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GENETIC ANALYSIS OF RESISTANCE TO *HELMINTHOSPORIUM TURCICUM* PASS. *[EXSEROHILUM TURCICUM* (PASS) LEONARD AND SUGGS] IN SOME VARIETIFS OF MAIZE

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Diallel crosses among eight open pollinated varieties of maize (*Zea mays* L.), including parents but excluding reciprocals when tested for resistance to *Helminthosporium turcicum* Pass. under natural epiphytotic conditions at Kalimpong (altitude of 1070 m) indicated that the disease resistance was mainly due to additive genetic variance. The varieties Kisan, Super I, (Syn. P 200 x Kisan), Comb. H_3 may be used as base population to develop disease resistant variety through reciprocal recurrent selection.

INTRODUCTION

Northern corn leaf blight caused by *Helminthosporium trucicum* Pass. (Syn *Exherohilum turcicum* (Pass.) Leonard) leads to severe leaf damage in cool and humid areas characterized by heavy dews, abdundunt rainfall and worm summer temperature inflicting losses in grain yield often as high as 50 percent (Chenulu and Hora, 1962 ; Jugenheimer, 1975).

The inheritance of resistance to this disease has extensively been studied in India by different workers (Jha and Dhawan, 1970 ; Rammurthy *et al.*, 1980 ; Das *et al.*, 1985 ; Das and Chaudhuri, 1985) but information on genetics of resistance to this disease is not sufficient under West Bengal Himalayan conditions. Hence this study was done on the inheritance of resistance to this disease in maize at this location.

MATERIALS AND METHODS

Diallel crosses among eight open pollinated varieties of maize, Super I, (Syn. P 200 x Kisan), Comb. H_3, Kisan, Vijay, Safeda, Diara, (BE x Ant. Gr. II) including parents but excludding reciprocals were made and hybrid seeds were sown in a randomized block design with replications having 30 plants each, at experimental station, Kalimpong, situated at an altitude of 1,070 meter in the

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Himalayan Hills in the district of Darjeeling, West Bengal. Usual cultural practices as adopted in general cultivation were followed.

Leaf blight caused by *Helminthosporium turcicum* Pass. appeared in epidemic from after silking of plants. The intensity of infection was recorded in the scale of 0 to 5 as adopted by Ullstrup *et al.* (1945).

The analysis II of Gardner and Eberhert (1966) for variety cross diallel was adopted to estimate general combining ability and various components of specific combining ability i. e. average heterosis, variety heterosis and specific heterosis. In the present case gca is equivalent to variety effect of Gardner and Eberhart (1966) and the components of heterosis combined together are equivalent to sca of Griffing's (1956) method 2 and model I.

RESULTS AND DISCUSSION

The general analysis of variance indicated that the mean resistance indices of the crosses including parents were significantly different and hence analysis of variance for combining ability was followed and the same is presented in Table 1. From Table 1 it is evident that both variety effect and heterosis are highly significant. Though the mean sum of squares for heterosis and its components were significant but their magnitudes were nearly 30 times smaller than the mean sum of sqnares of the variety effect.

Table 1. *Analysi of variance of variety cross diallel for* Helminthosporium turcicum *of Maize*

Source	d.f.	M S.
gca (gi)	7	6.25**
Heterosis (hij)	28	0.20**
Averag heterosis (h)	1	0.005*
Variety heterosis (hi)	7	0 233**
Specific heterosis (sij)	20	0.192**
Error	35	0.076

$\sigma^2 g = 0.91$ $\sigma^2 s = 0.06$

Among the components of heterosis, mean sum of squares for heterosis was the largest and that for average heterosis was the smallest, the mean sum of squares for specific heterosis being in between the two. The estimated variance for gca was 15 times greater than the estimated variance of sca (Table 1) indicating major role of additive genetic variance in controlling the resistance to this disease.

The mean scores of the disease and varietal gca, average, verietal and specific heterosis effects are presented in Table 2. It can be seen from this table that

Table 2. *Showing varietal mean for disease scores (upper diagonal), varietal effects (Vj) in parenthesis, varietal heterosis (hi), gca effects (gi), specific heterosis (sij') (in lower diagonal) and average heterosis (h) and respective standard errors.*

Parent	(Syn P200 x Kisan	(B.E. x Ant. Gr II	Kisan	Safeda	Super I	Diara	Vijay	Comb. H_3	Varietal hete-rosis effect	gca effect
(Syn. P200 x Kisan)	0.32 (-0.644)	1.55	1.23	0.75	0.40	1.00	1.45	1.04	0.46**	-0.041
(B.E. x Ant. Gr. III	0.25	1.13 (0.166)	0.68	1.03	1.56	0.80	0.79	1.47	0.14**	0.167**
Kisan	0.56**	-0.07	0.85 (-0.114)	0.52	0.20	0.42	0.30	0.76	-0.34**	-0.266**
Safeda	-0.20	0.01	0.14	2.24 (1.276)	0.23	1.40	1.02	0.80	-0.77**	0.176**
Super I	-0.48**	0.60**	-0.16	-0.38	0.30 (-0.664)	1.07	0.99	0.93	0.13**	-0.249**
Diara	-0.37	-0.64**	-0.39	-0.31	0.04	1.59 (-0.626)	1.53	2.05	-1.02	0.230
Vijay	0.42	-0.34	-0.19	0.27	0.30	0.34	0.80 (-0.164)	0.19	0,03	-0.060
Comb. H	-0.15	0.20	0.12	-0.12	0.08	0.70**	-0.84**	0.48 (-0.484)	0.35**	-0.027

Average heterosis (h)=0.91** S.E. (h)=0.10 S.E. (hi)=0.05 S.E. (hi-hj)=0.22 S.E. (Sij')=0.224 S.E. (Sij-Sik')=0.15
S.E. (Sij'-Skl')=0.30 S.E. (gi)=0.078 S.E. (gi-gj)=0.118

parental resistance did not exhibit any precise relationship with the gca effects for varietal heterosis. Kisan and Super I exhibited highest gca effects for resistance. The effects of varietal heterosis were also not related with gca effects of the parents. For example, Safeda exhibiting highest susceptibility, showed highest varietal heterosis for resistance. The cross between Comb. H_3 and Vijay has exhibited highest specific heterosis for resistance followed by the cross between (B. E. x Antigua Gr III) and Diara. Two best general combiners did not produce best specific combine and the same is true for two worst general combiners. Average heterosis being positive in sign indicated that susceptibility was dominant over resistance.

In view of the expressions of opposite types of genetic effects by parents and crosses, it is apparent that instead of re-inforcing of additive and dominance components towards expression of disease resistance, on hybridization these two types of genetic components tended either to cancel each others' effect or to complement to determine the genotypic expression of hybrids.

It is thus clear from the results that for evolving a synthetic variety resistant to *Turcicum* leaf blight, variety Kisan, Super I, (Sy. P 200 x Kisan), Comb. H_3 may be utilized as base population. Both recurrent selection for general combining ability and reciprocal recurrent selection for specific combining ability may be adopted taking above mentioned parents and promising crosses as base population. Additive gene control of the resistance to this disease was concluded by many workers (Jha and Dhawan, 1970 ; Ramamurthy *et. al* , 1980 ; Das *et. al*., 1985 ; Das and Chaudhuri, 1985). The present study also confirms the general findings of these authors that the resistance to *H. tnrcicum* is mainly due to additive genes.

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