in Fig. 1. The disease scores of F_1 plants to races 1, 2, and 3 were rated S (susceptible), S, and S, respectively, similar to those of TN1. The disease score to race 1 of the F_2 plants showed a wide range of variation and formed a clear-cut bimodal distribution. Similarly, two distinct modes for frequency distribution of disease scores to PX086 and PX079 were observed. The classification of the F_2 population into resistant and susceptible groups for disease rating to three strains was clear. Of 65 F_3 families derived from TN1/IR1545, 17 were homozygous resistant, 33 were segregating, and 15 were susceptible when inoculated with PX086. These data agree with the 1:2:1 ratio expected for single-gene control of resistance governed by *xa*-5.

The F_1 plants of cross IR20/IR1545 were scored as R (resistant), S, and S to races 1, 2, and 3, respectively. The F_1, when inoculated with races 2 and 3, was even more susceptible than the susceptible IR20. Two modes were observed in the frequency distribution of disease scores of the F_2 population when inoculated with race 1. Two groups could be distinguished with a dividing line at the 18-cm lesion length. Two distinct modes in the frequency distribution of the disease scores to races 2 and 3 were also observed and the classification into resistant and susceptible groups was clear (Fig. 2).

The data showing the disease scores of F_1 and F_2 populations from the crosses TN1/IR1545 and IR20/IR1545 are summarized in Table 1. F_1 plants of TN1/IR1545 showed S reactions to races 1, 2, and 3, suggesting that the resistance of IR1545 to the three strains is under recessive gene control. The observed F_2 segregation of 34 RRR and 90 SSS plants gave a good fit to the 1:3 ratio expected for single recessive gene control of resistance.

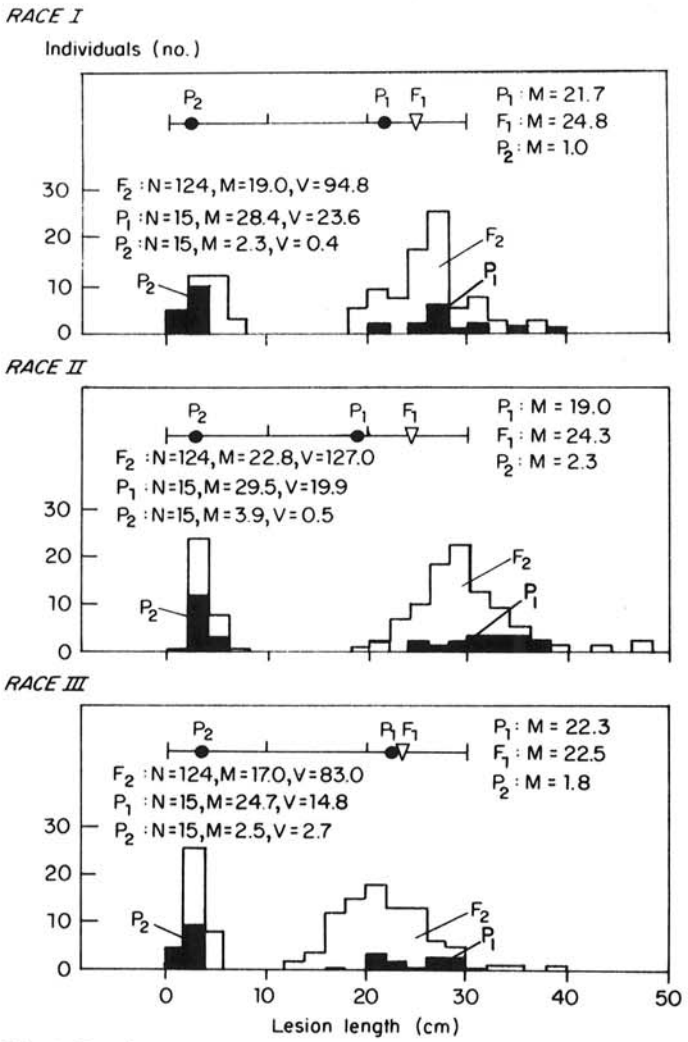

Fig. 1. Reactions to *Xanthomonas campestris* strains PX061 (Race I), PX086 (Race II), and PX071 (Race III) of parental and F_1 plants and frequency distributions of disease scores of parental and F_2 populations from the cross of rice cultivars Taichung Native 1 (P_1)/IR1545 (P_2). M = mean lesion length (cm), N = number of individuals, V = variance. Black area = parent, white area = F_2 plants.

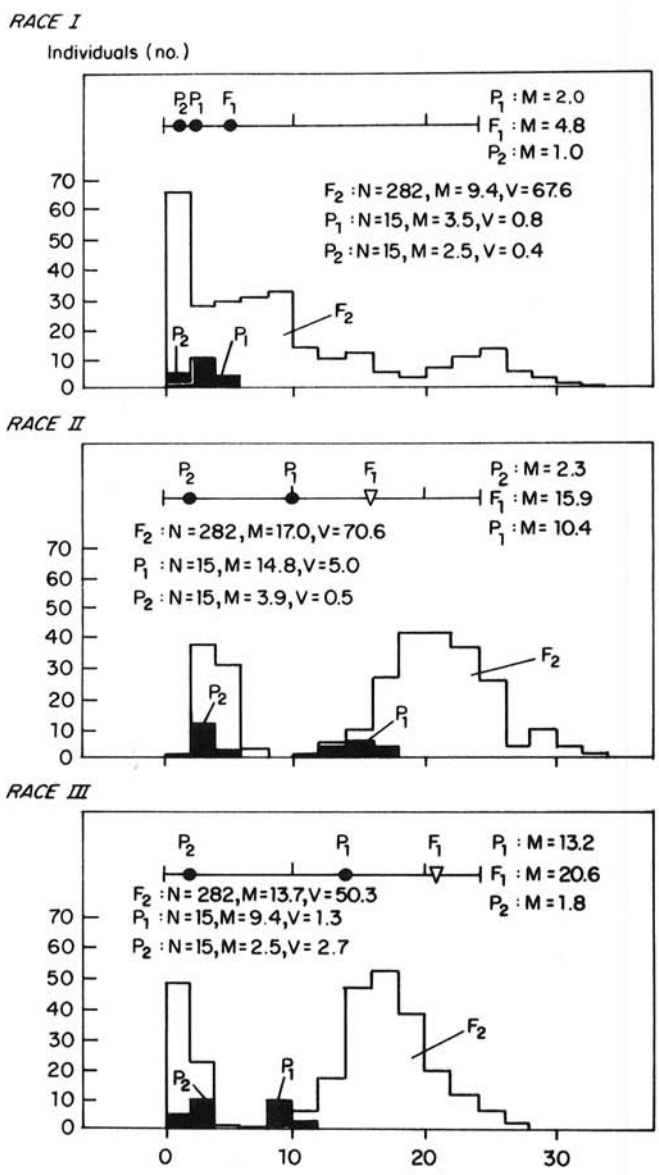

Fig. 2. Reactions to *Xanthomonas campestris* pv. *oryzae* strains PX061, PX086, and PX079 of parental and F_1 plants and frequency distributions of disease scores of parental and F_2 populations from the cross of rice cultivars IR20 (P_1)/IR1545 (P_2). M = mean lesion length (cm), N = number of individuals, V = variance. Black area = parents, white area = F_2 plants.

TABLE 1. Reaction of F_1 plants and number of F_2 plants in each of the reaction patterns from the crosses of rice cultivar IR1545-339 with Taichung Native 1 and IR20 to *Xanthomonas campestris* pv. *oryzae* strains PX061, PX086, and PX079

Cross	Reaction of F_1 plants	Reaction of F_2 populations			Total	χ^2	P
		RRR	RSS	SSS			
TN1/IR1545	SSS[a]	34 (31.0)		90[b] (93.0)[c]	124	0.387 (d.f. = 1)	0.75–0.50
IR20/IR1545	RSS	73 (70.5)	160 (158.6)	50 (52.9)[d]	282	0.257 (d.f. = 2)	0.90–0.75

[a] R = resistant, S = susceptible. For combined capital letters, the first letter stands for reaction to PX061 (Race 1), the second to PX086 (Race 2), and the third to PX079 (Race 3).
[b] Observed segregation.
[c] Calculated on the basis of 1:3.
[d] Calculated on the basis of 4:9:3.

TABLE 2. Bacterial blight lesion lengths caused by *Xanthomonas campestris* strains pv. *oryzae* PX061 (Race 1) and PX071 (Race 4) in plants of rice cultivars Taichung Native 1 (TN1) and IR1545 at the maximum tillering stage

Environment		Lesion length ± s.d.[a] (mm)			
		PX061		PX071	
Time inoculated	Place grown	TN1	IR1545	TN1	IR1545
20 July 1980	Screenhouse	217 ± 31	27 ± 15	304 ± 37	153 ± 38
9 January 1981	Screenhouse	217 ± 23	10 ± 0	193 ± 26	63 ± 10
14 May 1981	Greenhouse	364 ± 46	18 ± 7	293 ± 45	85 ± 23
20 July 1981	Screenhouse	270 ± 50	25 ± 8	311 ± 42	140 ± 21

[a] Scored at 14 days after inoculation.

The F_2 population from the cross IR20/IR1545 segregated in the ratio of 4RRR:9RSS:3SSS, which would be expected for independent segregation of two genes (*Xa*-4 of IR20 and *xa*-5 of IR1545), thus confirming the earlier results that *Xa*-4 is independent of *xa*-5 (11). In this F_2 population also, all the plants resistant to PX079 were also resistant to PX061 and PX086, again confirming the conclusion drawn from the F_2 population of TN1/IR1545.

Moderate resistance of IR1545 to PX071 and the mode of its inheritance. The lesion length caused by PX071 on TN1 was consistently greater than on IR1545 (Table 2). Lengths of lesions on both entries caused by PX061 are also shown in the table. Although the lesion length in different tests varied, lesions on TN1 were always longer than those on IR1545. Lesions of IR1545 caused by PX071 were considerably shorter than those on TN1 in all tests. To characterize the reaction of IR1545 to PX071, lesion development was observed from 6 to 24 days after inoculation. At each day, the lesions on IR1545 were considerably shorter than those on TN1. Lesions on TN1 expanded exponentially, but those on IR1545 expanded slowly and ceased growing by 18 days after inoculation. To linearize these lesion growth curves, the Gompertz model (1) was applied. A distinct difference was observed between lesion growth rates for PX071 on TN1 and on IR1545 (Fig. 3).

The lengths of lesions on parental and F_1 plants, and frequency distribution of disease scores of F_2 population from the cross TN1/IR1545 when inoculated with PX071 are shown in Fig. 4. F_2 population showed a wide range of variation with lesion length varying from 135 to 465 mm. The distribution deviated from normal ($P<0.005$) and appeared to possess two modes. One mode was within the lesion length range of 160–220 mm and the other within the lesion length range of 300–340 mm. The former corresponded to the reaction of IR1545; the latter, which included more of the plants, corresponded to the reaction of TN1. The results suggested that the moderate resistance of IR1545 to PX071 was recessive and appeared to be under major oligogenic control.

Relationship between *xa*-5 and the moderate resistance of IR1545 to PX071. The relationship between the moderate resistance of IR1545 to PX071 and high resistance to PX061, PX086, and PX079 governed by *xa*-5 was examined in the F_2 population from TN1/IR1545. The F_2 population, when scored for resistance to PX061, PX086, and PX079, segregated 94 RRR and 230 SSS plants. The plants showing RRR reaction were homozygous for *xa*-5. When the same F_2 population was inoculated with PX071, the plants with *xa*-5 showed lower susceptibility (Fig. 4). The distribution was approximately normal and corresponded to the reaction of IR1545. The plants heterozygous for *xa*-5 or homozygous dominant at this locus had large lesions. Their distribution of lesion length corresponded to that of TN1, showing normal distribution. The data revealing the relationship between *xa*-5 and moderate resistance to PX071 are summarized in Table 3. The lesion lengths caused by PX071 in this F_2 population were classified into nine classes having a 40-mm interval for lesion length. The mean lesion length of the group of plants homozygous for *xa*-5 was significantly lower than that of the group of plants which either lacked or were heterozygous for *xa*-5. Thus, there was a strong association between high resistance to PX061, PX086, and PX079 conveyed by *xa*-5 and the moderate resistance to PX071. There was considerable variation in lesion development on IR1545 when infected with PX071. Both groups showed wide variation and their ranges overlapped. However,

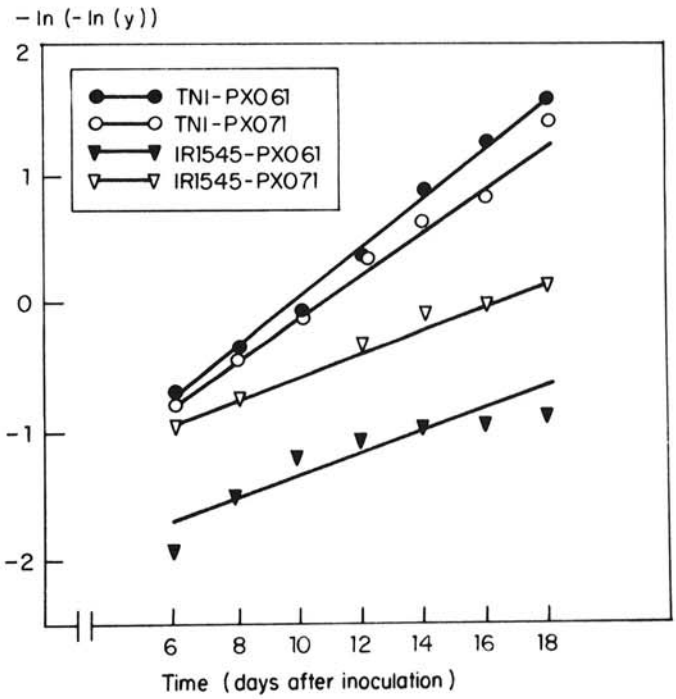

Fig. 3. Bacterial blight lesion development caused by *Xanthomonas campestris* strains PX061 (Race 1) and PX071 (Race 4) on rice cultivars Taichung Native 1 and IR1545-339. Lesion lengths were transformed with the Gompertz equation, $-1n(-1n(y))$, in which y = proportional lesion length (mean lesion length/mean leaf length). Each value is the average of 20 observations.

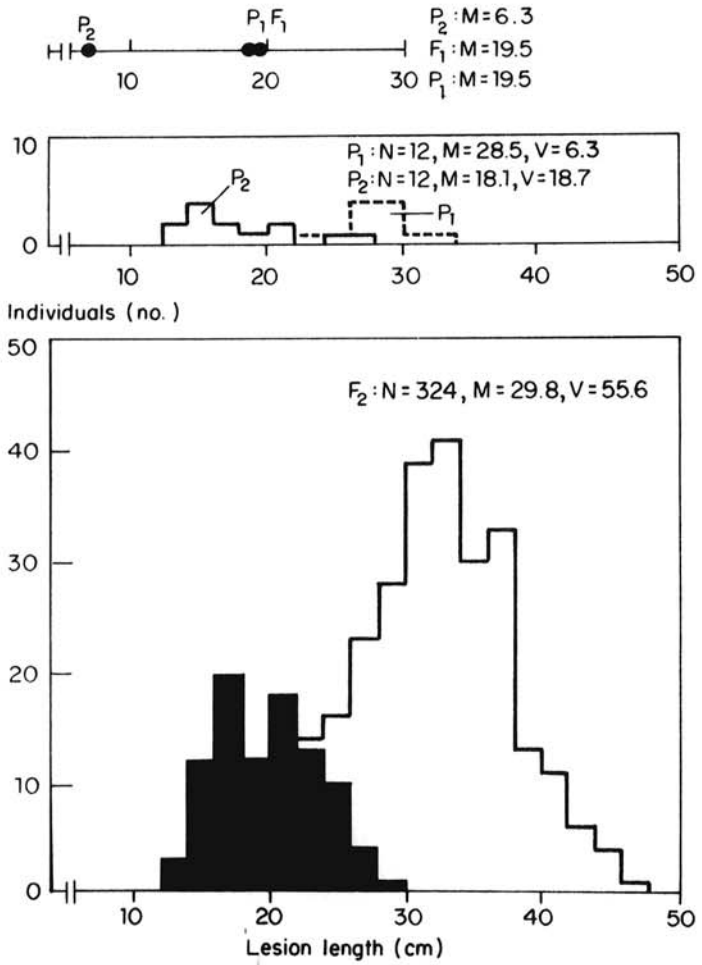

Fig. 4. Bacterial blight lesion lengths 14 days after inoculation with strain PX071 (Race 4) of *Xanthomonas campestris* pv. *oryzae* on parental and F_1 plants, and frequency distribution of disease scores of parental and F_2 population from the cross of rice cultivars Taichung Native 1 (P_1)/IR1545-339 (P_2). M = mean lesion length (cm), N = number of individuals, V = variance. The solid part = plants with *xa*-5 in homozygous state.

there was no plant in the group homozygous for *xa*-5 which showed high susceptibility to PX071 or a plant with low scores for PX071 in the group which lacked or was heterozygous for *xa*-5. Moreover, the variation pattern within each group appeared to be similar to that of the corresponding parents. The results suggested that the moderate resistance of IR1545 to PX071 is also governed by *xa*-5.

On the basis of results of the disease scores to PX086 (Race 1), the F_3 families from TN1/IR1545 were classified into three groups: *xa*-5 *xa*-5, *Xa*-5 *xa*-5, and *Xa*-5 *Xa*-5. The frequency distribution and the mean disease scores in lesion length of these families to PX071 are given in Table 4. The *xa*-5 *Xa*-5 families had short lesions and the *Xa*-5 *Xa*-5 families had long lesions. None of the *xa*-5 *xa*-5 families developed long lesions when scored at 14 days after inoculation. Analysis of variance for lesion length showed a significant difference among the three groups, and there was no significant difference between the disease scores of *xa*-5 *Xa*-5 families and that of IR1545 and between the disease scores of *Xa*-5 *Xa*-5 families and that of TN1. The results supported the conclusion drawn from F_2 data.

These results show that *xa*-5, which conveys high resistance to PX061, PX086, and PX079, is also responsible for slower lesion development caused by PX071.

Trisomic analysis of *xa*-5. F_2 populations from trisomic F_1 plants of the crosses of IR1545 with Triplo 7, Triplo 8, Triplo 9, and Triplo 12 segregated in a ratio of 1R:3S. However, the F_2 population from a trisomic F_1 plant of the cross Triplo 5/IR1545 deviated significantly from a disomic ratio of 1:3, although an F_2 population from a disomic F_1 plant of the same cross showed a good fit to the 1:3 ratio (Table 5). When the F_2 population from a trisomic F_1 plant was classified into disomic and trisomic fractions by morphological characteristics, the segregation for resistance gave a good fit to the theoretical trisomic segregation ratio calculated by assuming random complete assortment (12). These results suggest that the recessive gene for resistance to PX086 in IR1545 (*xa*-5) is located on the extra chromosome of Triplo 5.

DISCUSSION

The inheritance of resistance of IR1545 to bacterial blight was investigated earlier using a strain of race 1 of the bacterial blight organism. It was found that the single recessive gene *xa*-5 conveys

TABLE 3. Frequency distribution of lesion length of F_2 plants from the cross of rice cultivars Taichung Native 1/IR1545-339 with and without resistance gene *xa*-5 following inoculation with *Xanthomonas campestris* pv. *oryzae* strain PX071 (Race 4)

Genotype	Lesion length categories (mm)									Total	Mean lesion length (mm)
	120–160	160–200	200–240	240–280	280–320	320–360	360–400	400–440	440–480		
xa-5 *xa*-5	15 (4.4)[a]	32 (9.3)	31 (9.3)	14 (11.3)	1 (19.4)	0 (20.6)	0 (13.4)	0 (4.9)	0 (1.5)	94	203
Xa-5 *Xa*-5	0 (10.6)	0 (22.7)	1 (22.7)	25 (27.7)	66 (47.6)	71 (50.4)	46 (32.6)	17 (12.1)	5 (3.5)	230	337
Total	15	32	32	39	67	71	46	17	5	324	

$\chi^2 = 268.95$, d.f. = 8, $P < 0.005$ $t = 251$, $P < 0.001$

[a] Expected values calculated based on the observed ratio of 94:230. *T*-test was based on mean lesions of observed plants in each class of the two genotypes.

TABLE 4. Frequency distributions of mean lesion length on three genotypes of F_3 families from the cross of rice cultivars Taichung Native 1/IR1545-339 inoculated with bacterial strain PX071 of *Xanthomonas campestris* pv. *oryzae*

Genotypes	Lesion length categories (mm)											Total (lines)	Mean lesion length (mm)[a]
	80–100	100–120	120–140	140–160	160–180	180–200	200–220	220–240	240–260	260–280	280–300		
xa-5 *xa*-5	3	6	5	2	1							17	123.49 ± 23.99
Xa-5 *xa*-5		2	2	11	9	6	1	2				33	165.33 ± 27.80
Xa-5 *Xa*-5				2	4	4	2	2	0	0	1	15	189.66 ± 39.00
Total	3	8	7	15	14	10	3	4	0	0	1	65	

[a] Based on values among lines; the number of plants per line ranged from 17 to 20 plants. Note: Mean lesion lengths were: TN1, 202.63 ± 13.06 mm; and IR1545-339, 118.75 ± 15.55 mm.

TABLE 5. Trisomic segregation of gene *xa*-5 for resistance to *Xanthomonas campestris* pv. *oryzae* strain PX086 in F_2 populations from the cross rice cultivar IR1545-339 with trisomic line Triplo 5

Cross	Fraction of population	Observed number		Total	χ^2 for		
		+	*xa*-5		3:1	8:1	44:1
Triplo 5/IR1545[a]	2X	70	8	78	9.043*	0.058	...
	2X + 1	35	2	37	7.577*	...	1.725
Total		105	10	115	16.304*	...	...
Triplo 5/IR1545[b]	2X	125	42	167	0.002	...	...

[a] F_2 population from a trisomic F_1 plant.
[b] F_2 population from a disomic F_1 plant.
[c] Asterisk indicates statistical significance, $P = 0.05$.

resistance to race 1 (11). Because IR1545 is also resistant to races 2 and 3 (7), it is used as a differential for classifying bacterial strains into specific races. However, the mode of inheritance of IR1545 resistance to races 2 and 3 was not known. This study clearly reveals that *xa*-5 also confers resistance to races 2 and 3. Because IR1545 is also being used as a resistance source in our breeding program, any of the three races may be used for screening the segregating populations and the breeding lines thus selected would be resistant to all three races.

IR1545 is moderately resistant to strain PX071, which is a representative of race 4. Lesions elongate more slowly and do not become as large as on fully susceptible cultivars. During the course of this investigation, strong winds occurred after IR1545 and TN1 had been inoculated with bacteria of race 4. We observed that the secondary infection on IR1545 was much lower than that on the highly susceptible TN1. The implication of the moderate resistance of IR1545 in a bacterial blight epidemic deserves further study.

The results clearly show that the resistance of IR1545 to the three strains is controlled by the same recessive gene. Olufowote et al (10) reported that the resistance of IR1545 to a strain belonging to race 1 was controlled by *xa*-5. Our data confirm their results and also show that the resistance of IR1545 to the strains belonging to races 2 and 3 is also governed by *xa*-5.

The analysis of F_2 and F_3 populations inoculated with race 4 indicated that *xa*-5 may also be responsible for slow lesion development on IR1545. This would mean that the slow lesion development characteristic could be incorporated into the newer material even if they are screened with races 1, 2, and 3.

The trisomic analysis revealed that *xa*-5 is located on the extra chromosome of Triplo 5. Triplo 5 corresponds to the L-type of trisomic developed in a *japonica* cultivar by Iwata et al (4). The extra chromosome of the L-type trisomic corresponds to chromosome 2 of Nishimura's designation (3,8). Therefore, it is concluded that *xa*-5 is located on chromosome 2 of Nishimura's designation.

Eleven genes for resistance to bacterial blight of rice (*Xa*-1, *Xa*-2, *Xa*-3, *Xa-kg*, *Xa*-4, *xa*-5, *Xa*-6, *Xa*-7, *xa*-8, *xa*-9, and *Xa*-10) have been identified to date (2,9,11,13,16,18). *Xa*-1 and *Xa*-2 and *Xa*-2 and *Xa-kg* are closely linked and are located on chromosome 11 (2,9). *Xa*-6, *xa*-9, and *Xa*-10 are linked with *Xa*-4 (15,16,18). Thus, these four genes are located on the same chromosome. It seems there is a tendency for bacterial blight resistance genes to cluster on a few chromosomes.

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