Indian Journal of Agricultural Sciences **80** (11): 952–5, November 2010

Genetic analysis of resistance to Karnal bunt caused by *Neovossia indica* in bread wheat (*Triticum aestivum*)

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Received: 20 March 2009; Revised accepted: 10 August 2010

ABSTRACT

The genetics of Karnal bunt resistance was studied in populations derived from crosses involving resistant stocks ('HD 29' and 'WL 6975') and 1 highly susceptible cultivar ('WL 711'). The plant material screened for Karnal bunt reaction consisted of F_1, F_2, BC_1 and BC_2 populations from resistant × susceptible and resistant × resistant crosses. The data recording for Karnal bunt incidence was done under optimal conditions for disease development with a mixture of virulent isolates prevailing in the northern states, Punjab, Haryana, Uttaranchal, Delhi, Himachal Pradesh and western Uttar Pradesh. The generation means analysis and scaling test was performed. In all the 3 cross combinations, almost all the scaling tests were found significant which indicated the presence of all 3 type of interactions. Six parameter model indicated the additive as well as dominance effects of genes significant in crosses obtained involving reaction types of resistant × susceptible ('HD 29' × 'WL 711') and resistant × resistant ('HD 29' × 'WL 6975') while additive, dominance and additive × additive; dominance × dominance effects were found significant only in resistant × resistant cross ('WL 6975' × 'WL 711').

Key words: Bread wheat, Gene action, Generation means analysis, Karnal bunt, *Neovossia indica*, Resistance, *Triticum aestivum*

Karnal bunt (KB) of wheat caused by *Neovossia indica* (Mitra) Mundkur (Syn. *Tilletia indica*) was first reported from India by Mitra in 1931. It continued to be a minor disease till 1968, however, in 70s and 80s of 20th century, it emerged as an important disease of wheat (*Triticum aestivum* L. emend. Fiori & Paol.) in the country. Subsequently the occurrence of Karnal bunt has also been reported from Pakistan, Nepal, Afghanistan, Iran, Iraq, South Africa, Mexico and USA. The Karnal bunt pathogen causes infection on the floral parts and infected portions of the kernel are replaced with masses of fungal teliospores. Infection varies from traces to invasion of the entire kernel. Although yield losses are generally low but wheat grains with more than 3% infection are considered unsatisfactory for human consumption. It has also serious implications for the international trade of commercial grain and exchange of wheat germplasm. Blanket embargoes are in place in many countries on wheat import from Karnal bunt regions. Conventional methods for Karnal bunt control include cultural practices, viz crop rotation, sowing of disease free seeds and adjustment of the sowing time. Chemical control of the disease with fungicides applied near flowering stage is only partially effective owing to the varied modes of spore transmission. It is commercially impractical because of the zero Karnal bunt tolerance level for Karnal bunt spores imposed by importing countries. Most of the fungicides tested against teliospores are fungistatic. At this juncture, the development of resistant varieties is the only economic and effective method of Karnal bunt disease control. Not much work has been done on genetics of Karnal bunt resistance, as studies on this aspect are comparatively tedious due to labour intensive screening methods. The absence of well-known pathogen isolates and heterothallic nature of pathogen also hamper precise genetic analysis. In the present study, an effort has been made to study the genetics of resistance by involving 2 highly resistant parents ('WL 6975' and 'HD 29') and 1 susceptible cultivar ('WL 711') in different cross combinations.

MATERIALS AND METHODS

The 2 Karnal bunt resistant stocks ('HD 29' and 'WL 6975') were crossed with 1 susceptible parent ('WL 711') to obtain 2 resistant × susceptible cross combinations and at the same time the 2 resistant parents ('HD 29' and 'WL

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6975') were also intercrossed for studying the genetics of Karnal bunt resistance. The F_1 seeds of 3 crosses, namely 'HD 29' × 'WL 711' (resistant × susceptible), 'HD 29' × 'WL 6975' (resistant × resistant) and 'WL 6975' × 'WL 711' (resistant × susceptible) were raised at Directorate of Wheat Research, Karnal during 2004–05 crop season (November to April) to advance the material to F_2 generation. Simultaneously the backcrosses (BC_1 and BC_2) were attempted to produce first back cross generation during summer (May to September) of 2005 at Directorate of Wheat Research (DWR), Regional Research Station, Dalang Maidan (Lahaul-Spiti) situated around 10000 feet above mean sea level. To have enough F_1 seed for genetic analysis, fresh crosses between the parents were also attempted at Dalang Maidan. Thus, the complete set of experimental material finally composed of 6 generations, viz P_1, P_2, F_1, F_2, BC_1 (backcrossed to first parent, P_1) and BC_2 (backcrossed to second parent, P_2), was planted in winter (*rabi*) season of 2005–06 at Directorate of Wheat Research, Karnal in a randomized block design with 3 replications. The variable plot size for parents, F_1s, F_2s and backcrosses (BC_1 and BC_2) were used by keeping fixed row length of 3 m. The plot size for each of 3 parents was paired rows, while 15 rows were assigned to each F_2 and BC_1 and BC_2 generations. The row-to-row and plant-to-plant distances were kept at 23 cm and 10 cm, respectively. The recommended agronomic practices including doses of fertilizers, irrigations and weed control were followed to grow good crop. When plants attained the physiological growth stage 49 (Zadoks *et al*. 1974), microsporidia harvested from representative cultures of protein profile types were inoculated into the boot of the spikes following Aujla *et al.* (1987) and Kumar *et al.* (2009). By mid April, the fully matured spikes were harvested and threshed separately by taking utmost care. Grains from each spike were utilized for scoring Karnal bunt disease intensity and per cent coefficient of infection was calculated following Aujla *et al.* (1989). The 6 generations data were subjected to the joint scaling test (Cavalli 1952), generation mean analysis and perfect fit solution (Mather and Jinks 1971) were used to estimate the gene effects for inheritance studies of Karnal bunt disease in bread wheat.

RESULTS AND DISCUSSION

The generation means analysis and scaling tests were performed and the results are presented in Table 1. In all the 3 cross combinations, almost all the scaling tests were found significant which indicated the presence of all 3 (additive, dominance and epistatic) types of gene effects. Significance of A and B scaling tests will indicate presence of j (additive × dominance) type of epistasis, whereas significance of C and D scaling tests will indicate presence of l (dominance/dominance) and I (additive × additive) component of epistasis. The estimates of different types of gene effects and their interactions in the individual cross combination illustrated the variation. Mean values and their corresponding standard errors for Karnal bunt infection, ie per cent coefficient of infection in different generations of the 3 crosses are presented in Table 2. Differences between crosses were observed for Karnal bunt incidence calculated as per cent coefficient of infection. In the F_1s of all the 3 crosses, the differences were observed for per cent coefficient of infection of Karnal Bunt in comparison to their corresponding parental values. In F_1s of both the resistant × susceptible crosses ('HD 29' × 'WL 711' and 'WL 6975' × 'WL 711'), the per cent coefficient of infection of KB were neither similar to resistant, susceptible parents nor to mid-parental value. It also did not show transgression. For the resistant × resistant cross ('HD 29' × 'W 6975'), per cent coefficient of infection in F_1 was slightly higher (1.07) than the corresponding better parent (0.91). An increase in the mean value of F_2 population, as compared with that of corresponding F_1 was observed in all three crosses. However, the per cent coefficients of infection values were still lower than that of its corresponding susceptible parents and higher than the resistant parent.

Table 1 Values of scaling tests (A, B, C and D) for per cent coefficient infection of Karnal bunt in 3 cross combinations of wheat

Scales	Scaling tests		
	'HD 29'/ 'WL 711'	'WL 6975'/ 'WL 711'	'HD 29'/ 'WL 6975'
A	23.77±3.90**	–1.1635±2.83	–1.14±2.12
B	37.63±8.05**	15.6213±8.04**	19. 13±3.04**
C	58.62±8.61**	50.6487±9.41**	45.87±5.02**
D	–1.39±5.65	18.0954±5.58**	8.09±2.87**

*, ** P=0.05, P=0.01
Scaling tests according to Mather (1949)

The results obtained on gene interaction for Karnal bunt revealed that based on the 6 parameter model, all the 3 cross combinations showed predominance of additive and dominance × dominance types of gene effects for conferring resistance against Karnal bunt in wheat (Table 3). One of the crosses between resistant × susceptible ('WL 6975'×'WL

Table 2 Generation means and their standard errors (X, ±) for per cent coefficient of infection of Karnal bunt in 3 crosses

Generation	'HD 29'/ 'WL 711'	'WL 6975'/ 'WL 711'	'HD 29'/ 'WL 6975'
P_1	00.91 ±0.16	1.92±0.37	0.91±0.16
P_2	36.88 ±1.77	36.88±1.77	1.92±0.37
F_1	05.76 ±1.84	5.36±2.23	1.07±0.58
F_2	15.22 ±1.72	25.04±2.02	1.65±0.24
BC_1	00.93 ±0.19	3.05±0.85	1.23±0.34
BC_2	40.14 ±3.82	28.93±3.76	0.93±0.19

Table 3 Estimates of different types of gene effects for Karnal bunt resistance in 3 crosses involving susceptible and resistant varieties of wheat

Parameter	'HD 29'/ 'WL 711'	'WL 6975'/ 'WL 711'	'HD 29'/ 'WL 6975'
m	0.27±0.19**	0.25±0.20**	0.16±0.23**
d	–0.25±0.42**	–0.26±0.38**	–0.30±0.08**
h	–0.10±0.12	–0.50±0.11**	–0.25±0.13
i	0.28±0.11	–0.36±0.11**	–0.23±0.22
j	–0.69±0.43	–0.83±0.39*	–0.86±0.43
l	–0.64±0.18**	–0.21±0.08**	–0.28±0.02**

*, ** see above.

711') parents also showed the predominance of dominance and additive×additive type of gene effects. It was noticed that Karnal bunt percent infection was lowest in resistant×resistant type of cross that revealed the involvement of more than one genes and their additive or dominant nature of interaction. The two resistant×susceptible crosses have the same susceptible parent ('WL 711') in common yet, the incidence of Karnal bunt in F_2s and BC_2s varied relative to their corresponding parents, which indicates that the degree of dominance of the genetic control is different between the progenies of the 2 crosses and there exists a partially dominant nature of the resistance. It can also be hypothesized that gene(s) controlling Karnal bunt resistance in 2 resistant parents are not necessarily the same. This was confirmed by a higher per cent coeficient of infection in F1s than in the 2 resistant parents in cross 'HD 29'×'WL 6975'. Susceptible plants were observed in resistant×resistant cross indicating the distinctness of resistant genes among parents thereby supporting the hypothesis that KB resistance is controlled by multiples genes.

Singh *et al.* (2009) also reported that both additive and dominance gene effects from a diallel set involving parents, 'WL 711', 'HD 2009' (Karnal bunt susceptible) and 'WL 2217', 'UP 1008', 'WL 1562', Sonalika, 'VL 421', 'HB 208', 'TZPP', 'WG 2038' (Karnal bunt resistant). Predominant role of additive gene effects was shown in the inheritance of Karnal Bunt resistance (Sharma *et al*. 2005). While, Jag Shoran *et al*. (2009) reported 3 independently segregating loci with partial dominance. Besides Sharma *et al.* (2005) reported 9 loci governing Karnal bunt resistance in wheat. Other previous genetic studies on Karnal bunt resistance in wheat have indicated that one to nine major genes controlling resistance to Karnal bunt in various wheat germplasm have been identified as influencing reaction to the pathogen (Villareal *et al.* 1995). Singh *et al.* (1995) reported results based on analysis of 6 basic generations of intervarietal crosses between 3 resistant ('HD 29', 'W 485' and 'HP 1531') and 2 susceptible ('WL 711', 'HD 2329') parents that had shown the involvement of 1–2 major genes along with some minor genes/modifiers imparting resistance. In the widely studied cross, 'HD 29'×'WL 711', resistance has been shown to be controlled by a single recessive gene by Bag *et al.* (1999). In another study done by Sharma *et al.* (2001), the additive gene action was observed to be more important in the genetic control of Karnal bunt per cent infection whereas the dominant gene action was pronounced for coefficient of infection. In some earlier studies, 6 wheat chromosomes (1D, 2A, 3B, 3D, 5D and 7A) have been identified carrying Karnal bunt resistant genes (Singh *et al.* 2009).

From the present study and the results reported by earlier researchers, it could be concluded that resistance to Karnal bunt in wheat is primarily governed by a complement of more than 2 dominant genes along with some minor genes. Among genetic interactions, predominantly the dominance type of gene effects governed the Karnal bunt resistance, while additive, additive × additive and dominance × dominance type of gene interactions also played a partial role in conferring resistance against Karnal bunt. This type of varying results reported by various researchers could be due the fact that differential response of pathogen with host leads to variable levels of resistance and to some extent the type of gene combinations in resistant and susceptible parents used by them. Such variable results suggest that there is need to initiate efforts for identifying pathogen-specific gene(s) and their mode of interaction. The challenge for plant breeders is now to use the genes from resistant stocks as single Karnal bunt resistance gene only confers the incomplete resistance and at the same it is not possible to monitor these genes in the segregating generations. The approach for pyramiding of Karnal bunt resistance genes using molecular markers will facilitate the introgression of diverse genes needed for the development of wheat varieties resistant to Karnal bunt.

ACKNOWLEDGEMENT

The authors are grateful to the Indian Council of Agricultural Research, New Delhi for providing financial help.

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