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The inheritance of host plant resistance and its effect on the relative infection efficiency of *Magnaporthe grisea* in rice cultivars

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Abstract The inheritance of host plant resistance and its effect on the relative infection efficiency for leaf blast was studied in the crosses 'IR36'/'CO39' (partially resistant × highly susceptible) and 'IR36'/'IR64' (both partially resistant). On the natural scale, gene action appeared multiplicative. After log transformation, additive effects described most of the genetic variation in the cross 'IR36'/'CO39', while additive and dominance effects were about equal in magnitude in the cross 'IR36'/'IR64'. Dominance was towards increased resistance. No transgressive segregation occurred in the cross 'IR36'/'CO39'. The number of genes that reduce lesion number was estimated to be zero in 'CO39' and five or more in 'IR36'. The cross 'IR36'/'IR64' showed transgressive segregation in both directions, and 'IR36' and 'IR64' each contain at least one gene that is not present in the other cultivar. The heritabilities (narrow sense) in the F_2 were low (range 0.06–0.16), while narrow sense heritabilities based on F_3 lines were much higher (range 0.41–0.68). Lesion numbers in F_3 lines were reasonably correlated with those in F_5 progenies derived from the same F_2 plant (r was ± 0.6 in both crosses). Partial resistance can be effectively improved by selecting the most resistant plants from the most resistant F_3 lines.

Key words Partial resistance · *Oryza sativa* *Magnaporthe grisea* · Blast · Inheritance

Introduction

Magnaporthe grisea, which causes blast disease of rice, occurs in nearly all rice growing areas (Ou 1985). The disease can seriously damage the crop, but effective control is possible by planting resistant cultivars. Highly resistant cultivars can be obtained with relative ease by selecting genotypes with effective hypersensitivity genes, but unfortunately the resistance of such cultivars usually breaks down soon after the cultivars are released (Ahn and Mukelar 1986; Ezuka 1972). On the other hand, in some traditional and improved cultivars that develop lesions of a typical susceptible type, the progress of the disease remains limited, and these cultivars are rarely damaged. The resistance in these cultivars, called partial resistance (Parlevliet and van Ommeren 1975), has remained effective for many years and seems to be durable (Johnson 1984). A major problem for breeders, however, is that the expression of partial resistance (PR), unlike that of the hypersensitivity resistance, is strongly influenced by environmental conditions, thus complicating its selection, especially in the early generations of a breeding cycle when replicated testing is often not possible.

With more knowledge of the inheritance of PR, the efficiency of selection in segregating generations could be improved. The most important component of PR to leaf blast in rice is the relative infection efficiency (RIE), measured as the number of sporulating lesions that develop (Yunoki et al. 1970; Notteghem 1985; Roumen 1992 a), and it is this component that was studied in order to get more insight into the genetics of PR in this pathosystem.

Materials and methods

Progenies of the crosses 'IR36'/'CO39' and 'IR36'/'IR64' were studied. 'CO39' is a highly susceptible cultivar (Mackill et al. 1988; Roumen 1992 a) that is assumed to carry few, if any, genes that hamper the formation of sporulating lesions (hereafter called lesions). Genetic analysis of PR of a cross between this cultivar and the resistant cultivar 'Moroberekan' by means of molecular techniques did not show any PR-enhancing factors in 'CO39' (Wang et al. 1994). The PR of 'IR36' is sufficiently high to prevent losses due to blast under irrigated conditions and has lasted even though 'IR36' has been grown on a wide scale for many years (Yeh and Bonman 1986; Bonman and Mackill 1988). The PR of 'IR64' resembles that of 'IR36'

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in field tests (Bonman et al. 1989; Sah and Bonman 1992). In the greenhouse, 'IR64' develops a slightly lower number of lesions than 'IR36', but their size is larger (Roumen 1992 a, b). This suggests that the PR in these cultivars is controlled by partly different genes.

Over 250 F_2 plants were grown per cross, all derived from the same F_1 plant. F_3 lines were grown from 50 F_2 plants taken at random. From 1 randomly selected F_3 plant per line, 5 F_4 plants were grown. The seed of 4 of these F_4 plants was harvested to produce 50 groups of 4 F_5 lines (hereafter called F_5 groups).

For the study of the inheritance, reserve seed of the 50 F_3 lines and freshly harvested seed of the 50 F_5 groups was sown together with seed of the parents and F_1. This was done in two trials per cross. Each of the 25 F_2 progenies per trial consisted of 10 F_3 plants and 10 plants of each of the 4 F_5 lines, which were grown in a completely randomized design together with 10 F_1 plants, and 20 plants of each parent. The trials of the cross 'IR36'/'IR64' included 20 'CO39' plants as a check. The plants were grown in a greenhouse in plastic trays ($30 \times 24 \times 10$ cm) in six rows of 5 plants. All generations were sown directly in the trays, except the F_1, which was sown in petri dishes and planted a few days after germination. Nitrogen fertilizer and water were applied as previously described (Roumen 1992 a). The plants were inoculated with the virulent isolate Po6-6 inside a plastic cage (2×2 m) when most plants had six leaves on the main culm. Per square meter of cage floor area, 400 ml of a conidia suspension with 5×10^4 conidia/ml in 0.5% gelatine was evenly sprayed over the plants as a fine mist shortly before sunset. The plants were kept in the cage at 100% humidity overnight and returned to the greenhouse the next morning. The number of lesions in each leaf on the main culm was counted 6 days after inoculation, and their total number was calculated per plant.

Preliminary analysis indicated that gene effects (number of lesions) were multiplicative on the natural scale. Therefore, genetic parameters were estimated after log transformation of the data using the function ln(x+1), with × being the number of lesions per plant relative to the mean number for 'CO39'. Genetic analysis was done assuming an additive-dominance genetic model. The midparent value (m), and the additive ([d]) and dominance ([h]) effects in each cross were estimated by weighted least squares analysis of the generation means across the two trials, using the reciprocal of the variance of the means as weight (Mather and Jinks 1982). For estimation of the narrow-sense heritability, a separate ANOVA was performed per trial for the F_3 and F_5 generations. The phenotypic variance components of the F_3 and F_5 were estimated from the equations of their expected mean squares (Table 1). In addition, the covariance between the F_3 lines and the corresponding F_5 groups was calculated. The expected contributions of the additive variance (D), the dominance variance (H) and the environmental variance (E) to these phenotypic variances and the covariance are listed in Table 2 (Mather and Jinks 1982). The environmental variance within each trial was estimated by solving the equations of the residual variances within the F_3 and F_5 lines. The estimates were substituted into the equations, and the additive variance was then estimated as the average of the solution for each equation ignoring dominance variance. Disregarding of the dominance variance is unlikely to bias the estimate of the additive variance much, because the parameters for H in most of the equations were much smaller than those for D (Table 2). No estimates were made for H. Heritabilities were calculated as the expected additive variance divided by the phenotypic variance.

The frequency of off spring with parental values occurring in the F_3 and F_5 generations was used for estimating the number of effective factors controlling the RIE in the cross 'IR36'/'CO39'. Assuming that all favourable alleles are in the more resistant parent ('IR36'), and with -k- factors segregating, the chance of finding at least 1 F_3 line, F_5 group or F_5 line with the genotype of the susceptible parent among 50 progenies is $1-(1-P)^{50}$, with P being the chance of recovering this genotype in a single F_3 line, F_5 group, or in at least 1 F_5 line within a single F_5 group. For an F_3 line, P is approximately $(¼)^k$, the chance that a random F_2 plant is like the susceptible parent. The additional chance that the susceptible genotype is recovered in all 10 F_3 plants when the F_2 plant is heterozygous, is negligible. Likewise, P is approximately $(⅜)^k$ for a F_5 group, i.e. the chance that the susceptible parent is recovered in a random F_3 plant. Finally, P is approximately

$$\sum_{s=0}^{k}\binom{k}{s}\cdot(3/8)^{k-s}\cdot(1/4)^{s}\cdot(1-(1-1/4^{s})^{n})$$

for the F_5 lines within a group, with s being the number of loci for which the F_3 plant was heterozygous and $n = 4$ since 4 F_4 plants were grown per F_3 plant.

Estimates for the number of segregating loci in both crosses were obtained from $(\Sigma d_a)^2/D$, the squared sum of the additive gene effects divided by the additive variance (Mather and Jinks 1982). For the cross 'IR36'/'CO39', the squared sum of the additive gene effects was estimated as $[d]^2$, and as the square of half the difference between the parents, i.e. $¼ \cdot (P_1-P_2)^2$. For the cross 'IR36'/'IR64', it was estimated as the square of half the range that was observed among the F_5 lines, $¼ \cdot (F_{5max}-F_{5min})^2$. Estimates were also obtained by genotype assay (Jinks and Towey 1976; Towey and Jinks 1977). The differences between the line means within the 50 F_3-derived F_5 groups were evaluated by t-tests based on the residual variance of each ANOVA for making an estimate of the proportion of heterozygous F_3 plants in each cross. The estimated proportion was then related to the number of effective factors using the equations for P_{max} and for P_{int}.A as proposed by Towey and Jinks (1977) and Mulitze and Baker (1985 a). The number of effective factors as estimated by these equations when 4 F_3-derived F_5 lines are evaluated is shown in Fig. 1.

Table 1 Degrees of freedom (*df*) and expected mean squares (EMS) for the ANOVAs of the F_3 and F_5 generations

Generation	Source	*df*	EMS[a]
F_3	Lines	24	$\sigma^2 F3_2+10\,\sigma^2 F3_1$
	Residual	225	$\sigma^2 F3_2$
F_5	Groups	24	$\sigma^2 F5_3+10\,\sigma^2 F5_2+40\,\sigma^2 F5_1$
	Lines within groups	75	$\sigma^2 F5_3+10\,\sigma^2 F5_2$
	Residual	900	$\sigma^2 F5_3$

[a] $\sigma^2 F3_1$, variance between means of F_3 lines; $\sigma^2 F3_2$, variance within F_3 lines; $\sigma^2 F5_1$, variance between F_5 groups; $\sigma^2 F5_2$, variance between means of F_5 lines within groups; $\sigma^2 F5_3$, variance within F_5 lines

Table 2 Expected contributions of the additive (D), dominance (H) and environment (E) variances to phenotypic variances and a covariance realized in the trials of two rice crosses assuming an additive-dominance genetic model

Variance component	Expected contributions of the additive, dominance and the environmental variances					
$10\,\sigma^2 F3_1$	=	5.25	D +	0.25	H +	E
$\sigma^2 F3_1$	=	0.25	D +	0.125	H +	E
$40\,\sigma^2 F5_1$	=	31.3125	D +	0.65625	H +	E
$10\,\sigma^2 F5_2$	=	1.3125	D +	0.1875	H +	E
$\sigma^2 F5_3$	=	0.0625	D +	0.03125	H +	E
COV (F3, F5)	=	0.5	D +	0.015625	H	

$\sigma^2 F3_1$, variance between means of F_3 lines; $\sigma^2 F3_2$, variance within F_3 lines; $\sigma^2 F5_1$, variance between means of F_5 groups; $\sigma^2 F5_2$, variance between means of F_5 lines within groups; $\sigma^2 F5_3$, variance within F_5 lines; COV(F3F5), covariance between means of F_3 lines and the corresponding F_5 groups

Results

Gene action

The number of lesions increased with the advance of generations in the cross 'IR36'/'IR64', indicating dominance

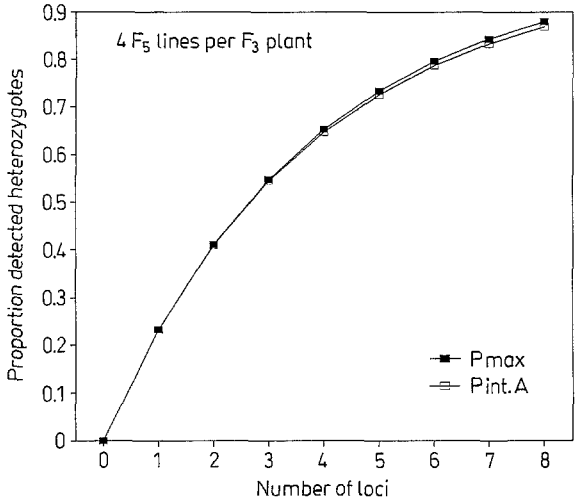

Fig. 1 The relation between the proportion of heterozygous F_3 plants and the number of segregating loci as estimated by P_{max} and $P_{int.}A$ (Towey and Jinks 1977) when four F_3 derived F_5 progenies are tested

Table 3 Relative number of lesions in the parents and in the F_1, F_3 and F_5 generations of the crosses 'IR36'/'CO39' and 'IR36/'IR64' (mean of two trials per cross)

	Cross IR36/CO39	Cross IR36/IR64
IR64	–	7.7
IR36	10.6	13.4
F_1	31.1	6.6
F_3	28.3	10.4
F_5	31.6	11.0
CO39	100.0	100.0

of resistance, but no increasing trend was observed in the cross 'IR36'/'CO39' (Table 3). In both crosses, the F_5 generation means of the untransformed data were close to the (midparent) value that was expected when gene action is multiplicative without epistasis (Table 3). The scaling tests of the log-transformed data indicated that the inheritance in both crosses was satisfactorily explained by additive and dominance effects; Chi-square values for models without epistatic interaction were low (Table 4). The pooled dominance effects were towards higher resistance. As expected from the generation means, gene action in the cross 'IR36'/'CO39' was mostly additive. The dominance effects in this cross were not significant. In the cross 'IR36'/'IR64', additive and dominance effects were both significant and about equal in magnitude (Table 4). A subsequent scaling test assuming complete dominance in this cross showed a very good fit of expected and realized generation means ($\chi^2_{3df} = 0.23$; $P \geq 0.995$).

Heritability

The ANOVAs for each trial are shown in Table 5. The phenotypic variance within F_5 lines was smaller than that within F_3 lines in both trials of each cross, whereas the variance between F_2-derived lines was larger in the F_5 than that in the F_3. The values for E, estimated from the residual variances of the F_3 and F_5, and the values for D, obtained after substituting the estimates for E in the equations of Table 2, are shown in Table 6. The heritabilities for individual plants in the F_2 calculated from these estimates were 0.08 and 0.12 for trials 1 and 2, respectively, of the cross 'IR36'/'CO39', and 0.16 and 0.06, respectively, for those of the cross 'IR36'/'IR64'. The corresponding heritabilities for F_3 lines were 0.47 and 0.58 in the cross 'IR36'/'CO39', and 0.68 and 0.41 in the cross 'IR36'/'IR64'.

Table 4 Estimates for the midparent value (m), and the pooled additive ([d]) and dominance ([h]) effects for two crosses. The estimates were calculated by joint scaling tests using log-transformed data and assuming just additive (model 1) or additive and dominant gene effects (model 2). The goodness of fit (χ^2; *df*) and the significance (*P*) of these models are included

	Cross IR36/CO39		Cross IR36/IR64	
	Model 1	Model 2	Model 1	Model 2
m	3.04	3.08	2.04	2.07
[d]	1.41	1.38	0.27	0.30
[h]		0.43		0.35
χ^2	1.93	1.60	2.61	0.18
df	3	2	3	2
P	0.50–0.75	0.25–0.50	0.25–0.50	0.90–0.95

Table 5 ANOVA summaries of the F_3 and F_5 generations after log transformation of the data for two trials of the crosses 'IR36'/'CO39' and 'IR36/'IR64' (*ns* not significant)

Cross	Source		*df*	MS	*F*	Significance
IR36/CO39 Trial 1	F_3	Line	24	3.70	2.256	$P \leq 0.01$
		Residual	224	1.64		
	F_5	Group	24	12.96	8.596	$P \leq 0.01$
		Line/Group	75	2.17	1.439	$P \leq 0.05$
		Residual	900	1.51		
IR36/CO39 Trial 2	F_3	Line	24	1.86	3.183	$P \leq 0.01$
		Residual	224	0.58		
	F_5	Group	24	5.14	9.584	$P \leq 0.01$
		Line/Group	75	0.66	1.241	ns
		Residual	894	0.54		
IR36/IR64 Trial 1	F_3	Line	24	2.61	4.173	$P \leq 0.01$
		Residual	224	0.62		
	F_5	Group	24	7.27	12.840	$P \leq 0.01$
		Line/Group	75	1.25	2.203	$P \leq 0.01$
		Residual	896	0.57		
IR36/IR64 Trial 2	F_3	Line	24	2.04	1.607	$P \leq 0.05$
		Residual	225	1.27		
	F_5	Group	24	6.64	5.960	$P \leq 0.01$
		Line/Group	75	1.57	1.408	$P \leq 0.05$
		Residual	897	1.11		

Table 6 Estimates for the additive (D) and the environmental (E) variance in two trials of two rice crosses

Variance component	Cross	
	IR36/CO39	IR36/IR64
Trial 1		
D	0.25	0.21
E	1.46	0.55
Trial 2		
D	0.14	0.14
E	0.52	1.06

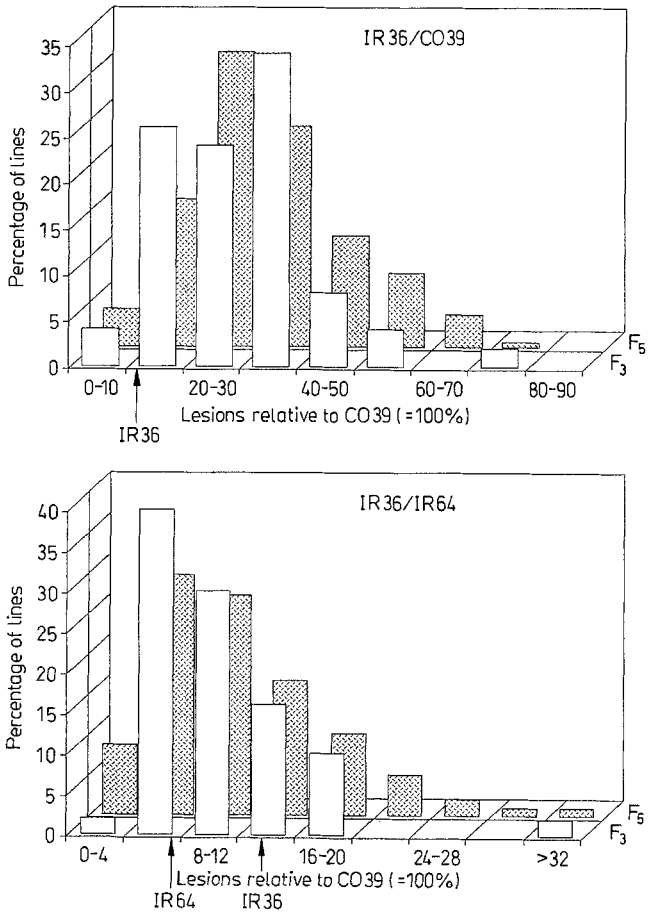

Fig. 2 Distribution of the average RIE of F_3 and F_5 lines derived from 50 random F_2 plants in the crosses 'IR36'/'CO39' and 'IR36'/'IR64'

Transgressive segregation

The distribution of the F_3 and F_5 line means of the cross 'IR36'/'CO39' was centred around the expected mid-parent value of 32.5% (= number of lesions relative to 'CO39'), and nearly all means were between those of the parents (Fig. 2). The means of the F_3 lines and those of the F_5 groups that were derived from the same F_2 plant were reasonably well correlated ($r = 0.62$). None of the progeny lines developed as many lesions as 'CO39'. A small percentage of the F_3 and F_5 lines developed fewer lesions than 'IR36' (Fig. 2), but except for 1 (0.5%) of the F_5 lines, the differences between these lines and 'IR36' were not significant (LSD; $P \leq 0.05$). Because no clear transgressive segregation occurred, all genes for a reduced RIE that segregated in this cross are apparently from 'IR36'.

Within the F_3 and the F_5 of the cross 'IR36'/'IR64', many lines developed either fewer lesions than 'IR64' or more than 'IR36' (Fig. 2). A lower number of lesions in the F_3 was again correlated with a lower number of lesions in the corresponding F_5 group derived from the same F_2 plant ($r = 0.61$). The percentage of lines with means outside the parental range was 76% for the F_3, and 71% for the F_5. Most of the differences between these lines and 'IR36' or 'IR64' were not significant. However, 2 (4%) of the F_3, and 12 (6%) of the F_5 line means were significantly lower than that of 'IR64'. In addition, 1 (2%) of the F_3, and 10 (5%) of the F_5 line means were significantly higher than that of 'IR36' (LSD; $P \leq 0.05$). Because a significant proportion of the F_5 lines showed significant transgressive segregation towards lower or higher resistance, 'IR36' and 'IR64' each have at least one gene controlling the RIE that is not present in the other cultivar.

Estimates for the number of effective genetic factors

Because the phenotype of 'CO39' was not recovered in any progeny line of the cross 'IR36'/'CO39', five or more independently segregating genes that reduce the RIE are probably present in 'IR36'. If four loci were segregating in this cross, the probability of retrieving at least 1 F_5 line with the phenotype of 'CO39' would be 0.97. On the basis of the value of 1.41 for [d] (Table 3) and the average value of 0.195 for D (Table 6), the minimum number of effective factors in 'IR36' was estimated to be 9 or 10; on the basis of the difference between the parents, the minimum number was 7 or 8. The genotype assay indicated that in the F_3, 40% of the plants were heterozygous, since 20 F_5 groups contained lines with significantly different means (LSD; $P \leq 0.05$). For this proportion, the number of effective factors was between 2 and 3 (Fig. 1).

The transgressive segregation that occurred in both directions in the cross 'IR36'/'IR64' rules out the possibility that all of the favourable alleles were concentrated in either one of the parents. No estimate for the number of segregating loci was thus calculated from [d] or from the difference between the parents and the additive variance. On the basis of the phenotypic range of the F_5 lines and the average additive variance of the two trials, the number of segregating loci was estimated to be 10 or 11. The percentage of F_3 plants in this cross that was declared heterozygous by genotype assay was 48%, and the estimate for the number of segregating loci in 'IR36'/'IR64' by this method was thus between 2 and 3 (Fig. 1).

Discussion

Because most of the gene action in the cross 'IR36'/'CO39' on the log-transformed scale appeared to be additive, the appearance of several progeny lines with a phenotype lower than or similar to that of 'IR36', and the absence of lines with a phenotype as high as that of 'CO39' was somewhat unexpected (Fig. 2). The discrepancy can be explained by the decreasing absolute gains that occur for each additional gene in a multiplicative acting genetic system. For a RIE of 'IR36' that is 10% that of 'CO39'and under the control of five genes with equal effects, and assuming no RIE-reducing genes in 'CO39', the presence of each of these genes is expected to increase the RIE by a factor 0.63. The expected RIE in a pure line from the cross 'IR36'/'CO39' with one gene is then 63% that of 'CO39' (37% difference to 'CO39'), and in a line with four genes, it is 16% that of 'CO39' (6% difference to 'IR36'). With an error of 6–10%, lines with four genes could develop a similar or even slightly lower number of lesions than the resistant parent, whereas lines with one gene would not develop as many lesions as 'CO39'.

The result that the inheritance of the RIE can be described by an additive-dominance genetic model is in agreement with those of Lin (1986) and Notteghem et al. (1981) who also investigated the inheritance of this component of PR to leaf blast. However, Wang et al. (1989) whose study included backcrosses, reported that some epistatic interaction among loci was likely. As in the present study, Lin (1986) and Wang et al. (1989) found the pooled dominance effects to be directed towards increased resistance, suggesting that most non-additive genes for a reduced RIE have a dominant expression. However, Notteghem et al. (1981) found that the number of lesions in the F_1 in a diallel of five cultivars was on average 41% more than that found in the parents and concluded that dominance and recessiveness of genes affecting the RIE occurred in about the same frequency.

The results of the present study indicate that PR to leaf blast is controlled by several genes. This is in agreement with the results from most other inheritance studies of PR to leaf blast (Higashi and Kushibuchi 1978; Higashi and Saito 1985; Wang et al. 1994). In the present study, the estimate of the number of segregating loci in the cross 'IR36'/'CO39' that is based on the proportion of offspring with a parental phenotype is considered the most reliable, because this estimate simply assumes the Mendelian segregation of genes and, unlike the other estimates, it is not sensitive to alterations of the scale (Mather and Jinks 1982). Since no clear transgressive segregation was observed in the cross 'IR36'/'CO39', all of the segregating loci were probably derived from 'IR36'. The conclusion drawn, therefore is that this cultivar contains at least five genes that reduce the RIE. No RIE-reducing genes were detected in 'CO39', which supports the results of Wang et al. (1994). However, the possibility that 'CO39' carries RIE-reducing genes cannot be entirely ruled out. One of the F_5 lines from the cross 'IR36'/'CO39' developed significantly fewer lesions than 'IR36' and, of course, 'CO39' and 'IR36' may have one or more RIE-reducing genes in common. The conclusion that 'IR36' contains at least five RIE reducing genes is supported by the estimate of the sum of the squared additive gene effects divided by the additive variance, but the estimate from the genotype assay method seems to indicate the opposite. Mulitze and Baker (1985 a, b) showed that estimates based on the genotype assay depend on the number of plants assessed and on the magnitudes of the type-I and type-II errors in the statistical tests. High type-I errors cause an upward bias of the estimate, whereas low heritabilities mean large type-II errors, which cause a downward bias of the estimate. For single plant-based heritabilities as low as those found in the present study, a considerable downward bias of the estimate by the genotype assay was expected, even though differences between line means were evaluated at a relatively high-type I error rate (Mulitze and Baker 1985 b). Evaluating differences among four line means by LSD tests at $\alpha = 0.05$ is equivalent to performing an F-test at $\alpha = 0.30$.

The transgressive segregation in the cross 'IR36'/'IR64' showed that 'IR64' and 'IR36' each have at least one gene that is not present in the other cultivar. However, it is very unlikely that the RIE of 'IR64' is controlled by just one gene. If so, this gene would have a very large effect (–92% relative to 'CO39'). On the assumption that 'IR64' without this hypothetical gene would have the 'CO39' phenotype, about one-half of the F_5 lines (lacking this gene) should have developed more lesions than 'IR36' and the distribution of these lines would be expected to show a peak near 32.5% of the number of lesions of 'CO39' (Fig. 2). Biometric estimates support the presence of at least as many segregating loci in the cross 'IR36'/'IR64' as in 'IR36'/'CO39'. This suggests that a considerable amount of genetic variation exists among cultivars with a good agronomic performance and that breeding for higher PR to leaf blast should thus be possible without having to sacrifice yield or other agronomically important traits.

The low narrow sense heritability estimates suggest selecting individual plants in the F_2 is unlikely to be efficient for improving PR. However, the combination of plant and line selection in early generations, such as selection of the best plants within the better F_3 lines, is expected to be reasonably efficient in inproving PR, since the narrow sense heritability of F_3 lines was reasonably high in both crosses. In addition, a reasonably high correlation between the F_3 line means and the F_5 group means derived from the same F_2 plant was observed in both crosses. Because the RIE is controlled by several genes, selection in early generations will be necessary for efficient accumulation of RIE reducing genes into new cultivars in breeding programmes.

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