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STUDY OF GENE EFFECTS FOR KARNAL BUNT (*NEOVOSSIA INDICA* MITRA) RESISTANCE IN BREADWHEAT (*TRITICUM AESTIVUM* L.)

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ABSTRACT

The gene effects for Karnal bunt were studied by deploying the parental, F_1, F_2, BC_1 and BC_2 generations in four crosses: HD 29 x HD 2009, HD 29 x WL 711, WL 6975 x HD 2009 and WL 6975 x WL 711 of common wheat. Varieties HD 29 and WL 6975 were resistant and WL 711 and HD 2009 susceptible. The resistance was dominant over susceptibility. The A, B, C scaling tests and weighted least square analysis of generation means indicated the presence of nonallelic interactions. The components (d), (h) and (j) were significant in all the crosses, whereas (i) was significant in cross WL 6975 x WL 711, and (l) in HD 29 x HD 2009 and HD 29 x WL 711. The correlation coefficients between (h) and (l) depicted duplicate type of gene action. The results are discussed.

Key words: Gene effects, Karnal bunt, breadwheat.

Karnal bunt, caused by *Neovossia indica*, has become a serious disease of wheat in some parts of the country in recent years, causing significant qualitative and quantitative losses to wheat [1]. Since the disease is soil, air and seed borne, only a limited success in its control can be achieved through fungicides [2]. Breeding of resistant varieties is a more effective method of combating this disease [3]. The genetic sources with resistance or low level of incidence of Karnal bunt have been identified. Resistance can be transferred from these genotypes to the high yielding wheat varieties only through a definite breeding programme. This would be possible if the nature of gene effects in the material is known. However, very scanty information is so far available on the inheritance of Karnal bunt [4]. The study reported here was, therefore, taken up to investigate the gene effects controlling Karnal bunt resistance deploying generation mean analysis.

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MATERIALS AND METHODS

The experimental material comprised six basic generations, viz., P_1, P_2, F_1, F_2, BC_1 and BC_2 of the crosses HD 29 x HD 2009, HD 29 x WL 711, WL 6975 x HD 2009, and WL 6975 x WL 711. HD 29 and WL 6975 are resistant to Karnal bunt, and HD 2009 and WL 711 are susceptible. The experimental material of these generations was grown in randomized block design with two replications, single-row plots of 1 m length, and 25 x 10 cm spacing. In each replication, the parents and F_1 were represented by two rows. Each Backcross was accommodated in five rows, whereas the F_2 were grown in ten lines.

Fresh inoculum suspensions from the actively growing cultures were made in winter. Two ml of inoculum suspension containing 10,000 sporidia per ml were injected into the boot preceding awn emergence with the help of hypodermic syringe between 4–6 P.M. Two ears from each plant and 10 plants from each row were inoculated. The mist spray of water with perfo-sprayer was carried out regularly thrice a day for 1 h at a time to maintain relative humidity above 80%. At maturity, the inoculated ear heads were harvested separately. The diseased seeds were categorized on the basis of severity of infection into four grades: 0.25, 0.50, 0.75 and 1.00. The number of grains in each infection group was multiplied with its numerical value and a general grade point was obtained. Further, the coefficient of infection was calculated by dividing the grade point with number of grains from the inoculated ears and multiplied by 100.

For statistical analysis, the data were transformed according to angular transformation. The A, B, C scaling tests were applied according to [5]. The components of generation means were computed following $F\alpha$ metrix by fitting models of increasing complexities following the weighted least squares analysis [6]. The correlation coefficients between (h) and (l) were also worked out.

RESULTS AND DISCUSSION

The varieties HD 29 and WL 6975 were resistant to Karnal bunt, whereas WL 711 and HD 2009 were susceptible. The F_1 mean infection was significantly less than the midparental value, indicating the predominance of resistance over susceptibility in all the four crosses (Table 1). In F_2, the mean infection was significantly less than in their respective F_1 in all the crosses except WL 6975 x HD 2009. The infection scores in BC_1 were less than those of F_1 and F_2 generations, which indicated the transfer of dominant resistance genes from P_1 (resistant parent), whereas in BC_2 it was higher than in F_1 and F_2, indicating the corresponding dilution of resistance genes by crossing with P_2, the susceptible parent.

The results of A, B, C scaling tests are presented in Table 2. In the cross HD 29 x HD 2009, B and C scaling tests were significant, whereas in the cross WL 6975 x HD 2009 only B scaling test was significant. All the three scaling tests were significant in the crosses HD

29 x WL 711 and WL 6975 x WL 711. This indicates the presence of nonallelic interactions in all the crosses.

Table 1. Mean coefficient of infection for Karnal bunt in crosses of wheat

Cross	Infection coefficient in different generations					
	P_1	P_2	F_1	F_2	BC_1	BC_2
HD 29 x HD 2009	0.16 ± 0.12	28.47 ± 1.53	7.92 ± 0.49	6.09 ± 0.41	4.30 ± 0.90	8.45 ± 0.95
HD 29 x WL 711	0.36 ± 0.17	32.11 ± 2.12	7.79 ± 0.66	6.53 ± 0.37	7.82 ± 1.44	12.76 ± 1.12
WL 6975 x HD 2009	0.10 ± 0.10	27.84 ± 0.69	8.65 ± 0.79	8.43 ± 0.58	5.42 ± 1.07	11.08 ± 1.23
WL 6975 x WL 711	0.65 ± 0.28	28.67 ± 1.94	8.72 ± 0.48	7.28 ± 0.44	5.59 ± 0.62	10.48 ± 0.59

The step-wise model fitting was done in all the 23 models and the model with the most significant parameters and minimum χ^2 value was selected (Table 3). The additive–dominance model was inadequate in all the crosses, indicating the presence of nonallelic interactions in this material, supporting the inferences drawn from the scaling tests. The components (d), (h), (j) and (l) were significant in the crosses HD 29 x HD 2009, and HD 29 x WL 711. The components (d), (h) and (j) were significant in cross WL 6975 x HD 2009 and (d), (h), (i) and (j) in WL 6975 x WL 711. The digenic epistatic model was inadequate in the cross HD 29 x WL 711, indicates the prevalence of higher order genic

Table 2. A, B, C scaling tests for Karnal bunt resistance in wheat

Cross	Values of scaling tests		
	A	B	C
HD 29 x HD 2009	2.03 ± 2.31	–15.09 ± 2.32**	–13.71 ± 2.35**
HD 29 x WL 711	9.83 ± 2.94**	–8.85 ± 2.32**	–13.48 ± 2.38**
WL 6975 x HD 2009	4.87 ± 2.56	–9.41 ± 2.58**	–3.08 ± 2.81
WL 6975 x WL 711	4.19 ± 1.69*	–9.96 ± 1.89**	–9.75 ± 2.43**

*$P < 0.05$, **$P < 0.01$.

interactions for the expression of this trait. Taking all the four crosses together, it may be noted that the additive component (d) was significant, therefore, the genes controlling Karnal bunt resistance may be dispersed in the parents. The dominance component (h) was negative and significant in all the crosses, indicating the dominance of resistance over susceptibility. The absence of additive x additive (i) epistasis in three of the four crosses

Table 3. Estimates of gene effects for Karnal bunt resistance in four crosses of wheat

Cross	Gene effects						
	(m)	(d)	(h)	(i)	(j)	(l)	χ^2
HD 29 x HD 2009	19.69 ± 0.53**	13.02 ± 0.53**	-15.83 ± 2.26**	—	17.09 ± 3.16**	13.55 ± 2.19**	0.03
HD 29 x WL 711	20.78 ± 0.57**	14.21 ± 0.57**	-14.83 ± 2.34**	—	14.34 ± 3.44**	10.94 ± 2.28**	14.60**
WL 6975 x HD 2009	18.61 ± 0.45**	12.67 ± 0.46**	-1.69 ± 0.87*	—	14.02 ± 3.44**	—	1.97
WL 6975 x WL 711	14.69 ± 0.98**	12.48 ± 0.66**	-3.29 ± 1.28*	4.90 ± 1.21**	12.12 ± 2.45**	—	0.14

*$P < 0.05$, **$P < 0.01$.

indicated relatively less importance of the fixable type of epistasis. The parameters (h), and (l), on which the classification of epistasis depends, were predominantly of different signs. The correlation coefficients between (h) and (l) components were worked out in m, (d), (h), (l); m, (d), (h), (i), (l) and m, (d), (h), (j), (l) models (Table 4). In all the crosses, the parameters (h) and (l) depicted highly negative correlations in all the models, indicating the role of duplicate epistasis for the control of Karnal bunt resistance. The use of extreme parents may have resulted into this situation.

Table 4. Correlation coefficients between (h) and (l) parameters for Karnal bunt

Cross		(h)	(l)	χ^2	r
HD 29 x HD 2009	1	-14.05**	12.48**	**	-0.90
	2	-15.89*	13.65**	**	-0.92
	3	-15.82**	13.55**	NS	-0.94
HD 29 x WL 711	1	-13.88**	10.62*	**	-0.90
	2	9.88	-4.68	**	-0.89
	3	-14.83**	10.95**	**	-0.88
WL 6975 x HD 2009	1	-3.40	2.74	**	-0.85
	2	-9.77	6.77	**	-0.82
	3	-4.66	3.49	NS	-0.81
WL 6975 x WL 711	1	-6.32**	5.90**	**	-0.92
	2	2.03	0.80	**	-0.83
	3	-9.66**	8.02**	NS	-0.94

Note. 1, 2 and 3 stand for m, (d), (h), (l); m, (d), (h), (i), (l); and m, (d) (h), (j), (l) models, respectively.
*$P < 0.05$, **$P < 0.01$.

Under a situation of this type, it would be difficult for a breeder to get genotypes with desired resistance through conventional breeding methods. By following the breeding procedures of multiple crosses, growing large sized populations in each generation, and attempting biparental matings in early segregating generations may be useful in generating transgressive segregates.

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