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Short Communication

Genetics of resistance to charcoal rot in maize (*Zea mays* L.)

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Among the prevalent diseases of maize, post flowering stalk rot (PFSR) is a disease of economic significance in majority of the maize growing regions of the world. The disease complex is caused by a number of fungal pathogens, of which *Macrophomina phaseolina* (Tassi) G. Goid (charcoal rot), *Fusarium* spp. (fusarial stalk rot), *Cephalosporium maydis* Samra, Sabet and Hingorani (late wilt) and *Cephalosporium acremonium* Cda (black bundle) are of significance under Punjab conditions [1]. In the PFSR complex, percent incidence varied from 5 to over 40 percent in various locations [2]. Breeding for disease resistance is must for such a disease complex. The genetics of charcoal rot resistance was worked out under artificial epiphytotic conditions. Forty-five crosses developed through diallel crossing in ten inbred lines of maize (Table 1: five lines resistant and five lines susceptible) and inbred parents were evaluated in a randomized complete block design using three replications and two environments [winter 2001-02 (E_1) and spring 2002 (E_2)]. At flowering stage, 5 random plants were inoculated using tooth pick method of inoculation [3] in a row to create epiphytotics for charcoal rot. Moisture stress was created immediately after inoculation for 10 days to facilitate early establishment of infection. The infection was recorded on 1-9 rating scale as suggested by Payak and Sharma [4]. The Percent Disease Index (PDI) was calculated by using the formula suggested by Mc Kinney [5].

$$PDI = \frac{\text{Sum of all numerical ratings}}{\text{No. of plants observed} \times \text{maximum grade}} \times 100$$

Analysis of diallel cross given by Hayman [6] was followed.

The analysis of variance of diallel crosses showed significant differences among genotypes for reaction to disease. Diallel cross analysis was performed for various traits to get the over all picture and nature of genetic variance. For PDI, all the components of genetic variance *viz.*, D, H_1 and H_2 were significant in both the environments with predominance of additive genetic variance (D). Significant and positive estimates of F in E_1 indicated dominant genes control whereas non-significant value of F in E_2 indicated the prevalence of both dominant and recessive genes. This indicated that two sets of genes are operating i.e. one set of genes expressed in E_1 whereas the second set expressed in E_2. Non-significant mean level of dominance effect (h^2) in E_1 indicated ambidirectional nature of dominance but significant value in E_2 indicated unidirectional nature of dominance. The component $(H_1/D)^{1/2}$ was less than one, indicating partial dominance. The deviation of $H_2/4H_1$ from 0.25 in E_1 indicated asymmetrical distribution of increasing and decreasing alleles having positive and negative effects. In E_2 value was almost near to 0.25, which indicated

Table 1. Pedigree, origin and rating scale of the ten inbred lines of maize selected for the study

Parental inbred lines	Pedigree	Origin	Rating scale (1-9)
P_1	P_{24} (STE) C_2-29-BBBB-#-2-BBBBBB-B-B-1-1-2	Ldh. W 5084	2.06
P_2	CM123-(H 3191-1xb-2xb-1-2-1-1)	Ldh. W CM 123	5.81
P_3	EC-383380-8-1-xb-#b-1-1	Ldh. W 5223	1.93
P_4	Sw-93D-313-23-Pop 49-S_4-1-1-3-1-1-1-2-4-1-3-2-2	Ldh. W 5038	2.00
P_5	Ms Pool C_2IC_2-11-2-2-1-1-1-1-1-#-Fsb-1-1-#-#-#-1-4-2-1	Ldh. W. 5216	2.18
P_6	Pop 36 (STE) C_2-15-B-7-1-1-1-1-1-xb-1-1	Ldh. W 5228	5.86
P_7	CML 31 (POB 27 C_5 H_E 117-1-4-B-ff-#-#-#-x-1-1-2)	Ldh. W 114	6.06
P_8	J_{54} Mo_{17}-21-2-3-2-2-1-1-2-1-2-9-#b#b-xb-2-2-1	Ldh. W 1697	5.92
P_9	JCY_3-2-1-1-3	Ldh. W 5150	6.00
P_{10}	ACROSS 8931-1-3-1-1-xb-2-1	Ldh. W 5230	1.85

1 - resistant; 9 - susceptible

symmetrical distribution of genes. In E_1, the ratio of $(4\ DH_1)^{0.5} + F/4\ DH_1)^{0.5} - F$ was more than one indicating the symmetrical distribution of dominant and recessive alleles in the parent i.e. for every single recessive gene or gene group, there was one dominant gene or gene group whereas in E_2 the situation was reverse. Heritability of disease index was found to be very high in both the environments (Table 2). Non-significant value of t^2 in E_2 showed absence of epistasis whereas it was present in E_1.

Table 2. Estimates of components of variation for Percent Disease Index (PDI)

Parameters estimated	Environment I (E_1)	Environment II (E_2)
D	84.03**±4.08	74.80**±1.69
H_1	33.75**±8.68	12.72**±3.60
H_2	21.74**±7.38	11.58**±3.06
F	22.16*±9.41	−6.78±3.90
E	3.54**±1.23	5.14**±0.51
h^2	−1.04±4.94	4.70*±2.05
$(H_1/D)^{1/2}$	0.63	0.41
$H_2/4H_1$	0.16	0.23
$(4DH1)^{05} + F/(4DHI)^{05}-F$	1.53	-
h^2/H_2	-	0.41
Hn	80.45	83.73
r	−0.58	−0.36
b	0.66**±0.08	0.88**±0.07
t^2	9.57*	1.86

*,**Significant at 5 and 1 percent level, respectively;
- : Not calculated since non significant.

The results indicated the importance of additive as well as dominance components with additive gene action being more prevalent for resistance. Hence initially selection for this trait to improve the disease resistance will be effective. This may result in improving the disease resistance in the populations as well as in fixing these genes in the inbred lines. Populations exploit all kinds of variance i.e. additive as well as non-additive variance. Thus population improvement in addition to imparting durable resistance will also provide necessary diversity which is desirable in evolving disease resistant varieties. The results are in conformity with Anuradha *et al.* [7], Singh [8] and Pecina *et al.* [9] who reported the importance of both additive and non-additive gene action for resistance to charcoal rot.

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