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BREEDING FOR WHITE RUST RESISTANCE IN INDIAN MUSTARD (*BRASSICA JUNCEA* (L.) CZERN AND COSS.)

B L Meena[1]*, Sudhir Kumar[2], S.K Meena[3] and Reena Kumari[4]
[1]ICAR-Directorate of Rapeseed-Mustard Research, Bharatpur (Rajasthan)
[2] ICAR-Indian Institute of Agricultural Biotechnology, Ranchi (Jharkhand)
[3]Department of Education, Govt of Rajasthan, Bharatpur (Rajasthan)
[4]Department of Agriculture, Govt of Rajasthan, Dausa (Rajasthan)
E-mail: blmeena.icar@gmail.com

Abstract

White rust caused by Albugo candida have been reported to be most wide spread and destructive fungal disease of rapeseed-mustard throughout the world (Kolte, 1985). It is one of the important diseases of rapeseed-mustard in India causing a yield loss of 17-34 %. The disease is characterized by both local and systemic symptom expression.

The major challenge in breeding for white rust resistance in Brassicas is the prevalence of large number of pathotypes of Albugo parasitizing different cruciferous species. Race 2 of A. candida infects B. juncea. Genetic analysis of available white rust resistance through biometrical techniques has revealed a digenic mode of inheritance with duplicate gene action in B. napus and monogenic dominant resistance in B. juncea as well as in B. rapa, B. carinata and B. Nigra.

Introduction

Indian mustard (*Brassica juncea* (L.) Czern and Coss.) is predominantly cultivated oilseed crops in India. Due to its higher seed yield and adaptation, it is widely cultivated under different agro-climatic conditions and cropping patterns. The species of *Brassica* are altered by number of diseases. Among them white rust (*Albugo candida*) is most important and widely distributed in tropical and temperate climates. In India, rapeseed-mustard *(Brassica species*) are placed at second position in total acreage (23.91 percent) and production (27.19 percent) after soybean among oilseeds crops. There are three ecotypes of *Brassica* (2n = 20) *i.e. Brassica campestris* ssp. Oleifera *viz.*, yellow sarson, brown sarson and toria, collectively called rape (syn. *B.rapa*L.), *B. juncea* or brown mustard (2n = 36) and *Eruca sativa* or Taramira (2n = 22)are commercially cultivated. Among these, Indian-mustard *(Brassica juncea)* accounts for > 80 per cent of the area under rapeseed-mustard crops in the country. Breeding system for a crop species mainly depends upon its mode of pollination. Therefore, in plant breeding, breeders need to be aware of the existing genetic variability in terms of nature and magnitude, and wild relatives of a crop species, its

reproductive behaviour, adaptation to environments and cropping systems, and usage for the objective and methods chosen for its genetic improvement. The effectiveness and efficiency of selection is greatly advanced when the magnitude and nature of genetic variation is understood, and rapid and reliable screening techniques are available.

Indian mustard (*Brassica juncea* (L.) Czern and Coss.) is predominantly cultivated oilseed crops in India. Due to its higher seed yield and adaptation, it is widely cultivated under different agro-climatic conditions and cropping patterns. The species of *Brassica* are altered by number of diseases. Among them white rust (*Albugo candida*) is most important and widely distributed in tropical and temperate climates (Saharan and Verma, 1992). Appreciable losses were caused by *Albugo candida*to seed yield up to a tune of 17 to 37 per cent (Kolte, 1985).

White rust caused by *Albugo candida* Kuntze is an economically important disease of Indian mustard (*Brassica juncea* (L.) Czern and Coss.) particularly in india. The disease is characterized by two types of symptoms. Initially isolated white pustules or blisters are formed on the leaves and often appear in a circular arrangement around a big central pustule. In later stages, the young stem, inflorescence, floral parts and young siliqua will be infected. The pathogen become systemic in infected tissues and causes various types of malformations with prominent hypertrophy and hyperplasia that appears swelling or distortion and finally form stagehands. These malformations and deformities are the result of hormonal imbalance induces by the pathogen. The major challenge in breeding for white rust resistance in brassica is the prevalence of large number of pathotypes of *Albugo* parasitizing different cruciferous species. Genetic analysis of available white rust resistance through biometrical techniques has revealed a digenic mode of inheritance with duplicate gene action in *B.napus* and monogenic dominant resistance in *Brassica juncea* (L.) as well as *B.rapa*, *B.carinata* and *B.nigra.*

Most of Indian cultivars (except few) released for commercial cultivation of *B. juncea* are susceptible to white rust and sources of resistance to white rust disease are available in *B. juncea* germplasm. Despite the availability of stable donors for white rust, no breakthrough has been made in the development of white rust resistant varieties of *B. Juncea.* The chemical control has not been much economical and successful. Therefore, genetic resistance is the most practical, economical and environment friendly method to overcome this menace. Therefore resistant / tolerant genotypes of Indian mustard for white rust will be developed for development of high yielding disease resistant cultivars with enhanced and stable production of Indian mustard.

A variant of race 2 (race 2V) was identified on the resistant cultivar Cutlass for which there is no resistance available in *B. juncea* (Rimmer and Buchwaldt 1995). Resistance to white rust in the *Brassica* species for which information is available is governed by simple Mendelian genes, including a single dominant gene in *B. juncea* against race 2 (Tiwari et al. 1988; Rimmer and Buchwaldt 1995), three dominant genes in *B. napus* against race 7 (Fan et al. 1983; Liu et al. 1996), and a single dominant gene in *B. rapa,* also against race 2 (Kole et al. 1996).

Inheritance of white rust (*Albugo candita*, race 2) resistance in Indian mustard (*Brassica juncea*) was studied by Tiwari *et.al*. (1988) using crosses between one resistant and two susceptible cultivars. The reaction of F_1 (all resistant) and segregation of resistant and susceptible plants in F_2 (3:1) and backcross (1:1) indicated the resistance as dominant

monogenic and controlled by nuclear genes and can be easily transferred to adopted susceptible genotypes *via* backcrossing.

Vignesh *et.al*. (2009) investigated the mode of inheritance using indigeneously developed resistance source and allelic relationship of genes for white rust resistance in two different sources viz.,Bio-YSR (*Brassica juncea*) and NPC-12 (*B.carinata)*. The Inheritance of resistance in donors when crossed with two highly susceptible cultivar,Varuna and Bio-902 indicated the presence of single dominant gene for white rust resistance. The cross between two resistant sources from *Brassica juncea* and *B.carinat* segregated in 15:1(resistsnt: susceptible) ratio in F_2 indicated the involvement of two different genes governing white rust resistance in these sources.

Adhikari *et.al*. (2003) reported a virulence gene AvrAc1 in *A. candita* and suggested that a single dominant gene controls avirulence in race Ac2 to *B rap* acv Torch.

Breeding Methods

The effort have also been made through inter-specific hybridization between *B. juncea* and *B. carinata* to transfer white rust resistance into well adapted high yielding back-ground of *B. juncea* from *B. carinata* through pedigree selection. The monogenic resistance with complete dominance in *B. carinata* could be partially introgressed into *B. juncea* cultivars through selection of disease-free plants in segregating gene-rations grown under heavy disease pressure and repeated back crossing. With the use of resistant gene (L6) from Canada, many white rust resistance lines have been developed in the genetic background of high yielding varieties. The varieties Basanti , JM-1, JM-2 and JM-3 have been released for general cultivation in white rust prone areas of India.

Molecular Studies

In the genus Brassica, molecular mapping of genes has been reported for various traits like seed coat colour, growth habit, oil content, fatty acid content and resistance to diseases, including white rust (Quiros *et al.,* 2001; Lakshmi kumaran *et al.,* 2003). Sources of resistance against white rust are available in east European *B. juncea* gene pool. Genetic analysis of white rust resistance in *B. juncea* has been undertaken at the molecular level to locate gene/sand to identify markers for marker-assisted introgression of the traits using RFLP (Cheung *et al.,* 1998), RAPD (Prabhu *et al.,*1998; Mukherjeeet *al.,*2001), AFLP (Somers *etal.,*2002), CAPS (Varshney *etal.,*2004) and IP markers (Panjabi-Massand *et al.,* 2010). A locus (ACAl) controlling resistance to white rust has been mapped in *B. napus* (Ferreira *et al.,* 1995) and *B. rapa* (Kole*etal.,*1996), using restriction fragment length polymorphism (RFLP) markers.

Tanhuanpaa (2004) tagged a locus for white rust resistance in a F2 population of *Brassica rapa* ssp. *oleifera* employing bulked segregant analysis with random amplified polymorphic DNA (RAPD) markers, linkage mapping and a candidate gene approach based on resistance gene analogs (RGAs) using Finnish line Bor4109 as resistance source. The reaction against white rust races 7a and 7v was scored in 20 seedling F2. The proportion of resistant plants among F3families varied from 0 to 67 per cent. Bulked segregant analysis did not reveal any markers linked with resistance, thus a linkage map with 81 markers was created. A locus accounting for 18.4 per cent of the variation in resistance for white rust was mapped to linkage group (LG) 2 near the RAPD marker Z19a. During the study, a bacterial resistance gene homologous to *Arabidopsis* RPS2 and six different RGAs were sequenced. RPS2 and

five of the RGAs were mapped to linkage groups LGl, LG4 and LG9.Unfortunately, none of the RGAs could be shown to be associated with white rust resistance. Singh *et al.* (2015) effectively applied the already identified *Arabidopsis-derived* intron polymorphic (IP) markers At5g41560 and At2g36360, which were highly linked with AcB1-A4.1 and AcB1-A5. l, respectively and validated in a set of 25genotypes of Indian Mustard and three F_2 populations. The relationships between the variation of PCR products of the two markers with the percent disease index (PDI) of the tested genotypes, and the co-segregation analysis of the markers with disease phenotype in F_2 populations clearly indicated that At5g41560 and At2g36360 are genotype-nonspecific markers and are closely linked to white rust resistance loci AcB1-A4.1 and AcB1-A5. l, respectively. It also became evident from the study that AcB1-A4.1 and an another white rust resistance locus Ac (2) t are likely the same gene locus. These markers can further be used in marker assisted breeding for gene pyramiding.

Conclusion

The most efficient and cost-effective way of protecting mustard plants from white rust disease is through genetic resistance. The DH population was used to screen for RAPD markers associated with white rust resistance/ susceptibility using BSA. Two markers, WR2 and WR3, linked to white rust resistance, flanked the resistance locus *Ac21* and were highly effective in identifying the presence or absence of the resistance gene in the DH population. It is concluded that the availability of these RAPD markers will enhance the breeding for white rust resistance in *B. juncea*. Inheritance of white rust resistance in P_1, P_2, F_1, F_2 and backcross generation of two crosses of *Brassica juncea* revealed that one gene with complete dominance conferred resistance to white rust. A segregation ratio of 3 resistant: 1 susceptible in F_2 generations of these crosses further confirmed that white rust resistance is governed by a single dominant gene which can easily be transferred by backcrossing from a resistant to susceptible genotypes.The breakdown of resistance is the prime concern to search for new genes to develop a durable resistance against white rust in mustard.

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