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Short Communication

Evaluation of Indian mustard (*Brassica juncea*) genotypes against white rust (*Albugo candida*)

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Abstract

Fifty-seven genotypes of Indian mustard (*Brassica juncea*) were evaluated for white rust resistance using one susceptible; NRCHB 101 (*B. juncea*) and two resistant; GSL 1 (*B. napus*) and DLSC 1 (*B. carinata*) checks. The most promising rapeseed-mustard genotypes for white rust resistance were AJ-34, 252025, DRMR-2035, Heera, Donskaja-IV, Basanti, Pusa Jaikisan WRR, Pusa Bold WRR and DTM-34. These genotypes were immune and recorded 0.0% disease severity at both locations.

Keywords: *White rust, evaluation, Indian mustard, genotypes*

Introduction

In terms of economic significance, oilseeds are second only to cereal crops. Out of nine oilseed crops that are cultivated in India, seven (rapeseed-mustard, groundnut, soybean, sunflower, sesame, safflower, and niger) are edible. Rapeseed-mustard is a major winter season (*Rabi*) crop and sustains itself on residual moisture. The low irrigation water requirement makes it the best fit for many cropping systems. In India, rapeseed-mustard is cultivated under a wide range of agro- climatic conditions. Among all species, Indian mustard [*Brassica juncea* (L) Czern. & Coss.] has the lion's share (about 36%) in India's edible oil basket and a profound impact on the national economy (DRMR, 2023).

As Indian mustard is grown under a diverse range of agro-climatic conditions, its production is severely hampered by various biotic and abiotic stresses. It is attacked by numerous plant pathogens and some of the economically significant diseases are leaf blight (*Alternaria brassicae* and *A. brassicicola*), white rust (*Albugo candida*), stem rot (*Sclerotinia sclerotiorum*), downy mildew (*Hyaloperonospora parasitica*) and powdery mildew (*Erysiphe cruciferarum*). White rust, caused by an obligate biotrophic oomycete *Albugo candida* (Pers. ex Lev.) Kuntze is a serious impediment in realizing the potential production of *B. juncea*. Vegetative infection is marked by white pustules formation on the abaxial surface while staghead formation is the characteristic of reproductive stage infection (Kolte, 1985). Yield losses, ranging from 1% to 90%, have been reported which is dependent upon host genotype, plant population, nutrition, and planting time (Saharan and Verma, 1992). The pathogen survives in the soil or as a contaminant of the seed with oospores. Primary infection is caused by the oospores forming an infection tube depending upon climatic conditions and by zoospores formed from germinating oospores while the secondary spread occurs by zoospores and sporangia formed in diseased plant parts (Saharan *et al.,* 2005). Though chemicals such as metalaxyl are the most used resort against white rust, no single method of disease management can be termed as viable, effective, and environmentally safe. Therefore, integration of various control methods has been suggested and resistant varieties play a very important role because of the most feasible and economical options. Host resistance is termed the best way to manage any disease as it is compatible with all other means of disease management. It has double advantages, as environmentally desirable and economically feasible. As *A. candida* is a biotrophic fungus, it shares an intricate relationship with the host. This allows faster evolution of virulent races/biotypes against resistant genotypes. Thus, there is a need for a continuous search of resistance genotypes so that they can be assimilated into breeding programs. Further, with the growing environmental and economic concern against chemicals, there has been a rapid shift towards identifying resistant sources for white rust. Though some resistant genotypes have been reported based on field studies at multiple locations, an intensive and continuous screening program is a must to identify new resistant genotypes against wide range of *A. candida* pathotypes. The present study was undertaken to screen Indian mustard genotypes against *Albugo candida* at adult plant stage.

Materials and Methods

The plant material in present investigation comprised

24 varieties and 36 germplasm accessions including checks; NRCHB 101 (*Brassica juncea*) and GSL 1 (*B. napus*) and DLSC 1 (*B. carinata*). Seeds of genotypes accessions were obtained from germplasm unit of ICAR-Directorate of Rapeseed-Mustard Research, Bharatpur and ICAR-National Bureau of Plant Genetic Resources, New Delhi. All 60 genotypes were screened at two locations *viz*., experimental farm of ICAR-Indian Agricultural Research Institute, New Delhi (28° 38' 8.736" N and 77° 13' 28.02 E) and ICAR-Directorate of Rapeseed-Mustard Research, Bharatpur (27° 12' 36" N and 77° 17' 24 E) during the crop season 2022-23. At both the locations, the experiment was conducted in a completely randomized block design. A single 3 m row of single entry was sown. Row to row and plant to plant spacing was maintained as 30 × 10 cm. After every two test entry, the susceptible check variety NRCHB-101 was grown. The variety GSL 1 (*B. napus*) and DLSC 1 (*B. carinata*) were used as resistant checks. The recommended agronomic practices like field preparation, thinning, weeding, fertilizer application, irrigation etc., were followed. No fungicide application was done during the crop season to ensure a maximum disease pressure in the natural field condition. At New Delhi, the sowing was done November 05, 2022 and at Bharatpur, the sowing was done on November 07, 2022.

The observations on occurrence and severity of disease were recorded fortnightly on 10 randomly selected plants of each entry including checks. The final observation was recorded 90 days after sowing (DAS) under natural conditions following the 0-9 disease assessment key (Khajan *et al.,* 2018) illustrated as: 0 (no pustules), 1 (less than 5% leaf area covered by pustules), 3 (5-10% leaf area covered by pustules), 5 (11-25% leaf area covered by pustules), 7 (26-50% leaf area covered by pustules) and 9 (26-50% leaf area covered by pustules). The final observation on disease was recorded 90 DAS under natural conditions (Table 1), and data were analyzed for coefficient of variation and critical difference at 5% level of significance.

Table 1: Disease assessment key (0-9) (Khajan *et al*., 2018)

Rating score	Leaf area covered (%)	Disease reaction
0	No symptom	Immune (I)
1	Less than 5	Highly resistant (HR)
3	5-10	Resistant (R)
5	11-25	Moderately resistant (MR)
7	26-50	Susceptible (S)
9	More than 50	Highly susceptible (HS)

$$\text{Disease index (\%)} = \frac{\text{Sum of all numerical ratings}}{\text{Number of leaves examined} \times \text{Maximum ratting}} \times 100$$

Results and Discussion

Screening of the genotypes in natural field conditions is used to evaluate resistance since long time back. In the present investigation, a total of 57 genotypes of Indian mustard were selected to screen for white rust resistance at the adult plant stage. The evaluation was carried out at two different locations *viz.,* ICAR- IARI, New Delhi and ICAR-DRMR, Bharatpur, for establishing resistant genotypes. The result is presented in Table 2 and Fig. 1. At Bharatpur, out of 57 genotypes, 09 (AJ-34, Heera, 252025, DTM-34, Donskaja-IV, DRMR-2035, Basanti, Pusa Jaikisan WRR, Pusa Bold WRR) were immune, 09 (DYJ-1, IM-152, DRMR 1-5, DRMRIJ 12-40, DRMRIJ-4, DRMR 2018-37, BIO-YSR, Rohini WRR, and Varuna WRR) were highly resistant, 04 (PBY-1, PBY- 3, Rohini, RH 1400) were resistant and 10 genotypes (B-394, BJ-24-5, K-9284, EJ-19, IM-39, IM-59, DRMRJA-35, Pusa Karishma, IM-127, ATC-94394) were moderately resistant. At New Delhi, 13 (AJ-34, DYJ-1, IM-152, DRMRIJ 12-40, DRMR 2018-37, Heera, 252025, DTM-34, Donskaja-IV, DRMR-2035, Basanti, Pusa Jaikisan WRR, Pusa Bold WRR) were found immune, 03 (DRMR 1-5, BIO-YSR, Rohini WRR) were highly resistant, 06 (PBY-1, PBY-3, IM-127, Varuna WRR, DRMR IJ 12-48, Rohini) were resistant and 09 (B-394, BJ-24-5, K-9284, EJ-19, IM-39, IM-59, Pusa Karishma, RH 1400, ATC-94394) were found to be moderately resistant. The most promising genotypes across both the locations were AJ-34, 252025, DRMR-2035, Heera, Donskaja-IV, Basanti, Pusa Jaikisan WRR, Pusa Bold WRR, DTM-34, as they recorded 0.0% disease severity and were found to be immune. Similarly, many researchers found white rust resistant sources based on field screening. Srivastava and Verma (1987) evaluated 45 lines of *B. juncea* and *B. carinata* under natural field conditions and only 2 of them (EC338997 and PBN 002) were found to be resistant. Kumar and Kalha (2005) checked 40 germplasms of *B. napus* and *B. carinata* and found all of them were resistant. Singh and Mall (2007) screened 170 rapeseed mustard genotypes and 5 of them to be immune (WRR-98-01, NDRS-2004, NDRS-2013, NDRS-2005 and NDRS-2007), while 18 were resistant (NNRE-10, NDYR-29 x NDRE-04, NDRE 190 x NDRE-4CSCN-5, CSCN-3, CSCN-10, RC-781, RIK-75-5, RIK78-4, CSR-721, PI-43, YRT-3, NDRE-7, CSCN-12, NSRS-2006, NDRS-2009, NDRS-201, and NDRS-12) against *A. candida*. Ahmad *et al.* (2014) screened twelve lines of *B. juncea* and *B. napus* against white rust under field conditions. *B. juncea* 44S01 and *B. napus* Sps-N7/26 were termed as "highly resistant". Brassica materials were evaluated by Bisht *et al.* (2015) who reported

Table 2: Evaluation of Indian mustard varieties/ accessions against white rust in field conditions at Bharatpur and New Delhi

Varieties/accessions	% Disease severity*		Disease reaction	
	Bharatpur	New Delhi	Bharatpur	New Delhi
PBY-2	28.9 (32.4)	29.5 (32.9)	S	S
B-394	15.6 (23.3)	19.3 (26.0)	MR	MR
BJ-24-5	11.1 (19.4)	13.2 (21.2)	MR	MR
DYJ-1		4.4 (12.1)	0.0 (0.0)	HRI
AJ-34	0.0(0.0)	0.0 (0.0)	I	I
AJ-59	37.8 (37.9)	45.4 (42.3)	S	S
PBY-1	8.9 (17.3)	8.5 (16.9)	R	R
PBY-3	6.7 (14.9)	6.5 (14.6)	R	R
MP-7	33.3 (35.2)	41.6 (40.1)	S	S
Heera	0.0 (0.0)	0.0 (0.0)	I	I
K-9284	11.1 (19.4)	11.1 (19.4)	MR	MR
27049	42.2 (40.4)	35.8 (36.7)	S	S
252025	0.0 (0.0)	0.0 (0.0)	I	I
DJ-5	40.0 (39.2)	35.3 (36.4)	S	S
PBR-357	33.3 (35.2)	35.1 (36.3)	S	S
RE-14	33.3 (35.2)	33.2 (35.2)	S	S
EJ-19	15.6 (23.2)	12.8 (20.9)	MR	MR
DTM-34	0.0 (0.0)	0.0 (0.0)	I	I
IM-39	15.5 (23.2)	16.4 (23.9)	MR	MR
IM-59	20.0 (26.5)	23.8 (29.1)	MR	MR
IM-97	37.8 (37.9)	39.2 (38.7)	S	S
IM-87	42.2 (40.5)	45.6 (42.5)	S	S
IM-127	11.1 (19.4)	9.6 (18.0)	MR	R
IM-152	1.8 (6.82)	0.0 (0.0)	HR	I
PRG-908(Y)	42.2 (40.5)	51.7 (45.7)	S	HS
Donskaja-IV	0.0 (0.0)	0.0 (0.0)	I	I
ATC-94394	15.6 (23.2)	23.0 (28.5)	MR	MR
DRMR-2019	46.7 (43.0)	53.0 (46.7)	S	HS
DRMR-2035	0.0 (0.0)	0.0 (0.0)	I	I
DRMRMJA-35	24.4 (29.6)	27.0 (31.0)	MR	S
DRMR 1-5	2.2 (8.5)	4.4 (12.1)	HR	HR
DRMRIJ 12-48	4.4 (12.1)	8.8 (17.9)	HR	R
DRMRIJ 12-40	2.2 (8.5)	0.0 (0.0)	HR	I
DRMR 2018-37	2.2 (8.5)	0.0 (0.0)	HR	I
BIO YSR	2.2 (8.57)	1.0 (4.7)	HR	HR
Basanti	0.0 (0.0)	0.0 (0.0)	I	I
Pusa Bold	64.4 (53.3)	71.8 (58.0)	HS	HS
Varuna	51.1 (45.6)	55.8 (48.3)	HS	HS
Pusa Karishma	22.2 (28.1)	11.10 (19.4)	MR	MR
Giriraj	73.3 (58.9)	89.3 (71.0)	HS	HS
Radhika	40.0 (39.2)	55.8 (48.4)	S	HS
Brijraj	60.0 (50.7)	67.4 (55.2)	HS	HS
DRMR 1165-40	46.7 (43.0)	39.8 (39.1)	S	S
DRMR 150-35	51.1 (45.6)	59.1 (50.2)	HS	HS
Kranti (do)	77.8 (61.8)	81.2 (65.2)	HS	HS
Pusa Mustard 27	51.1 (45.6)	55.9 (48.4)	HS	HS
Pusa Mustard 28	42.2 (40.5)	39.4 (38.8)	S	S
JM-2	31.1 (33.9)	33.4 (35.2)	S	S
JM-3	51.1 (45.6)	61.1 (51.4)	HS	HS
Rohini	8.9 (17.3)	9.2 (17.6)	R	R

Pusa Jaikisan	60.0 (50.7)	73.1 (58.8)	HS	HS
RH 1400	8.9 (17.3)	10.8 (19.1)	R	MR
Rohini WRR	4.4 (12.1)	3.7 (11.1)	HR	HR
Varuna WRR	6.7 (14.9)	5.2 (13.1)	HR	R
Pusa Jaikisan WRR	0.0 (0.0)	0.0 (0.0)	I	I
Pusa bold WRR	0.0 (0.0)	0.0 (0.0)	I	I
PHR-2	37.8 (37.9)	46.0 (42.7)	S	S
NRCHB-101 (SC)	82.2 (65.0)	88.4 (70.3)	HS	HS
DLSC-1 (RC)	0.0 (0.0)	1.4 (5.6)	I	HR
GSL-1 (RC)	0.0 (0.0)	0.0 (0.0)	I	I
CD (5%)	0.81	2.31		
CV	2.39	5.42		

* Values in parenthesis are arcsine transformed values. I: Immune; HR: Highly resistant; R: Resistant; MR: Moderately Resistant; S: Susceptible; HS: Highly

multiple disease resistance with 5-25% disease severity in seven lines *viz.,* IC-255498, IC-296685, IC- 326253, IC-335847, IC-339589, IC-339597 and IC- 417020 and IC-401570 had zero disease severity. Ravi and Awasthi (2016) evaluated 30 germplasms, out of which DRMRIJ 12-37 and NDRE–08-14-01 were recorded as immune. DRMRIJ 12-41 and DRMR 12-03 were found to be resistant. Gairola and Tewari (2017) evaluated thirty germplasm, out of which DRMRIJ 12-37, RH 1234 and NDRE-08-14-01 were reported immune and DRMRIJ 12-41, DRMRJA 35 and DRMRIJ 12-03 were found to be resistant. Yadav *et al.* (2018) screened 2000 Indian mustard accessions against white rust. Out of 2000 germplasm accessions, 168, 46 and 185 accessions were found resistant at Ludhiana, Pantnagar, and Hissar, respectively but only 27 were identified as resistant across the locations indicating the pathogenic variability at different locations. These 27 accessions were further validated under artificial inoculation and eight of them, namely, IC265495, IC313380, EC766091, EC766133, EC766134, EC766192, EC766230 and EC766272 were identified as highly resistant with disease severity reaction (PDI = 0) to *A. candida* at both cotyledonary and true leaf stages. Arora *et al.* (2019) introgressed *BjuWRR1* located on *ACB1-A5.1* in Varuna, Rohini, Pusa Jaikisan and Pusa Bold and the developed NILs were r resistant to the 6 isolates tested against.

Fig. 1: Field screening of rapeseed mustard germplasms against *Albugo candida* at Bharatpur

Conclusion

Out of 57 genotypes evaluated, 11 were found immune, 09 highly resistant, 04 resistant and 10 moderately resistant, 26 were categorized to be susceptible to highly susceptible at Bharatpur and at New Delhi; 14 were immune, 04 were highly resistant, 06 were resistant, and 09 were moderately resistant. 27 genotypes were found to be susceptible to highly susceptible. Eight genotypes; AJ-34, 252025, Heera, Donskaja-IV, Basanti, Pusa Jaikisan WRR, Pusa Bold WRR, DTM-34, gave immune response at both the locations.

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