Inheritance and Selection of Field Resistance to Sheath Brown Rot Disease in Rice

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ABSTRACT

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Chilling temperatures induce sheath brown rot disease (ShBR) in rice (*Oryza sativa*) caused by *Pseudomonas fuscovaginae*. The major symptoms of the disease are poor exsertion of the panicle from the boot and spikelet sterility. The inheritance of field resistance was studied in F_3 families derived from three rice crosses involving a field-resistant (R) parent, two intermediate (I) parents, and a susceptible (S) parent. Each cross was assessed at a different altitude to give three independent data sets. Heritability (h^2) estimates of disease resistance traits were determined by analysis of F_3 family means in crosses Raksali (S) × Makwanpur-15L (I), Makwanpur-3L (I) × Chhomrong (R), and IR36 (S) × Chhomrong (R). There were highly significant differences between F_3 family means in all cases and heritabilities were consistently high (0.72 to 0.84). High h^2 estimates and high variance between F_3 families resulted in high predicted genetic gains from selection, so field screening in the F_3 generation for ShBR resistance should be effective. Mean, additive, and dominance gene effects were estimated from generation means analyses. A statistically significant model could be fitted in all three crosses, and additive effects were always important. Dominance was also an important genetic effect, and its direction was significantly toward resistance in two of the crosses but, with lower significance, toward susceptibility in the third. Significant correlations were found between ShBR field resistance and plant height and panicle exsertion from the boot. Selection, in the presence of the disease, for taller plants and better panicle exsertion will give a genetic gain for greater chilling tolerance and higher ShBR field resistance.

Bacterial sheath brown rot (ShBR), caused by *Pseudomonas fuscovaginae* (9, 10,12) is the most important disease of rice (*Oryza sativa* L.) in high-altitude, high-rainfall environments of Nepal (16), and poor rice yields in these areas are often associated with ShBR spikelet sterility. This disease is also economically important in rice in the mid- to high-altitude regions of Burundi (2), Japan (12), Latin America (15), Rwanda (16), and Zaire (16). It was reported in wheat in the highlands of Mexico (1). Chilling daytime temperatures (17°C and below) and high relative humidity at the booting and heading stages are favorable to infection and development of the disease (8,9). *Pseudomonas fuscovaginae* survives in infested straw, on grain crops, and on wild grasses. It is also seed-borne and seed-transmitted (8). Severe ShBR infection, the symptoms of which first appear at the heading stage, causes total spikelet sterility by preventing normal exsertion of the panicle from the boot. The disease can eventually lead to the death of the entire plant. In mild ShBR infection, adult plants have characteristic gray brown to brown lesions on the sheaths of the flag leaves (8) from the booting to the heading stages, but panicles still emerge and produce a reduced grain yield. Although estimates have not been reported for yield losses caused by ShBR, the disease is likely to be economically important. Breeding disease-resistant cultivars appears to be the best option for controlling this disease.

Little is known about the inheritance of ShBR resistance in rice, and about Nepalese rice germ plasm reactions. Exotic chilling-tolerant cultivars, when introduced into the highlands of Nepal, are often highly susceptible to ShBR disease, while some indigenous cultivars such as Chhomrong, Seto Bhakunde, Kalopatle, and Darmali are field resistant.

The objectives of this study were to determine the inheritance and heritability of resistance to ShBR in the progeny of three crosses made among two chilling-tolerant Nepalese cultivars that were field-resistant or partially field-resistant to ShBR, and two ShBR susceptible cultivars, one exotic and one indigenous.

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MATERIALS AND METHODS

Genetic material. F_1, F_2, and F_3 generations were derived from three crosses with the least resistant parent of each cross listed first: Raksali-5L × Makwanpur-1-15L (R × M-15L), Makwanpur-1-3L × Chhomrong-3L (M-3L × C), and IR36 × Chhomrong-1L (IR36 × C). In M-3L × C the second parent was the female, and in the other two crosses the first parent listed was the female.

For brevity, the suffixes in the cultivar names are omitted or abbreviated hereafter. The ShBR field-resistant parents were the two lines of Chhomrong (strongly field-resistant to ShBR, chilling-tolerant). The two intermediate parents were Makwanpur-15L and Makwanpur-3L (moderately field-resistant to ShBR, chilling-susceptible, and late-maturing). In disease evaluations at Lumle, the lines of Chhomrong have been consistently scored as 1, and both lines of Makwanpur as 5, using a 1 to 9 scale in which 1 = no disease symptoms to 9 = severely diseased. IR36 and Raksali, the susceptible parents, in the same evaluation had ShBR scores from 7 to 9. In 1991, crosses were made between the parents in a glass house at the University of Wales, Bangor.

Production of F_2 and F_3 seed. In 1992, the F_2 and F_3 seed was produced in two growing seasons at low altitude (475 m) in Nepal to avoid chilling stress and ShBR disease. In the first of these, F_2 seed was produced from each of the three F_1s. In the second, F_3-family seed was harvested from individual F_2 plants. For all genotypes, 20-day-old seedlings were singly transplanted into puddled rice fields in a square grid pattern with 25 cm between the seedlings. At maturity, seed was harvested from the individual plants. The fields were fertilized at transplanting with the equivalent of 90 kg ha^{-1} N, 30 kg ha^{-1} P_2O_5, and 30 kg ha^{-1} K_2O. Thirty kg ha^{-1} each of N, P_2O_5, and K_2O was applied at the time of puddling, and two further top-dressings, each of 30 kg ha^{-1} of N, were applied as urea 25 and 45 days after transplanting. Before each top-dressing, the plots were weeded.

Evaluation of genetic material. In 1993, rice crosses were evaluated in terraced fields at three research sites of the Lumle Agricultural Research Centre, Nepal: Chhomrong (2,000 m), Ghandruk (2,000 m) and Lumle (1,450 m). At each location, one cross was evaluated by growing the parental lines, and the F_2 bulk and F_3 families from the cross (Table 1) in a randomized complete block design with three replications. To increase the accuracy of the measurements on parental lines (P_1 and P_2) each line was replicated 12 times. Seedlings were transplanted into plots that consisted of single 2-m-long rows. The rows were 30 cm apart and the plants 25

cm apart within the rows. Plots were fertilized at transplanting with 60-30-30 kg NPK ha^{-1} at Chhomrong. No chemical fertilizer was applied at Ghandruk nor at Lumle as the soil fertility was already very high.

Evaluations were made under natural conditions. The environment during the growth period was favorable for development of the disease. In all three locations average minimum temperatures were below 17°C after September when the plants began to boot (Table 1). Plots were not artificially inoculated since infections in previous seasons indicated a sufficiency of natural inoculum at all sites, and because no standard inoculation technique has been developed for this pathogen. Instead of the severity scale used to score the parents in the routine nursery evaluations at Lumle, incidence was determined. The total number of tillers and number of infected tillers were counted on each plant in each plot. The percent tillers infected by ShBR at maturity was then calculated as disease incidence.

Isolation of *P. fuscovaginae* from diseased tissues of cvs. IR36 and Raksali by cooperators at IRAT, France, and the International Mycological Institute, U.K., confirmed the bacterium was associated with the disease.

Analyses. Analyses of variance (ANOVA) for a randomized block design (11) were used to evaluate the significance of differences among the parental, F_2, and F_3 generations for disease incidences. Standard errors of generation means were determined. Separate ANOVA for randomized blocks were used to examine the differences among the F_3 families for disease incidence.

Using the variance components of the ANOVA, family-mean heritabilities were calculated for the across-replicate means of the disease incidence of F_3 families (5). Heritabilities were also determined on the basis of individual replicate (i.e., plot) values (5). The expected mean square for families (MS_f) is $\sigma^2 + r\sigma^2_g$ and for the mean square for error (MS_e) is σ^2. The family-mean heritability is $\sigma^2_g/(\sigma^2_g + \sigma^2/r)$ where r is the number of replicates. Therefore the family-mean heritability = $[(MS_f - MS_e)/r]/(MS_f/_r)$ since the phenotypic variance of family-mean values is: $\sigma^2/r + \sigma^2_g = MS_f/r$ (5,14). The plot heritabilities $\sigma^2_g/(\sigma^2_g + \sigma^2)$ were calculated as $[(MS_f - MS_e)/r]/[(MS_f - MS_e/r) + MS_e]$.

The predicted response to direct selection (R) was estimated as $R = h^2S$, where h^2 is the family-mean heritability of the selected trait, and S is the unstandardized selection differential (the mean superiority of the selected parents) and was the difference between the mean of selected families (at 10% selection intensity) and the overall population mean (4). The predicted genetic gain of various traits was also estimated as a percentage of the original unselected population mean.

We used computer software developed and provided by John Snape, John Innes Centre, Norwich, U.K., to carry out a generation means analysis (6,7,13) for each cross. Means and variances of four generations, P_1 (always the more susceptible parent), P_2 (always the more resistant parent), F_2, and F_3, of each of the three crosses were used to estimate genetic parameters. Standard errors of estimates of genetic parameters were obtained from variances of individual plant data after removal of replicate effects. The significance of the deviation of the estimated genetic parameters from zero was tested by a Student's t test (11).

Correlations were calculated between various traits on the basis of the F_3 family phenotype means.

RESULTS

Genetic variation. Significant differences were found between the generation means for ShBR infection in an ANOVA that included the four generations (P_1, P_2, F_2 bulk, and F_3 families) (Table 2). Levels of field resistance to ShBR in the parents were similar to those in previous evaluations. Chhomrong had a high level of resistance, Makwanpur-3L was partially resistant, and IR36 and Raksali were susceptible (Table 2). However, Makwanpur-15L was almost as resistant as Chhomrong, probably because Makwanpur-15L was evaluated at a lower altitude. Such a reduction in disease at lower altitudes has been reported in Malagasy (3).

A separate analysis of the F_3 families revealed significant ($P \leq 0.001$) variation

Table 1. Description of tests in which four generations of three rice crosses were evaluated for plant characteristics and resistance to sheath brown rot in Nepal in 1993

Cross[a]	No. of F_3 families	Location	Altitude (m)	Date of transplanting	Date of 50% booting in F_3 families	Temperature[b]
M-3L × C	193	Chhomrong	2,000	4 April 1993	7 to 25 August	11 to 16
IR36 × C	101	Ghandruk	2,000	7 April 1993	8 to 22 August	15 to 16
R × M-15L	161	Lumle	1,675	21 April 1993	18 to 30 October	14 to 16

[a] R × M-15L = Raksali × Makwanpur-15L, M-3L × C = Makwanpur-3L × Chhomrong, IR36 × C = IR36 × Chhomrong.
[b] Average minimum temperature from booting to heading (°C).

Table 2. F_3 and parental means for disease incidence (tillers $plant^{-1}$ infected with sheath brown rot), ranges of F_3 family means, and the F_3 heritabilities in three rice crosses evaluated in Nepal in 1993

			Disease incidence (%)			h^2 in F_3	
Cross[a]	Generation and parents	Plots[b]	Generation means ± SE	Range of F_3 family means	Significance[c]	Entry mean	Plot
R × M-15L	F_3	579	36.9 ± 14.9	10.2 to 77.3	***	0.84	0.64
	F_2	30	28.1 ± 11.5				
	P_1 (R)	120	49.8 ± 4.8				
	P_2 (M-15L)	120	9.4 ± 2.1				
M-3L × C	F_3	303	12.8 ± 7.1	2.5 to 34.1	***	0.72	0.46
	F_2	30	7.0 ± 0.7				
	P_1 (M-3L)	120	27.3 ± 2.9				
	P_2 (C)	120	2.6 ± 0.9				
IR36 × C	F_3	483	24.4 ± 10.7	2.3 to 56.2	***	0.76	0.51
	F_2	30	21.2 ± 10.1				
	P_1 (IR36)	120	56.6 ± 4.4				
	P_2 (C)	120	7.9 ± 1.5				

[a] R × M-15L = Raksali × Makwanpur-15L, M-3L × C = Makwanpur-3L × Chhomrong, IR36 × C = IR36 × Chhomrong.
[b] Total number of plots in trial for that generation.
[c] *** = $P \leq 0.001$ for significance of differences among generation means, and among F_3 families.

Table 3. Estimates of genetic effects (m, d, h, SE) fitted to a three-parameter model in three rice crosses evaluated for disease incidence of sheath brown rot in Nepal in 1993[a]

	Genetic parameter			
Cross[b]	m	[d]	[h]	χ^2value[c]
R × M-15L	30.4 ± 2.5	20.7 ± 2.6***[d]	25.4 ± 10.5*	1.8 >0.1(NS)
M-3L × C	17.1 ± 1.0	14.1 ± 1.2***	−19.2 ± 2.8***	3.2 >0.1(NS)
IR36 × C	32.0 ± 2.3	24.1 ± 2.3***	−30.2 ± 9.3**	0.2 >0.1(NS)

[a] Three parameter model, m = mean, [d] = additive, [h] = dominance, genetic effects for the model y = m+[d]+[h] where y = the generation mean.
[b] R × M-15L = Raksali-× Makwanpur-15L, M-3L × C = Makwanpur × Chhomrong, IR36 × C = IR36 × Chhomrong.
[c] Value for deviations from the model. Nonsignificance (NS) indicates that model fits.
[d] *** = $P \leq 0.001$, ** = $P \leq 0.01$, and * = $P \leq 0.05$.

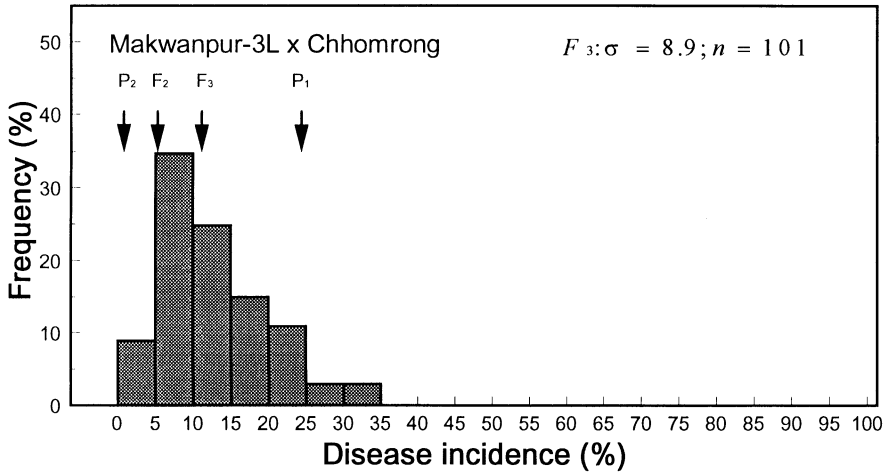

Fig. 1. The Makwanpur-3L × Chhomrong cross. Frequency distributions of F_3 family means for bacterial sheath brown rot (ShBR) disease incidence (percent tillers plant^{-1} infected). Means of the parental lines, and of the F_2, and F_3 generations are indicated by arrows.

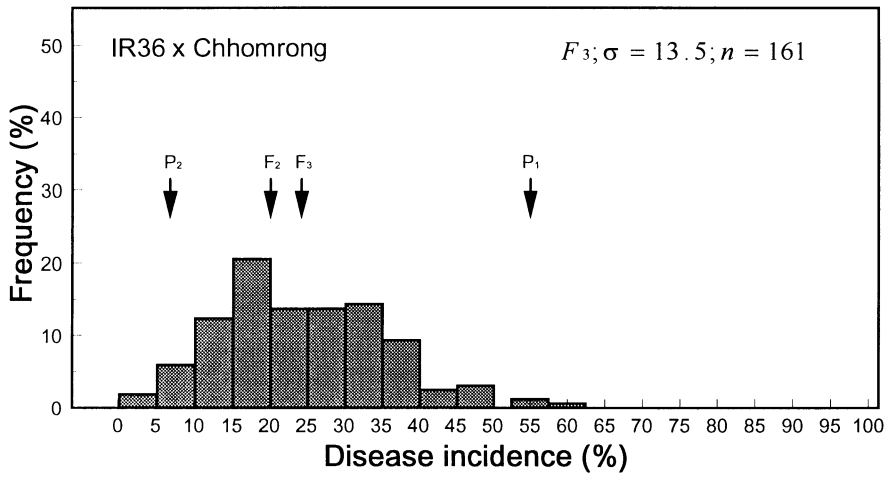

Fig. 2. The IR36 × Chhomrong cross. Frequency distributions of F_3 family means for bacterial sheath brown rot (ShBR) disease incidence (percent tillers plant^{-1} infected). Means of the parental lines, and of the F_2, and F_3 generations are indicated by arrows.

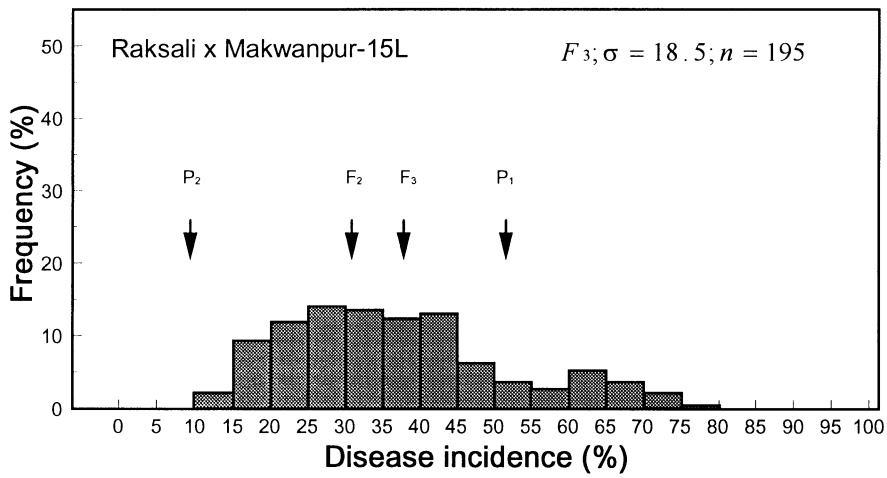

Fig. 3. The Raksali × Makwanpur-15L cross. Frequency distributions of F_3 family means for bacterial sheath brown rot (ShBR) disease incidence (percent tillers plant^{-1} infected). Means of the parental lines, and of the F_2, and F_3 generations are indicated by arrows.

in all crosses for disease incidence (Table 2).

The three crosses involved a field-resistant (R) parent, two intermediate (I) parents, and a susceptible (S) parent, and the overall F_3 family means differed in accordance with the resistance of the parents (Table 2). Disease incidence was lowest in the Makwanpur-3L (I) × Chhomrong (R) cross, despite being grown at the highest of the three altitudes, and was higher in the IR36 (S) × Chhomrong (R) and Raksali (S) × Makwanpur-15L (I) crosses, each of which had one very susceptible parent.

No ShBR symptoms were observed in any generation of any cross grown under nonstressed (mean air temperature of 24 to 32°C at heading) conditions at Yampaphant at 475 m (data not shown).

Generation means analysis. There was a significant fit to a three parameter generation mean model in all three crosses (Table 3). Inspection of the generation means (Figs. 1 and 2), shows that for M-3L × C and IR36 × C the disease incidence in the F_2 and F_3 generations was not equal to the average of the two parents, but that resistance was dominant. In these two crosses, the F_3 generation mean changed with respect to the F_2 mean in accordance with the expected reduction in dominance from the F_2 to the F_3. This was confirmed by significant dominance parameters calculated for these two crosses (Table 3). In contrast, in R × M-15L susceptibility was dominant, but at a low level of probability (Table 3). Since susceptibility appeared dominant, the F_2 was expected to be more susceptible than the F_3. However, the F_2 was more resistant (Fig. 3), and deviated from the expected model value of 43% disease incidence by −15%. Overall we conclude that there is a weak case for susceptibility being dominant in this cross.

Heritability in the F_3 families. The heritability of the means of the F_3 families for incidence of ShBR disease was high in all three crosses (Table 2). Component traits of percent disease, number of tillers plant^{-1} and infected tillers plant^{-1}, were also highly heritable (data not shown). Family-mean heritabilities increase with increasing replication so they will vary greatly across experiments with differing numbers of replications. For comparative purposes, the effect of replication was removed by estimating the plot heritabilities for ShBR disease incidence. Although the plot heritabilities have to be lower than family-mean heritabilities, the plot heritabilities were still high (Table 2).

Predicted genetic gain to selection. The predicted genetic gains for disease resistance by selection for reduced disease incidence among F_3 families in the three crosses at 10% selection intensity were high (gains of 46 to 50% of the unselected population mean) and varied by only 4% across the three crosses (Table 4). The predicted responses to selection for reduction in the number of ShBR infected tillers were also consistently high (47 to 50%) (Table 4).

Phenotypic correlations. In all three crosses, the incidence of ShBR disease was negatively correlated ($P \leq 0.001$) with plant height at growth stage 9 (near to maturity) ($r = -0.38$ to -0.55) indicating that high disease incidence was associated with reduced plant height (Table 5). Similarly, a significant positive correlation was

Table 4. Estimates of genetic gains and their associated parameters for two sheath brown rot disease traits in three crosses of rice evaluated in Nepal in 1993

Cross and traits	F_3 families: All n^a	All X^b	Selected x^c	S^d	h^2	R^e	Gain (%)[f]
Raksali × Makwanpur-15L							
ShBR disease incidence (%)	193	36.9 ± 14.9	16.5 ± 2.3	20.4	0.84	17.14	46
Infected tillers (No plant^{-1})	193	5.2 ± 2.5	2.1 ± 0.4	3.1	0.79	2.45	47
Makwanpur-3L × Chhomrong							
ShBR disease incidence (%)	101	12.8 ± 7.1	3.8 ± 1.0	9.0	0.72	6.48	50
Infected tillers (No plant^{-1})	101	1.3 ± 0.8	0.4 ± 0.1	0.9	0.71	0.64	49
IR36 × Chhomrong							
ShBR disease incidence (%)	161	24.4 ± 10.7	8.20 ± 2.9	16.2	0.76	12.31	50
Infected tiller (No plant^{-1})	161	0.2 ± 0.2	0.02 ± 0	0.2	0.54	0.10	50

[a] Number of F_3 families.
[b] Mean of unselected population.
[c] Mean of 10% selected F_3 families.
[d] X – x.
[e] Predicted response to selection ($S \times h^2$).
[f] Genetic gain (%) = [R/X] × 100.

found between the ShBR disease incidence and panicle exsertion in all the crosses (Table 5), indicating that disease incidence was associated with poor exsertion of panicles from the boot. However, the effects of this disease and chilling injury cannot be separated. ShBR disease and chilling injury both require chilling temperatures to develop and both give rise to poor panicle exsertion.

No other consistent and significant relationship was found between ShBR disease and the other agronomic and phenological traits that were measured (Table 5).

Table 5. Phenotypic correlations (r) among means of F_3 families for sheath brown rot disease (%) and various plant characteristics in three rice crosses evaluated in Nepal in 1993

Traits	Correlation (r) to ShBR disease: R × M-15L[a]	M-3L × C[a]	IR36 × C[a]
Number of tillers plant^{-1}	0.14	–0.18	–0.07
Chilling tolerance at GS6 (scale)[b]	–0.06	0.10	0.11
Plant height at GS9 (cm)[c]	–0.55***[d]	–0.38***	–0.48***
Panicle exsertion (scale)	0.49***	0.43***	0.50***
Panicle weight (g plant^{-1})	–0.05	–0.01	–0.16
Seed yield (g plant^{-1})	0.01	–0.03	–0.16
Spikelet sterility (%)	–0.02	0.06	0.26**
Time to 50% boot (days)	–0.08	–0.11	0.04
Time to 50% flowering (days)	–0.13	–0.07	–0.07
Panicles plant^{-1}	0.10	–0.21*	–0.01
Sterile floret weight (g plant^{-1})	–0.09	0.06	–0.08
Seeds plant^{-1}	0.00	–0.03	–016
Total florets plant^{-1}	–0.11	0.11	–0.16

[a] R × M-15L = Raksali × Makwanpur-15L, M-3L × C=Makwanpur-3L × Chhomrong, IR36 × C = IR36 × Chhomrong.
[b] Growth stage at flowering.
[c] Growth stage near maturity.
[d] *** = $P \leq 0.001$, ** = $P \leq 0.01$, and * = $P \leq 0.05$.

DISCUSSION

Resistance to ShBR was shown to be under genetic control in the three crosses. This was shown in three separate evaluations each of which had sufficient replication and numbers of families to give reliable results. Since the inheritance was demonstrated in three environments under natural disease inoculum pressures, it is more likely that the results will be reproducible by others. High heritability in three crosses estimated in a single environment could be due to location-specific effects such as a very high disease pressure and exceptionally low environment heterogeneity.

Estimates of family-mean heritability vary with the generation to which the families belong. The heritability estimates used here to calculate responses to selection were relevant to selection among F_3 families, a generation in which plant breeders commonly exert high selection pressures in pedigree breeding schemes. Heritabilities and genetic variances were high enough in all crosses to give worthwhile predicted responses to selection. There was very little variation in the predicted responses to selection among the crosses.

The heritabilities were also high enough to indicate that rejection of infected plants in the F_2 will give genetic advances. However, dominance of resistance was an important genetic component of variance in two of the crosses, indicating that many disease-free plants selected in the F_2 generation will give rise to susceptible segregants. The different directions of dominance, toward either resistance or susceptibility, among the two crosses involving Makwanpur may be due to genetic differences, or due to genotype × location interactions, or due to lack of precision in the dominance estimates. The estimate of dominance of susceptibility in R × M-15L (Table 3) was significant only at $P \leq 0.05$, so the last explanation is most likely.

Phenotypic correlations between disease incidence and the easily measured traits of plant height and panicle exsertion means that resistance to ShBR could be gained as an indirect response to selection for plant height and panicle exsertion. However, for indirect selection to be effective, natural disease pressure must be sufficiently high to produce moderate to high disease incidence. Considerable resources would be saved by selecting for height instead of low disease incidence. Indirect responses are expected to be high because of high correlations between the traits (Table 5) and high F_3 family mean heritabilities of plant height (0.83 to 0.95) and panicle exsertion (0.75 to 0.82).

Although resistance was evaluated at growth stage 9, observations of disease progress after growth stage 6 may identify genotypes with slow ShBR development (i.e., those with partial resistance). More information is also needed on the relationship between ShBR resistance and yield.

This study showed that resistance to ShBR in rice is under genetic control, that indirect or direct selection for resistance in the field is predicted to be effective, and that there is evidence for more than one source of genetic resistance to the disease. Cultivars derived from germ plasm with ShBR resistance may have higher and more stable yields, and if this ShBR resistance is combined with sufficient chilling resistance it will allow rice cultivation to be extended to higher altitudes.

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LITERATURE CITED

1. Duveiller, E., and Maraite, H. 1990. Bacterial sheath rot of wheat caused by *Pseudomonas fuscovaginae* in the highlands of Mexico. Plant Dis. 74:932-935.
2. Duveiller, E., Miyajima, K., Snacken, F., Autrique, A., and Maraite, H. 1988. Characterization of *Pseudomonas fuscovaginae* and differentiation from other fluorescent *Pseudomonas* occurring on rice in Burundi. J. Phytopathol. 122:97-107.
3. Duveiller, E., Nottenghem, J. L., Rott, P., Snacken, F., and Maraite, H. 1990. Bacterial sheath brown rot of rice caused by *Pseudomonas fuscovaginae* in Malagasy. Trop. Pest Manage. 36:151-153.
4. Falconer, D. S. 1989. Introduction to Quantitative Genetics. 3rd ed. Longman Scientific and Technical, London.
5. Fehr, W. R.. 1987. Principles of Cultivar Development. Vol. I, Theory and Technique. Macmillan, New York.
6. Gamble, E. E. 1962. Gene effects in corn (*Zea mays* L.). I. Separation and relative importance of gene effects for yield. Can. J. Plant Sci. 42:339-348.
7. Mather, K., and Jinks, J. L. 1982. Biometrical Genetics: The Study of Continuous Variation. 3rd edition. Chapman and Hall, London.
8. Mew, T. W. 1991. Rice diseases. Pages 187-236 in: Rice Production. Vol. 1, 2nd ed. B. S. Luh, ed. AVI Book, New York.
9. Miyajima, K. 1983. Studies on bacterial sheath brown rot of rice plant caused by *Pseudomonas fuscovaginae* Tanii. Hokkaido Prefect. Agric. Exp. Stn. Rep. 43.
10. Miyajima, K., Tanii, A., and Akita, T. 1983. Studies on bacterial sheath brown rot of rice plant caused by *Pseudomonas fuscovaginae* Tanii. Int. J. Syst. Bacteriol. 33:656-657.
11. Snedecor, G. W., and Cochran, W. G. 1980. Statistical Methods. The Iowa State University Press, Ames.
12. Tanii, A., Miyajima, K., and Akita, T. 1976. The sheath brown rot disease of rice plant and its casual bacterium, *Pseudomonas fuscovaginae* L. Ann. Phytopathol. Soc. Jpn. 42:540-548.
13. Wilkinson, C. A., Rufty, R. C., and Shew, H. D. 1991. Inheritance of partial resistance to black root rot in burley tobacco. Plant Dis. 75: 889-892.
14. Wricke, G., and Weber, W. E. 1986. Quantitative Genetics and Selection in Plant Breeding. W. de Grupter, Berlin.
15. Zeigler, R. S., and Alvarez, E. 1987. Bacterial sheath rot of brown rice caused by *Pseudomonas fuscovaginae* in Latin America. Plant Dis. 71:592-597.
16. Zeigler, R. S., and Alvarez, E. 1990. Characteristics of *Pseudomonas* spp. causing grain discoloration and sheath rot of rice, and associated pseudomonad epiphytes. Plant Dis. 74:917-922.