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TRIPLE TEST-CROSS ANALYSIS OF KARNAL BUNT RESISTANCE IN WHEAT (*TRITICUM AESTIVUM* L.)

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ABSTRACT

Genetics of Karnal bunt resistance was investigated using triple test-cross analysis in bread wheat. Twenty nine diverse genotypes were crossed with HD 29 (resistant) and WL 711 (susceptible), true-breeding testers, and their F_1 hybrid. The epistatic variation was found to be an integral part of inheritance of Karnal bunt resistance. Both additive (D) and dominance (H) genetic components were significant with partial degree of dominance. Due to the predominance of additive variance, breeding for Karnal bunt resistance may be successful with simple selection procedures.

Key words: *Triticum aestivum*, wheat, triple test-cross, Karnal bunt resistance, *Neovossia indica*.

Karnal bunt caused by *Neovossia indica* is a serious disease in different wheat growing areas of the world. Besides yield losses, it causes deterioration of grain and flour quality. Karnal bunt infection even as low as 5% renders grains unfit for human consumption [1]. There is little information on genetics of Karnal bunt resistance. Genetic analysis based on reaction of aneuploids [2, 3] has shown that resistance to Karnal bunt is a quantitative trait and controlled by many major and minor genes distributed over various homoeologous groups of chromosomes. The present study has been undertaken to analyse the inheritance pattern of Karnal bunt resistance through the triple test-cross analysis which allows for the independent testing of additive, dominance and epistatic genetic variations.

MATERIALS AND METHODS

Two true-breeding testers, HD 29 (resistant) and WL 711 (susceptible) were selected on the basis of phenotypically extreme expression of Karnal bunt. Over the past several years, HD 29 and WL 711 have shown highly resistant and susceptible reaction to Karnal bunt, respectively [4, 5]. These two true-breeding testers and their F_1 hybrids were crossed individually with 29 diverse cultivars of wheat (WG 357, WL 2265, Sonalika, Kalyan Sona, PBW 65, PBW 120, PBW 138, PBW 154, IWP 72, UP 368, HD 2009, HD 2204, HD 2329, CPAN 1676, CPAN 1922, CPAN 3013, WH 147, WH 157, Inia 66, Taluca 73, TZPP, Veery 5, HW

547, VL 616, NI 8188, Raj 3077, Chris, Crim and Yecora 70) to produce L_{1i}, L_{2i} and L_{3i} families, respectively. The experimental material comprising 2 testers, F_1 and 29 L_{1i}, L_{2i} and L_{3i} families was raised in randomized block design with three replications during the winter season of 1990 at the Punjab Agricultural University, Regional Research Station, Gurdaspur. Each family was assigned a single-row plot of 2.5 m length spaced at 30 cm with the plants spaced at 15 cm. Two ears from each of the ten plants tagged in a plot were inoculated with 2 ml of Karnal bunt inoculum suspension containing 10,000 sporidia per ml of water. The inoculations were done at the boot stage with the help of hypodermic syringe following the procedure of Aujla et al. [6]. Perfosprayer was used regularly; thrice a day for a 1 h at a time to maintain high humidity. At maturity, the inoculated ears were harvested and thrashed individually. The overall infection percentage (number of infected grains divided by the total number of grains harvested) was calculated for each progeny. The data on Karnal bunt infection were subjected to angular transformation for triple test-cross analysis as per Jinks et al. [7]. The variance of the comparison ($\bar{L}_{1i} + \bar{L}_{2i} - 2\bar{L}_{3i}$) was used to test the presence of epistasis, where $\bar{L}_{1i}$, $\bar{L}_{2i}$ and $\bar{L}_{3i}$ are the means of the ith family in respect of the tester concerned. The variances of sums ($\bar{L}_{1i} + \bar{L}_{2i}$) and differences ($\bar{L}_{1i} - \bar{L}_{2i}$) were used to detect the presence of additive (D) and dominance (H) components of genetical variation.

RESULTS AND DISCUSSION

The mean square for the comparison ($\bar{L}_{1i} + \bar{L}_{2i} - \bar{L}_{3i}$) detecting the presence of epistasis was found significant (Table 1). Although significant epistasis was detected in the present study the additive (D) and dominance (H) components were nevertheless computed in order to assess their relative contributions in the inheritance of Karnal bunt disease. Both additive and dominance component were significant and the former had a higher magnitude (D = 65.31, H = 28.03) than the latter which resulted into partial average degree of dominance ($\sqrt{H/D}$ = 0.66) for susceptibility of the genotypes to the disease. Further, the correlation (r = 0.34) between sums and differences revealed an ambidirectional dominance of both increases and decreases. Gill et al. [8] have also reported additive, dominance and epistatic gene effects to be significant for Karnal bunt resistance in a study on generation means analysis of four breadwheat crosses.

Table 1. Analysis of variance for triple test-cross between Karnal bunt resistant and susceptible cultivars

Parameter	Source	d.f.	M.S.	F value
Epistasis				
($\bar{L}_{1i} + \bar{L}_{2i} - 2\bar{L}_{3i}$	Epistasis	29	9.2	2.68**
	Epistasis x replicate	58	3.4	
Additive				
($\bar{L}_{1i} + \bar{L}_{2i}$)	Additive	29	60.7	5.16*
	Additive x replicate	58	11.8	
Dominance				
($\bar{L}_{1i} - \bar{L}_{2i}$)	Dominance	29	33.7	2.66**
	Dominance x replicate	58	12.7	

*,**Significant at 5% and 1% levels, respectively.

As the epistasis was conspicuously present in the material, the estimates of D, H and degree of dominance may

be biased to an unknown extent. However, the magnitude of additive genetic components (D) was higher than the dominance component (H) in the occurrence of Karnal bunt disease. Apparently, sufficient additive variation exists for selection of the resistant genotypes in the material and simple selection procedure like single seed descent will be effective in developing Karnal bunt resistant cultivars. Further, progress can be enhanced if more number of Karnal bunt resistant parents are involved in multiple crosses and large populations are sampled in each filial generation. Further improvement of such populations through recurrent selection may lead to the development of lines with increased Karnal bunt resistance.

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