

## COMBINING ABILITY ANALYSIS FOR RESISTANCE TO BANDED LEAF AND SHEATH BLIGHT OF MAIZE

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### ABSTRACT

Combining ability analysis of the reaction to banded leaf and sheath blight of maize caused by *Rhizoctonia solani* was carried out in 15 single crosses involving six inbred lines at two locations namely, Delhi and Pantnagar. Both general and specific combining ability variances significantly controlled disease reaction but general combining ability variance was predominant. Positive combining ability effects were associated with susceptibility, whereas negative effects conferred resistance. Inbred line CM104 was the most promising combiner for conferring resistance. Inbreds CM601 and CM 105, on the other hand, were combiners for susceptibility.

**Key words:** Resistance, banded leaf and sheath blight, *Rhizoctonia solani*, combining ability, diallel crosses.

Banded leaf and sheath blight caused by *Rhizoctonia solani* Kuhn f. sp. Sasakii Exner was recorded from India as a minor disease of maize by Ullstrup [1]. Warm temperature accompanied by high humidity has been found to be quite congenial for the epiphytotic development of the disease [2-4]. This disease has now become quite severe throughout the maize growing areas of the Indo-Gangetic plain, causing considerable reduction in yield. Development of resistant varieties appears to be the only alternative for controlling the disease. Since banded leaf and sheath blight disease has become a disease of concern in maize only recently, no work on breeding for resistance to this disease has yet been undertaken. However, a few resistance sources have been identified by artificial inoculation method [4-5]. This paper represents the first attempt at identifying inbred lines with respect to their combining ability characteristics for disease resistance so as to facilitate their utilization for developing resistant varieties, synthetics or hybrids.

### MATERIALS AND METHODS

Six inbred lines, namely, CM601, CM104, CM600, CM105, Aust. 25 and Eto 182 showing varying disease reaction from susceptible to resistant, were used in this study. These were crossed in diallel mating system to generate 15 all possible  $F_1$  crosses. Six parents and 15  $F_1$  crosses (excluding reciprocals) were grown in randomized block design with 4 replications at two locations: Delhi and Pantnagar. Delhi represented hot dry tropical conditions of the Indo-Gangetic plains, whereas Pantnagar represented hot

humid subtropical conditions of foot hills. Each plot consisted of two rows of 20 plants each, and the rows were spaced 75 cm apart. All individual plants were inoculated following the technique of Ahuja and Payak [5, 6]. Disease incidence was recorded after 45 days of inoculation based on 1-5 scale. Combining ability analysis was carried out according to Model 1, Method 2 of Griffing [8].

## RESULTS AND DISCUSSION

The analysis of variance for combining ability (Table 1) indicated that both general (gca) and specific combining ability (sca) variances were highly significant at both locations and also in the combined analysis. The magnitude of gca variance was considerably higher than the corresponding sca variance in all the three analyses, leading to the conclusion that gca or additive genetic component played a major role in controlling disease reaction. The combined analysis also indicated that both gca as well as sca variances interacted significantly with location. Such interaction for quantitative characters have been reported by several workers [9-11].

Table 1. Analysis of variance for combining ability for disease reaction

Source of variation	Mean sum of squares		
	Delhi	Pantnagar	combined
gca	1.180**	1.491**	2.392**
sca	0.192**	0.061**	0.190**
Location	—	—	0.729**
gca × location	—	—	0.041**
sca × location	—	—	0.056**
Error	0.004	0.008	0.006

\*\*Significant at 1% level.

The significant sca × location interaction is understandable because sca represents the nonadditive component of genetic variation, which is less stable over environment. The anomalous significant interaction of gca with location is not unusual and had been reported earlier in maize [12]. Since the gca variance includes additive genetic variance along with additive × additive type of epistasis, a greater proportion of such epistatic interaction might have given rise to the significant gca × location interaction. Earlier results of Matzinger et al. [9], in case of yield indicated that with relatively higher magnitude of gca variance there is a possibility of higher and significant interaction of gca with environment.

A comparison of mean disease reaction of parents and crosses at the two locations (Tables 2 and 3) leads us to the conclusion that the combining ability effects × environment interaction, though significant, has not made any disturbance in their overall mean performance at the phenotypic level.

Table 2 presents disease rating of the parents along with their gca effects. All the parents gave almost consistent disease reaction at both Delhi and Pantnagar. The combined analysis also indicated similar pattern of disease reaction. CM 104 was identified as the most resistant parent, whereas CM105 and CM601 were the most susceptible ones. A perusal of the combining ability effects indicated that the susceptible parents showed higher magnitude of positive gca effects, while the resistant parents had negative gca effects with the higher negative value conferring higher resistance. This fact could be inferred from the comparison of the actual disease rating with those of gca effects of individual parents. Overall results indicated that inbreds CM104 and Eto 182 can be considered as most promising for conferring resistance, whereas CM601 and CM105 were poor parents, since they conferred susceptibility, as indicated by their high positive gca effects.

The behaviour of CM105 in the present investigation is in conflict with earlier studies. This parent, found highly susceptible, was reported to be resistant under laboratory inoculation conditions [6]. The significant gca  $\times$  location interaction is not responsible for the conflicting results, since the actual field disease rating of this parent under artificial inoculation was highly consistent at both locations and in combined analysis. Presumably, the laboratory screening conditions [5, 6] failed to mimic the actual field screening conditions.

Table 2. Summary of general combining ability effects and mean disease rating of parental inbred lines

Inbred line	Delhi		Pantnagar		Combined	
	gca	rating	gca	rating	gca	rating
CM 601	0.067	3.46	0.016	3.28	0.020	3.37
CM 104	-0.303	2.48	-0.407	2.47	-0.337	2.48
CM 600	0.114	2.71	0.118	2.57	0.142	2.64
Aust. 25	-0.102	2.50	-0.124	2.60	-0.108	2.55
CM 105	0.406	4.07	0.590	4.91	0.436	4.49
Eto 182	-0.180	2.60	-0.175	2.56	-0.156	2.58
SE( $g_i - g_j$ )	0.033	—	0.045	—	0.039	—

Seven out of 15  $F_1$  crosses (Table 3) were identified as the most resistant specific combination. In general, crosses having at least one parent with negative gca effects and resistant reaction showed resistance. It was, however, discouraging to find that the  $F_1$  of the most resistant parents CM 104 and Eto 182 gave intermediate disease reaction. It may be assumed that these two parents possess higher frequency of genes with cancelling effects, leading to reduction in resistance. This may, however, be confirmed only by variance component analysis.

Considering the overall result, it may be suggested that parents CM104 and Eto 182, along with Aust. 25, may be used as sources for incorporation of resistance in otherwise agronomically superior inbred lines. These three parents can also be chain-crossed to

form a resistant pool and subjected to full-sib selection to concentrate the resistant genes as they have shown inter se better gca effects for resistance.

**Table 3. Summary of specific combining ability effects and mean disease rating of crosses**

Pedigree	Delhi		Pantnagar		Combined	
	sca	rating	sca	rating	sca	rating
CM 601 × CM 104	-0.115	2.38	-0.043	2.61	-0.097	2.49
CM 601 × CM 600	-0.212	2.47	0.163	3.11	0.005	2.79
CM 601 × Aust. 25	-0.396	2.30	-0.269	2.67	-0.335	2.48
CM 601 × CM 105	0.991	2.57	0.095	3.81	0.498	3.19
CM 601 × Eto 182	-0.068	2.55	-0.288	2.66	-0.166	2.60
CM 104 × CM 600	-0.188	2.26	-0.088	2.47	-0.109	2.36
CM 104 × Aust.25	-0.067	2.39	-0.147	2.40	-0.109	2.39
CM 104 × CM 105	-0.204	3.12	0.404	3.11	0.259	3.12
CM 104 × Eto 182	0.161	2.54	0.274	2.77	0.199	2.65
CM 600 × Aust.25	0.641	3.29	0.472	3.31	0.611	3.30
CM 600 × CM 105	0.326	3.84	0.400	3.84	0.100	3.84
CM 600 × Eto 182	-0.136	2.43	-0.045	2.74	-0.062	2.58
Aust. 25 × CM 105	0.157	3.68	0.008	3.80	0.018	3.74
Aust. 25 × Eto 182	-0.038	2.54	-0.119	2.58	-0.121	2.56
CM 105 × Eto 182	0.202	3.65	0.141	3.89	0.217	3.77
S.E.( $S_{ij} - S_{ik}$ )	0.089	—	0.120	—	0.102	—
S.E.( $_{ij} - S_{kl}$ )	0.082	—	0.110	—	0.095	—

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