Indian J. Genet., 72(1): 89-91 (2012)

Short Communication

Understanding the genetic relationship among resistant sources of white rust, a major fungal disease of *Brassica juncea*

D. K. Yadava*, M. Vignesh, Sujata V., N. Singh, R. Singh, B. Dass, M. S. Yadav[1], T. Mohapatra[2] and K. V. Prabhu

Indian Agricultural Research Institute, New Delhi 110 012; [1]NCIPM, [2]NRCPB, Pusa Campus, New Delhi 110 012

(Received: July 2011; Revised: December 2011; Accepted: January 2012)

In India four oleiferous *Brassicas viz*., *B. juncea*, *B. napus*, *B. rapa* and *B. carinata* are cultivated in about 6.18 million ha with a production of 7.36 million tons [1] contributing about 26.8% and 24.7% of the total oilseed production and acrage, respectively. The productivity of this group of crops in India is 1190 kg ha^{-1} which is far below than the world average (1510 $kgha^{-1}$). *Brassica juncea* (L.) Czern & Coss. prominently occupies major area in India contributing more than 80% of the total rapeseed mustard production. Both biotic and abiotic stresses adversely affect the yield of this crop resulting in low productivity. Fungal diseases alone can cause major damage resulting in yield losses upto 70% under natural epiphytotic conditions [2].

White rust, a fungal disease caused by *Albugo candida* (Pers. ex Lev.) Kuntze, is most prevalent and extremely destructive to *B. juncea*. The Indian cultivars are highly susceptible to white rust [3] and loss to the extent of 50% in seed yield has been reported, under late sown conditions [4]. 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 [5]. It has been estimated that combined infection of leaf and inflorescence causes yield losses to the extent of 62.7%. The loss becomes more severe (89.8%) as a result of staghead formation in the susceptible cultivars [6].

Exploitation of host-plant resistance is the most efficient, effective and economic control of white rust. Dynamic changes in race composition of the pathogen have often resulted in short-lived efficiency of host resistance in the improved varieties. A necessity to identify new sources of white rust resistance is thus imperative for breeding for durable resistance. The present study was, therefore, undertaken to investigate the genetic relationship among six different resistant sources to know the extent of allelic diversity for white rust resistance gene in Indian mustard and for their use in future breeding programme.

The resistant sources used in this study were Bio-YSR, a somaclone of *B. juncea* developed by NRCPB, IARI, New Delhi [7] and registered with NBPGR (INGR No. 04099), BEC-144, BEC-286 and EC-399301 exotic collections and JM-1 and JM-2 cultivars released from ZARS, Morena (MP) for cultivation in Madhya Pradesh. Crosses were attempted among the resistant sources in six different combinations *viz*., Bio-YSR x BEC-144, Bio-YSR x EC-399301, BEC-286 x Bio-YSR, EC-399301 x BEC-144, JM-1 x EC-399301 and JM-2 x BEC-286 to study the allelic relationship among resistance genes present in these resistant sources. The F_1s thus obtained were advanced to generate F_2 populations by selfing, for studying the genetic relationship among the resistant donors.

The F_2 populations were screened under artificial inoculated conditions in replicated trials, both in

[1]Corresponding author's e-mail: dkygenet@gmail.com
Published by Indian Society of Genetics & Plant Breeding, F2, First Floor, NASC Complex, PB#11312, IARI, New Delhi 110 012
Online management by indianjournals.com

containment in National Phytotron Facility (NPF), IARI, New Delhi and in the field conditions for two consecutive years *rabi* 2009-10 and 2010-11. The inoculum was prepared by collecting the white rust zoosporangia from heavily infected fresh leaves of the susceptible *B. juncea* cultivar Varuna and Pusa Bold maintained at NPF. The zoosporangia were collected in sterile distilled water and allowed to germinate for 4 hours at 8^{o}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 was kept standing in channels surrounding the plots during the period of inoculation. The plants were rated for white rust reaction two weeks after inoculation. Ratio of the susceptible to the resistant plants was recorded for understanding the allelic relationship of the genes responsible for the disease resistance. Observations for white rust infection were recorded on a minimum of 20 plants each from the parental genotypes (P_1 and P_2) as well as the first filial generation (F_1) and 250 plants of F_2 from both replications were phenotyped to record the manifestation of the disease in all six crosses. All the plants were tagged at seedling stage itself and observations were recorded on individual plants until the stage of staghead formation. To confirm the pathogenecity of the inoculum the susceptible check Varuna was also sown along with the F_1s, F_2, and parents.

The disease scoring was done as per the method described by Fox and Williams [8]. The plants were phenotyped for their reaction to white rust using 0-9 scale and classified into six categories: 0 (HR = highly resistant: no symptoms on either side of the leaves; 1(R = resistant: small, pinpointed to large brown necrotic flecks on the upper surface but no sporulation; 3 (MR = moderately resistant: one to a few minute scattered pustules on the upper surface and none to very few pinpoint pustules on the lower surface; 5 (MS = moderately susceptible: few to many scattered pustules on the upper surface and few scattered pustules on the lower surface; 7 (S = susceptible: few to many pustules on the upper surface and many scattered small to large pustules on the lower surface and 9 (HS = highly susceptible: very few or no pustules on the upper surface and many large coalescing pustules on the lower surface. Categories 0-3 were defined as resistant with complete absence of white rust pustules or with very low hypersensitive infection and categories 5-9 as susceptible with more rust pustules and development of stagheads at later stages. Chi-square (χ^2) test was employed to test goodness of fit of observed and expected frequency in segregating generations.

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_{1s} and F_2 generations of the six cross combinations involving six different white rust resistant sources *viz*., Bio-YSR, BEC-144, BEC-286, EC-399301, JM-1 and JM-2. All the F_1 hybrids showed resistance to white rust indicating that the resistance is dominant over susceptibility. The individuals in all the F_2 populations derived from the crosses, Bio-YSR x BEC-144, Bio-YSR x EC-399301, BEC-286 x Bio-YSR, EC-399301 x BEC-144, JM-1 x EC-399301 and JM-2 x BEC-286 were scored for resistance. No segregation for resistance and susceptibility was noticed as all F_2 individuals were resistant (0-3 disease score) and no susceptible plants were observed in both replications under both field and controlled (NPF) conditions during the first season *rabi* 2009-10. To confirm the disease reaction and to avoid any escapes of disease incidence, the F_2 population along with their parents and susceptible check Varuna were also screened for white rust incidence during *rabi* 2010-11 under similar conditions as mentioned above. A similar pattern of rust infection was observed as recorded during the preceding year. The susceptible check Varuna exhibited severe infection of white rust (>5 disease score) during both the crop seasons as well as under NPF. This indicated that the resistance gene(s) present in the six different resistant stocks *viz*., Bio-YSR, BEC-144, BEC-286, EC-399301, JM-1 and JM-2 were allelic to each other and the same gene for white rust resistance is operating in all the resistant sources against the race prevailing under Delhi conditions. It can be concluded that resistance to the white rust race is controlled by a single dominant gene. The results of the present study are in accordance with earlier findings and the same gene is widely distributed [9-11, 13]. The results of mode of inheritance of white rust resistance worked out through genetic analysis of BC_1, BC_2 and F_2 populations from the cross Bio-YSR x BEC-144 during *rabi* 2008-09 were also similar and that the resistance gene(s) present in Bio-YSR and BEC-144 were allelic [12].

The present study has shown the limited or no allelic diversity for white rust resistance in the Indian mustard germplasm, therefore, it is necessary to identify

and characterize new potential donors to prevent any disease epidemics likely to be caused by the breakdown of resistance. The identification of new and diverse sources for resistance will lead to the development of durable resistance against white rust in mustard.

Acknowledgement

The authors thankfully acknowledge the financial support provided by the Department of Biotechnology, New Delhi.

References

1. **AICRP-RM** 2009. 16th Annual Progress Report of All India Coordinated Research Project on Rapeseed and Mustard, Directorate of Rapeseed Mustard Research, Sewar, Bharatpur, pp. 1.
2. **Chopra V. L. and Prakash S.** 1996. Oilseeds and Vegetable Brassicas. An Indian Perspective. Oxford & IBH Publishing Co. Pvt. Ltd. New Delhi.
3. **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 Res., **106**: 1-8.
4. **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 Phytopath., **37**: 397.
5. **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.
6. **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.
7. **Katiyar R. K. and Chopra V. L.** 1990. Somaclonally induced earliness in *Brassica juncea* germplasm accessions with field resistance to important disease. Plant Breed., **104**: 262-264.
8. **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.
9. **Prabhu K. V., Somers D. J., Rakow G. and Gugel R. K.** 1998. Molecular markers linked to white rust resistance in mustard *Brassica juncea*. Theor. Appl. Genet., **97**: 865-870.
10. **Mukherjee A. K., Mohapatra T., Varshney A., Sharma R. and Sharma R. P.** 2001. Molecular mapping of a locus controlling resistance to *Albugo candida* in Indian mustard. Plant Breed., **120**: 483-487.
11. **Sachan J. N., Kolte S. J. and Singh B.** 2000. Inheritance of white rust (*Albugo candida* race 2) in *Brassica juncea*. Indian Phytopath., **53**: 206-209.
12. **Vignesh M., Yadava D. K., Sujata V., Mohapatra T., Jain N., Yadav A. K., Malik D., Yadav M. S. and Prabhu K. V.** 2009. Genetics of white rust resistance in *Brassica juncea* (L.) Czern. & Coss. and allelic relationship between interspecific sources of resistance. Indian J. Genet., **69**: 205-208.
13. **Vignesh M., Yadava D. K., Sujata V., Yadav A. K., Mohapatra T. and Prabhu K. V.** 2011. Characterization of an Indian mustard (*Brassica juncea*) indigenous germplasm line Bio-YSR for white rust resistance. Indian J. Plant Genetic Res., **24**: 40-42.