

# Genetics of white rust resistance in [*Brassica juncea* (L.) Czern. & Coss.] and allelic relationship between interspecific sources of resistance

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## Abstract

The breakdown of resistance is the prime concern to search for new genes to develop a durable resistance against white rust in mustard. The present study was undertaken to know the mode of inheritance using indigenously developed resistance source and allelic relationship of genes for white rust resistance in two different sources viz. Bio-YSR and NPC-12 from *Brassica juncea* and *B. carinata*, respectively. The inheritance pattern of resistance genes in donors when crossed with two widely cultivated, highly susceptible cultivars, Varuna and Bio-902 (Pusa Jai Kisan), indicated the presence of a single dominant gene for white rust resistance. Cross between the resistant sources from *B. juncea* and *B. carinata* segregated in 15:1 (resistant: susceptible) ratio in F<sub>2</sub> generation, indicating the involvement of two different genes governing white rust resistance in these sources.

**Key words:** Indian mustard, white rust, inheritance, resistance, allelic relationship

## Introduction

White rust, a fungal disease caused by *Albugo candida* (Pers. ex Lev.) Kuntze is most wide-spread and highly destructive to *Brassica juncea*, reported to cause 17-34% yield losses [1]. The Indian genotypes are highly susceptible to white rust [2] and an appreciable loss in seed yield has been reported to the extent of 50% under late sown conditions [3]. Amongst the four oleiferous *Brassica* spp. grown in India, *B. juncea* is the predominant one and occupies more than 80% of the total area under rapeseed-mustard. Though *B. juncea* is hardy in comparison to other oilseed *Brassica* spp. for different biotic and abiotic stresses, yet it is highly susceptible to white rust. This disease is recurring every

season with different degrees of intensity at different stages of crop growth, which leads to wide fluctuation in the production and productivity of the crop year after year. The disease is characterized by the formation of white pustules on the cotyledons, leaves, stems and inflorescence. Systemically infected inflorescence becomes hypertrophied, causing the characteristic staghead galls [4]. It has been estimated that combined infection of leaf and inflorescence causes yield losses to the extent of 62.7%, the loss being more severe (89.8%) as a result of staghead formation in the susceptible cultivars [5]. Although, some chemical control in the form of seed treatment and foliar spray is recommended, yet, it would be effective only when disease initiation is identified and chemical sprayed at appropriate time. Moreover, it is costly and could be harmful to the environment too. Hence, host resistance would be the most effective and economic alternative for managing this disease. Identification of different sources of resistance to white rust is an important prerequisite in managing this disease by means of effective genetic resistance.

The biggest challenge in breeding white rust resistant *Brassicacae* is that as many as 13 pathotypes of *Albugo* sp. parasitize different cruciferous plant species [6, 7]. The white rust races are classified based upon their ability to infect different host species. In the case of *B. juncea*, race 2 of *A. candida* has been identified, which infects this species [8, 9]. Genetic analysis of available white rust resistance through biometrical techniques has elucidated a digenic mode of inheritance with duplicate gene action in *B. napus* [10, 11] and

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monogenic dominant resistance in *B. juncea* [12-15] as well as in *B. rapa*, *B. carinata* and *B. nigra* [16, 17]. The earlier studies were based on the exotic sources of white rust resistance. A stable white rust resistant donor Bio-YSR has been developed indigenously in *B. juncea* and the genetics of its resistance source remains to be studied. In comparison to the different exotic sources taken for genetic studies and used in white rust resistance breeding programme, Bio-YSR is agronomically superior and also stable under Indian conditions.

Dynamic changes in race composition of the pathogen have often resulted in short-lived efficiency of host resistance in the improved varieties. Therefore, a necessity to identify new sources of white rust resistance is imperative in breeding for durable resistance for this disease. Hence, the present study was undertaken to investigate the inheritance and genetic relationship of two different resistant sources one each from *B. juncea* and *B. carinata* for their use in future breeding programme for white rust resistance.

## Materials and methods

### Plant material

The experimental material used in the study includes two popular, widely grown, high yielding cultivars viz., Varuna and Bio-902 (Pusa Jai Kisan) of *B. juncea*, which are highly susceptible to white rust. The resistant sources used were Bio-YSR, a somaclone of *B. juncea* developed by NRCPB, IARI, New Delhi and registered with NBPGR (INGR No. 04099) and NPC-12, an advanced generation *B. carinata* genotype identified for multiple disease resistance in the pathological trials of AICRP on Rapeseed and Mustard. Bio-YSR, the resistant donor of *B. juncea*, was crossed with susceptible cultivars Varuna and Bio-902, and NPC-12, the resistant donor of *B. carinata*, was crossed with Varuna during *rabi* 2006-07 at IARI Experimental Farm, New Delhi to study the mode of inheritance of white rust resistance gene. Also, Bio-YSR x NPC-12 (resistant x resistant) cross was attempted to study the allelic relationship between resistance genes available in both the donors. The F<sub>1</sub>s, thus obtained were advanced during *Rabi* 2007-08 to derive F<sub>2</sub> population by selfing, and backcrossed to both the parents to obtain Backcross1(B<sub>1</sub>) (with susceptible parent, F<sub>1</sub>xP<sub>1</sub>) and Backcross2 (B<sub>2</sub>) (with resistant parent, F<sub>1</sub>xP<sub>2</sub>) generations for studying the genetics of white rust resistance.

### Screening for resistance

The parents, F<sub>1</sub>, B<sub>1</sub>, B<sub>2</sub> and F<sub>2</sub> generations of these crosses were grown and their white rust reaction was determined during *rabi* 2008-09. Two rows of parents and F<sub>1</sub>, five rows of B<sub>1</sub> and B<sub>2</sub> and ten rows of F<sub>2</sub> were planted with a spacing of 30 cm between rows and 10 cm between plants within row. Recommended package of practices were followed for raising a good crop. The inoculum was prepared by collecting the white rust zoospore from heavily infected fresh leaves of the susceptible *B. juncea* cultivars Varuna and Pusa Bold maintained in the National Phytotron Facility, IARI, New Delhi. The zoospore were collected in sterile distilled water and allowed to germinate for 4 hours at 8°C. The zoospore suspension was sprayed on the foliage with a hand atomizer until runoff. Dark conditions were maintained for 24 hrs after the spray for development of the disease by covering the entire plot with light blocking PVC sheet. To maintain high humidity, which is congenial for the disease development, experimental plot was irrigated frequently and water level was maintained in the channels around the plots during the period of inoculation. The plants were rated for white rust reaction two weeks after inoculation. For recording the observations for white rust, a minimum of 20 plants each from P<sub>1</sub>, P<sub>2</sub>, F<sub>1</sub>, 100 plants in B<sub>1</sub> and B<sub>2</sub> and 250 plants in F<sub>2</sub> were taken. The plants on which observations for white rust were recorded, were tagged from seedling stage and scoring was done upto the stage of staghead formation.

The disease scoring was done as per the method described by Fox and Williams [18]. Chi-square ( $\chi^2$ ) test was employed to test goodness of fit of observed and expected frequency in segregating generations.

## Results and discussion

The results of the present study are based on the observations recorded and analysis carried out for inheritance of white rust resistance in Parents, F<sub>1</sub>, B<sub>1</sub>, B<sub>2</sub> and F<sub>2</sub> generations of the three crosses involving two different sources of resistance i.e. Bio-YSR (*B. juncea*) and NPC-12 (*B. carinata*).

### Mode of inheritance

Disease scores of parental lines indicated that the donors Bio-YSR and NPC-12 were resistant to local *A. candida* population. The observations on P<sub>1</sub>, P<sub>2</sub>, F<sub>1</sub>, B<sub>1</sub>, B<sub>2</sub> and F<sub>2</sub> generations of these crosses are given in Table 1. The F<sub>1</sub> plants of these crosses Varuna x Bio-YSR, Bio-902 x Bio-YSR and Varuna x NPC-12 were

resistant to white rust indicating the dominant nature of resistance.

The segregation pattern in  $F_2$  of the three crosses fits well in 3 resistant (R):1 susceptible(S) ratio ( $\chi^2 = 1.58, 0.79$  and  $1.18$ , respectively) (Table 1), indicating that each resistant cultivar carries a single dominant gene. This monogenic dominant nature of the resistance gene to white rust was confirmed from the results of backcross populations as well. In the backcross ( $B_2$ ) with the resistant parents, all the plants were resistant, whereas in the backcrosses ( $B_1$ ) with susceptible parents, test cross progenies segregated in 1R:1S ratio.

These results of monogenic dominant nature of resistance to white rust are in agreement with the earlier findings [12-15].

#### Allelic tests

The  $F_2$  of the cross Bio-YSR x NPC-12 was screened artificially and ratio of resistant and susceptible plants was taken for the allelic relationship studies. A segregation ratio of 15:1 (R:S) observed in the  $F_2$  populations with a non-significant  $\chi^2$  value (2.94) (Table 2) demonstrated a goodness of fit to two dominant genes working in duplicate interaction, and confirmed that the

**Table 1.** Segregation pattern for white rust resistance in crosses among susceptible parents and resistant donors

Cross	Generations	Total plants	Observed		Expected ratio	Expected		$\chi^2$	P value
			R	S		R	S		
Varuna x Bio-YSR	$P_1$	27	0	27	-	0	27	-	-
	$P_2$	21	21	0	-	21	0	-	-
	$F_1$	23	23	0	-	23	0	-	-
	$F_2$	257	184	73	3:1	192.75	64.25	1.58	0.20 - 0.30
	$B_1$	112	64	48	1:1	56	56	2.28	0.10 - 0.20
	$B_2$	109	109	0	1:0	109	0	-	-
Bio-902 x Bio-YSR	$P_1$	25	0	25	-	0	25	-	-
	$P_2$	22	22	0	-	22	0	-	-
	$F_1$	20	20	0	-	20	0	-	-
	$F_2$	263	191	72	3:1	197.25	65.75	0.79	0.30 - 0.50
	$B_1$	119	52	67	1:1	59.5	59.5	1.89	0.10 - 0.20
	$B_2$	113	113	0	1:0	113	0	-	-
Varuna x NPC-12	$P_1$	24	0	24	-	0	24	-	-
	$P_2$	17	17	0	-	17	0	-	-
	$F_1$	15	15	0	-	15	0	-	-
	$F_2$	237	170	67	3:1	177.75	59.25	1.18	0.20 - 0.30
	$B_1$	92	41	51	1:1	46	46	1.08	0.20 - 0.30
	$B_2$	78	78	0	1:0	78	0	-	-

**Table 2.** Segregation pattern for white rust resistance in crosses between resistant donors

Cross	Generations	Total plants	Observed		Expected ratio	Expected		$\chi^2$	P value
			R	S		R	S		
Bio-YSR x NPC-12	$P_1$	25	25	0	-	25	0	-	-
	$P_2$	21	21	0	-	21	0	-	-
	$F_1$	16	16	0	-	16	0	-	-
	$F_2$	208	189	19	15:1	195	13	2.94	0.05 - 0.10

two genes segregating in the population for resistance were non-allelic.

The study revealed that resistance is monogenic in both the resistant sources and it is governed by different genes in two resistant sources. Hence, the major gene governing white rust resistance could be easily transferred to the well adapted, high yielding but susceptible genotypes by backcross breeding. The presence of different resistance gene(s) in the two different resistant sources will be very useful in breeding for durable resistance by the diversification of resistant sources and gene pyramiding. However, more detailed studies would have to be conducted to analyse the virulence spectra and diversity in the pathogen population through controlled experiments with each of these genes.

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### References

1. **Kolte S. J.** 1985. Diseases of Annual Edible Oilseed Crops. Volume II Rapeseed-Mustard and Sesame diseases. CRC Press, Boca Raton, Florida, pp.135.
2. **Li C. X., Sivasithamparam K., Walton G., Fels P. and Barbetti M. J.** 2008. Both incidence and severity of white rust disease reflect host resistance in *B. juncea* germplasm from Australia, China and India. *Field Crops Research*, **106**: 1-8.
3. **Saharan G. S., Kaushik C. D., Gupta P. P. and Tripathi N. N.** 1984. Assessment of losses and control of white rust of mustard. *Indian Phytopathol.*, **37**: 397.
4. **Verma P. R. and Petrie G. A.** 1980. Effect of seed infestation and flower inoculation on systemic infection of turnip rape by *Albugo candida*. *Can. J. Plant Sci.*, **60**: 267-271.
5. **Lakra B. S. and Saharan G. S.** 1989. Correlation of leaf and staghead infection intensities of white rust with yield and yield components of mustard. *Indian J. Mycol. Plant Pathol.*, **19**: 279-281.
6. **Pound G. S. and Williams P. H.** 1963. Biological races of *Albugo candida*. *Phytopathol.*, **53**: 1146-1149.
7. **Liu J. Q., Parks P. and Rimmer S. R.** 1996. Development of monogenic lines for resistance to *Albugo candida* from a Canadian *Brassica napus* cultivar. *Phytopathol.*, **86**: 1000-1004.
8. **Lakra B. S. and Saharan G. S.** 1988. Morphological and pathological variations in *Albugo candida* associated with *Brassica* species. *Indian J. Mycol. Plant Pathol.*, **18**: 149-156.
9. **Petrie G. A.** 1988. Races of *Albugo candida* (white rust and staghead) on cultivated Cruciferae in Saskatchewan. *Can. J. Plant Pathol.*, **10**: 142-150.
10. **Fan Z., Rimmer S. R. and Stefanson B. R.** 1983. Inheritance of resistance to *Albugo candida* in rape (*Brassica napus* L). *Can. J. Genet. Cytol.*, **25**: 420-424.
11. **Verma V. and Bhowmik T. P.** 1989. Inheritance of resistance to a *Brassica juncea* pathotype of *Albugo candida* in *Brassica napus*. *Can. J. Plant Pathol.*, **11**: 443-444.
12. **Tiwari A. S., Petric G. A. and Downey R. K.** 1988. Inheritance of resistance to *A. candida* race 2 in mustard (*B. juncea* (L.) Czern). *Can. J. Plant Sci.*, **68**: 297-300.
13. **Bansal V. K., Thiagarajah M. R., Stringam G. R. and Tewari J. P.** 1999. Inheritance of partial resistance to race 2 of *Albugo candida* in canola-quality mustard (*Brassica juncea*) and its role in resistance breeding. *Plant Pathol.*, **48**: 817-822.
14. **Sachan J. N., Kolte S. J. and Singh B.** 2000. Inheritance of white rust (*Albugo candida* race 2) in *Brassica juncea*. *Indian Phytopathol.*, **53**: 206-209.
15. **Chauhan S. K. and Sharma J. B.** 2001. Inheritance of white rust resistance in Indian mustard incorporated from *Brassica napus*. *Indian J. Genet.*, **61**: 250-252.
16. **Delwiche P. A. and Williams P. H.** 1974. Resistance to *Albugo candida* race 2 in *Brassica* spp. *Proc. Am. Phytopathol. Soc.*, **1**: 66.
17. **Delwiche P. A. and Williams P. H.** 1981. Thirteen marker genes in *Brassica nigra*. *J. Hered.*, **72**: 289-290.
18. **Fox D. T. and Williams P. H.** 1984. Correlation of spore production by *Albugo candida* in *Brassica campestris* and a visual rating scale. *Can. J. Plant Pathol.*, **6**: 175-176.