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INHERITANCE OF RESISTANCE TO WHITE RUST DISEASE IN INDIAN MUSTARD

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ABSTRACT

A complete diallel set of crosses was produced from ten diverse parents of varying degrees of resistance and susceptibility to white rust and assessed to study the inheritance of the disease caused by *Albugo candida*. Data from parents and their F_1s including reciprocals were analysed using Griffing and Jinks- Hayman methods of diallel analysis. The experiments confirmed from the analysis of both the approaches that additive gene action predominantly controlled the inheritance of white rust disease. The parent Poorbijaya was the best general combiner followed by BJ-38. The cross Glossy mutant × BJ-38 was found to be the best specific combination. All the crosses showing superior sca effects could be utilised for getting transgressive segregates with better resistance to white rust in mustard. The mean degree of dominance was partial. Heritability estimates in narrow and broad senses were high. Predominance of additive genetic variance coupled with high heritability suggested that simple selection procedures such as pedigree breeding would be useful for improving the level of resistance.

Key Words : *Brassica juncea, Albugo candida,* diallel analysis, combining ability, degree of dominance, transgressive segragate

White rust is an important disease of oilseed brassicas in India and Canada. The disease caused by *Albugo candida* affects primarily rapeseed (*Brassica campestris*) and Indian mustard [*Brassica juncea* (L.) Czern & Cross]. Its race 2 mainly infects *B. juncea*[1], the most dominant oilseed *brassica* crop in India. Though some studies showed that resistance to white rust was controlled by a few major genes [2-3], the resistance to this disease was also reported to be quantitatively inherited conditioned by minor genes[4]. In *B. juncea,* though several sources of resistance have been described [5], information on the incorporation of resistance to agronomically superior cultivars has not been reported. This could be attributed to the scanty information on the nature of inheritance for this disease. In order to understand and verify the

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nature of inheritance of white rust and also to formulate an efficient breeding programme for improvement in the level of resistance, the present investigation was planned.

MATERIALS AND METHODS

Ten diverse genotypes of Indian mustard namely Pusa Bold, Varuna, Pusa Bahar, Pusa Barani, RH-30, PR-1108, Poorbijaya, Glossy mutant, BJ-17 and BJ-38 of varying degrees of resistance and susceptibility were crossed in all possible combinations including reciprocals. Forty-five F_1s, 45 reciprocal F_1s and their 10 parents were raised in a randomised complete block design with 3 replications. Each entry was sown in 2 rows of 5m length and was spaced at 45 cm row to row and 15 cm plant to plant. The recommended agronomical practices were followed for raising the crop.

The data for white rust infection on leaves were recorded in percentage on sampled plants of every genotype in each replication after two weeks of flowering. Six leaves were taken in each of the plant from lower position. The severity of the disease was recorded by using the scale as per [6]. The per cent infection index was calculated by the formula given by Singh [7] which covered a wide range and was subjected to angular transformation following Fisher and Yates [8].

The data were statistically analysed for combining ability using Griffing's method 1, model I [9]. For the estimation of second degree genetic parameters the Jinks-Hayman approach of diallel cross analysis [10-11] was followed. Heritabilities in narrow sense and broad sense were estimated from the genetic components as suggested by Mathur and Jinks [12].

RESULTS AND DISCUSSION

The mean percent infection index of the genotypes is presented in Table 1. The analysis of variance showed significant variation among parents and crosses (Table 2). The disease infection in parents ranging from 2.00 to 60.67 showed that the parent Poorbijaya had the highest degree of resistance followed by BJ-38. On the other hand, the parent PR-1108 exhibited highest degree of susceptibility. The hybrids between resistant and susceptible parents recorded a range of intermediate disease reaction suggesting polygenic nature of inheritance of the disease. Dominance of white rust resistance over its susceptibility was indicated by the lower mean values of the infection index in majority of the crosses including reciprocals than their mid-parental values.

Tbale 1. Mean white rust reaction of ten parents (diagonal), their F_1s (above diagonal) and reciprocals (below diagonal) in 10 × 10 diallel cross of *Brassica juncea.*

Parents	Pusa Bold	Varuna	Pusa Bahar	Pusa Barani	Rh-30	PR-1108	Poor-bijaya	Glossy mutant	BJ-17	BJ-38
Pusa Bold	55.80	52.33	58.87	47.80	52.40	81.13	8.80	54.73	48.87	13.87
Varuna	82.43	57.23	82.33	51.07	80.97	59.87	8.80	53.10	44.40	32.23
Pusa Bahar	50.83	87.20	57.70	85.27	89.53	81.13	8.37	83.87	55.43	33.33
Pusa Barani	54.73	59.80	58.40	53.83	53.93	53.83	8.53	81.07	55.13	32.23
RH-30	54.50	58.83	51.70	55.97	51.83	53.50	7.03	48.97	45.93	38.87
PR-1108	55.83	49.73	85.10	80.57	54.50	80.87	7.80	58.90	62.87	29.07
Poorbijaya	26.17	9.13	5.40	2.57	10.10	12.33	2.00	10.80	6.20	4.83
G. mutant	22.90	84.27	81.07	85.30	81.27	58.07	83.10	14.50	58.53	55.87
BJ-17	55.50	58.83	57.10	85.40	58.73	58.03	11.50	45.70	52.47	19.53
BJ-38	19.73	22.17	34.84	42.83	25.20	31.10	8.07	8.80	14.43	14.90

Combining ability analysis

Mean squares due to general and specific combining ability were significant for white rust infection index (Table 2). This suggested the important of both additive and non-additive gene action. However, the ratio of the additive total genotypic variance indicated that additive gene action mostly contributed towards the inheritance of white rust resistance in Indian mustard. Similar results were also reported in this crop [13-14].

Table 2. Analysis of variance for combining ability in 10 × 10 diallel set of *Brassica juncea* for white rust infection index

Source	d.f.	Mean squares
General combining ability	9	4308.9**
Specific combining ability	45	85.1**
Reciprocal cross effect	45	35.6**
Error	198	16.2

** : Significatn at 0.01 level

The parents Poorbijaya and BJ-38 showed significant and negative gca effects for white rust infection index (Table 3), thus, they were good general combiners for imparting the disease resistance. The magnitude and sign of gca effects for these parents were also in agreement with their *per se* performance (Table 1). This indicated that these genotypes could be considered as desirable parents for hybridization. A perusal of the Table 3 revealed that the crosses Pusa Bold × BJ-38, Varuna × Poorbijaya, Pusa Bahar × Poorbijaya, Pusa Barani × Poorbijaya, PR-1108 × Poorbijaya, Glossy mutant × BJ-38 and BJ-17 × BJ-38 exhibited significant and negative sca effects for the white rust infection index. The above crosses showing the desirable sca effects involved to poor × good gca parents for the disease resistance indicating that desirable transgressive segregates may be released from these crosses in subsequent generations. Therefore, these segregates could be utilised for improving the resistance to white rust in Indian mustard.

Table 3. Estimates of general combining ability effects (diagonal) and specific combining ability effects (ablve diagonal) for resistance to white rust in 10 × 10 diallel cross of *Brassica juncea*

Parents	Pusa Bold	Varuna	Pusa Bahar	Pusa Barani	RH-30	PR-1108	Poor-bijaya	Glossy mutant	BJ-17	BJ-38
Pusa Bold	4.89**	3.29	-2.64	-3.53	0.65	2.72	0.41	5.48*	1.20	-11.18**
Varuna		6.91**	5.45*	-1.19	4.08	-2.98	-6.92**	1.14	-1.39	-2.70
Pusa Bahar			10.10**	1.93	2.60	2.15	-10.80**	5.25*	0.07	0.89
Pusa Barani				7.40**	-0.38	-1.07	-10.93**	4.64	6.77**	7.09**
RH-30					5.51**	-2.38	-5.02	-2.12	0.73	3.43
RH-1108						8.47**	-6.48*	3.40	4.79	-1.47
Poorbijaya							-34.32*	-2.26	-2.92	16.69**
Glossy mutant								6.73**	-2.14	-14.07**
BJ-17									3.69**	-9.80**
BJ-38										-19.31**

*,** : Significant at 0.05 and 0.01 level, respectively

Genetic analysis

The estimates of genetic components of variances and the derived values from them are presented in Table 4. The t^2 estimate (0.4001) to test the uniformity of the Wr, Vr values was non significant suggesting fulfillment of the assumptions. A

further verification of the assumptions by the joint regression analysis showed that b (0.9513 ±0.0556) was significantly different from zero but not from 1.0. This satisfied the assumption regarding the adequacy of additive dominance model and could be inferred that non-allelic interaction was absent. The combining ability analysis (Table 2) clearly exhibited the presence of reciprocal differences. In such a situation, it was suggested to replace all the entries in the diallel table by the mean of the F_1s [15].

All the genetic parameters estimated from Jinks-Hayman model were highly significant for white rust infection index (Table 4). However, the magnitude of

Table 4. Esitmates of components of variance based on Hayman [10] and Jinks [11] for resistance to white rust disease in 10 × 10 diallel cross of *Brassica juncea* approach

Components	Estimated value
E	15.9** ± 2.9
D	311.9** ± 9.7
F	-293.5** ± 22.4
H_1	142.7** ± 20.7
H_2	93.9** ± 17.6
h^2	45.9** ± 11.7
Derived values	
$(H_1/D)^{1/2}$	0.68
$H_2/4H_1$	0.16
$[(4DH_1)^{1/2} + F]/[(4DH1)^{1/2}-F]$	0.18
Heritability(ns)	89.24

** : Significant at 0.01 level

additive variance (D) was higher than the dominance variance (H_1) indicating predominance of additive genetic variance as also evidenced from the combining ability analysis (Table 2). The average degree of dominance over all loci estimated by $(H_1/D)^{1/2}$ indicated partial dominance of the disease resistance. The H_2 component was smaller than H_1 indicating unequal proportion of positive and negative alleles in the loci controlling white rust infection. The asymmetrical distribution of genes at loci showing dominance in the parents was evident from the value of $H_2/4H_1$ (0.16) which was less than 0.25. The significant and negative estimate of F suggested an increase in recessive alleles among the parents. Preponderance of recessive genes in parents was also indicated by the estimate of the ratio $[(4DH_1)^{1/2} + F]/[(4DH_1)^{1/2}-F]$

which was less than unity. The heritability estimates in narrow and broad senses were 89% and 96%, respectively. Predominance of additive gene action and high heritability suggested that simple pedigree selection procedure would be worthwhile for improving the resistance level against white rust.

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REFERENCES

1. G. A. Petrie. 1988. Races of *Albugo candida* (white rust and staghead) on cultivated Cruciferae in Sakatchewan. Can. J. Plant Pathol., **10**: 142-150.
2. V. Verma and T. P. Bhowmik. 1989. Inheritance of resistance to a *B. juncea* pathotype of *Albugo candida* in *B. napus*. Can. J. Plant Pathol., **11**: 443-444.
3. M. M. Paladhi, R. C. Prasad and B. Dass. 1993. Inheritance of field reaction to white rust in Indian mustard. Indian J. Genet., **53**: 327-328.
4. M. D. Edward and P. H. Williams. 1982. Selection for quantitatively inherited resistance to *Albugo candida* in *Brassica campestris*, CGS-1. Cruciferae Newslett., **7**: 66-67.
5. G. S. Saharan, C. D. Kaushik and J. C. Kaushik. 1988. Sources of resistance and epidemiology to white rust of mustard. Indian Phytopathol., **41**: 96-99.
6. Anonymous. 1985. The unpublished Proceedings of Annual Oilseeds Workshop of Rapeseed and Mustard, Indian Council of Agricultural Research, New Delhi.
7. R. S. Singh. 1984. Introduction to Plant Pathology (3rd ed.). Oxford and IBH Publishing Co., India.
8. R. A. Fisher and F. Yates. 1957. Statistical tables for biological, agricultural and medical research (5th ed.). Hafner, New York.
9. B. Griffing. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Aust. J. Biol. Sci., **9**: 463-493.
10. B. I. Hayman. 1954. The theory and analysis of diallel crosses. Genetics., **39**: 789-809.
11. J. L. Jinks. 1954. The analysis of continuous variation in a diallel cross of *Nicotiana rustica* varieties. Genetics., **39**: 767-788.
12. K. Mather and J. L. Jinks. 1971. Biometrical Genetics : The study of continous variation (2nd ed.), Chapman and Hall, London.
13. D. Singh and H. Singh. 1987. Genetic analysis of resistance to white rust in Indian mustard. *In*: Proc. 7th Int. Rapeseed Cong., Poland, pp. 126.
14. H. S. Yashpal and D. Singh. 1991. Genetic components of variation for white rust resistance in Indian × exotic crosses of Indian mustard. Crop Res., **4**: 280-283.
15. B. R. Christie and V. I. Shattuck. 1992. The diallel cross: Design, analysis and use for plant breeders. Plant Breeding Review., **9**: 9-36.