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Phenotypic screening of Indian mustard genotypes against white rust (*Albugo candida*) under artificial and natural epiphytotic conditions

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Abstract

Due to the variation among the various fungal races of *Albugo candida*, the known resistance genes against white rust disease are often inoperative. Therefore, to ensure durable resistance, new sources of resistance that are effective against various races are required. The present experiment was carried out during *Rabi* season of 2022-23 to screen the 208 Indian mustard germplasm for identification of white rust tolerant genotypes under artificial epiphytotic conditions with three fungal isolates *viz*., Morena, Bharatpur and Jhansi, and natural conditions. Results, revealed that, out of 208 germplasm accessions evaluated, twenty-two accessions showed resistance reaction (PDI: Percent disease index = 0 to 5 %) towards white rust disease under natural conditions. Out of these twenty-two accessions ten accessions *viz*., IC-320378, EC-766312, EC-766594, EC-766470, EC-766234, EC-766292, EC-766576, EC-766134, IC-766313 and DRMR-132 showed resistance reaction (PDI = 5-10 %) under artificial conditions against all the three fungal isolates. Whereas, twenty-three genotypes showed moderate resistance in natural conditions. Out of these twenty-three, ten accessions *viz*., DRMR-582, AJ-11, DTM-50, EC-766141, EC -766557, EC-766069, EC-766115, EC-766616, EC-766555 and IC-766029 showed moderately resistant reaction against three isolates under artificially inoculated conditions. Therefore, these novel white rust resistant sources could be used as potential donors for developing durable white rust resistant cultivars of Indian mustard.

Keywords: *Albugo candida, Brassica juncea, disease reaction, resistance, PDI*

Introduction

Brassica juncea, also known as Indian mustard, brown mustard, *Rai, Sarson, Raya* and *Laha*, is an allotetraploid species (AABB, 2*n* =2x=36) that originated in west and central Asia through natural hybridization of *Brassica rapa* (L.) (AA, 2*n* = 20) and *Brassica nigra* (L.) WDJ Koch (BB, 2*n* = 16) (Kang *et al.,* 2021; Nagaharu and Nagaharu, 1935). Oil content in different varieties show slight variation in percentage and it ranges from 35.0 to 42.0 %. Particularly, it has been demonstrated that fermented mustard exhibits a variety of health advantages and impacts on preventing diseases (Oh *et al.*, 2017). However, the oil extracted from these seeds contains a relatively high percentage (38-57 %) of erucic acid.

Several biotic and abiotic stresses effecting Indian mustard productivity, among all, white rust is majorly a devasting disease which affects the overall productivity of the crop. It is caused by a biotrophic oomycete *i.e., Albugo candida* (Pers.) Kuntze. Yield losses due to white rust in *B. juncea* can range from 23.0 to 89.8 % in India based on the disease severity (Lakra and Saharan, 1989). The phenotypical characteristics of the white rust disease include white blisters or pustules on the cotyledon, the abaxial leaf surface, the inflorescence, the base of the leaf petiole/stem and the growth of the stag head at a later stage of the plant. The pathogen infection prefers warm days, cool nights and additional soil moisture after a shower. Nearly all commercially available Indian mustard cultivar are prone to this disease. Additionally, the Indian gene pool of *B. juncea* is far more vulnerable to *A. candida* than the east European gene pool (Cheung *et al.,* 1998).

The pathogen used to survive from oospores generated in hypertrophied plant tissues that fall from diseased plants or seeds, as they can serve as a source of inoculum once they have germinated. Even in dry storage circumstances, the oospore can survive for more than twenty-one years in infected host tissues. Oospores naturally germinate when appropriate climatic conditions (temperature 10-20 °C and RH > 70 %) are present and either directly or indirectly lead to primary infection in the host leaves (by entering *via* stomata or natural openings). However, sporangia and/or zoospores are responsible for the secondary infection, which appears as pustules (Verma and Petrie, 1980). After emerging from mature and dehiscence pustules, the sporangia travel from one location to another *via* wind. Primarily, moisture on the surface of the host leaf is

necessary for the germination and infection processes of zoospores and sporangia. Later oospores are developed in hypertrophied plant tissues such as the roots, stems, leaves, flowers and even on the siliquae.

Most of the mustard growers use fungicides which ultimately affects the environment, as a common disease management practice. Use of resistant varieties is one of the cheapest and environmentally safe method to tackle this problem (Awasthi *et al.,* 2012). Therefore, searching for new resistance gene(s) is always a necessary basic and continuous process. In the unpredictable climatic change and global warming, inbuilt resistance became imperative to stabilize and sustain the yield potential of Indian mustard cultivars under different growing conditions in India.

Materials and Methods

The present experiment was carried out during the period from November 2022 to March 2023 at Research Farm, College of Agriculture, Rani Lakshmi Bai Central Agricultural University, Jhansi, Uttar Pradesh. The experimental material for this study comprised of 208 genotypes, obtained from ICAR-National Bureau of Plant Genetic Resources, New Delhi and ICAR-Directorate of Rapeseed-Mustard Research, Bharatpur (Rajasthan) were considered for screening against white rust disease along with ten checks *viz*., Bio-YSR, Pusa Karishma, DRMR-MJA-35, NRCHB 101, DRMR-150-35, Pusa Jaikisan, Varuna, Pusa Vijay, PM-24 and Rohini under both natural and artificial epiphytotic conditions whereas extra six checks *viz.,* RLC-3, Donskaja-1, Donskaja-2, PDZ-1, Heera and JM-2 were examined specially under artificial conditions.

Phenotyping for white rust resistance

Phenotyping under natural conditions: Indian Mustard germplasms were sown on November 16, 2022 in an augmented block design. Each germplasm was sown in 3-meter-long row with spacing 30 × 10 cm. Susceptible checks were grown after every few rows as an infector row. The incidence of the white rust disease has been observed and documented during vegetative stage of the crop. For the purpose of calculating the Percent Disease Index (PDI), observations on the incidence of white rust were made from ten randomly selected plants in each genotype before the crop reaching flowering stage using 0-9 scale (Mayee and Datar, 1986) as shown in Table 1. During initiation of disease, congenial environment was created for disease development by providing additional irrigation which helps in creating the humidity. PDI and stag head incidence is estimated as per below procedure.

Percent Disease Index (PDI): PDI was calculated to estimate the disease severity as per the formula given by Wheeler (1969).

PDI = (Sum of all numerical ratings/ No. of cotyledons or leaves scored × maximum grade of scale) × 100

Stag head incidence : Plants affected by stag head symptoms along with total number of plants were counted in each genotype. The stag head incidence was calculated with the help of following formula:

Staghead incidence (%) = (No. of plants showing staghead/ Total number of plants) × 100

Phenotyping for white rust resistance under artificial epiphytotic condition

Inoculum preparation, purification and inoculation: The fresh leaves of white rust effected mustard plants were collected from three locations *viz.*, Morena, Bharatpur and Jhansi. The pustules present on the infected leaves were scraped to prepare the sporangial suspension in distilled water. The sporangial suspension was then incubated at 4°C for 4 hours for the release of zoospores. After adjusting the sporangial concentration with distilled water, the seedlings were inoculated with three isolates at both cotyledonary and true leaf stage. The screening is done in two replications for all the three isolates separately for calculating the PDI. The inoculation was done during the month of January 2023. The inoculated seedlings were then transferred into humid chamber in order to maintain relative humidity, the trays were covered with a transparent thin polythene sheet (thickness of 700 gauge) tightly. The humid chamber was then covered with shade net in order to restrict the entry of direct sunlight in order to allow the disease to spread. This kept the plastic chamber dark. For the congenial white rust disease to develop in Indian mustard cultivars, low temperatures and high levels of humidity are necessary. Consequently, water (up to 2-3 cm height) was regularly supplied once in every 2-3 days to maintain the low temperature and high humidity within the polythene chamber.

Disease scoring : Disease scoring is performed 12-15 days after inoculation. The disease scoring was done using 0-9 scale as proposed by Mayee and Datar (1986) at both cotyledonary and true leaf. After scoring, the PDI calculated as per Wheeler (1969) formula, which indicated the percentage of host tissue or plant part covered by lesions or symptoms or damaged by the disease. Disease incidence is the number or proportions of plant units that are diseased (i.e., plants, leaves, flowers etc.) in relation to the total number of plant units examined. The entire procedure right from inoculum collection and disease phenotyping is depicted in Fig. 1.

Results and Discussion

Disease reactions and percent disease index (PDI) of

Table 1: Rating scale (0-9) was used for measuring disease severity of white rust disease at cotyledonary and true leaf stage in Indian mustard

Rating scale	Cotyledonary stage	True leaf stage	PDI (%)	Disease reaction
0	No symptoms on abaxial (lower) and adaxial (upper) leaf surfaces	Absence of pustules	0	Highly resistant (HR)
1	Minute pinpoint to larger brown necrotic flecks under inoculation point on the cotyledonary leaves	<5% leaf area covered by pustules	<5	Resistant (R)
3	Few pustules on abaxial surface and absence of any pustules on adaxial leaf surface	5-10% leaf area covered by pustules	5-10	Moderately resistant (MR)
5	Few to many dispersed pustules with good sporulation on the abaxial surface, and 0 to few pustules on the adaxial surface	11-25% leaf area covered by pustules	11-25	Moderately susceptible (MS)
7	Several pustules with copious sporulation on the abaxial surface with none to few pustules on the adaxial surface	26-50% leaf area covered by pustules	26-50	Susceptible (S)
9	Countless large coalescing pustules on the abaxial surface with scarce to several pustules on the adaxial surface of the cotyledonary leaves	>50% leaf area covered by pustules	>50	Highly (HS) susceptible

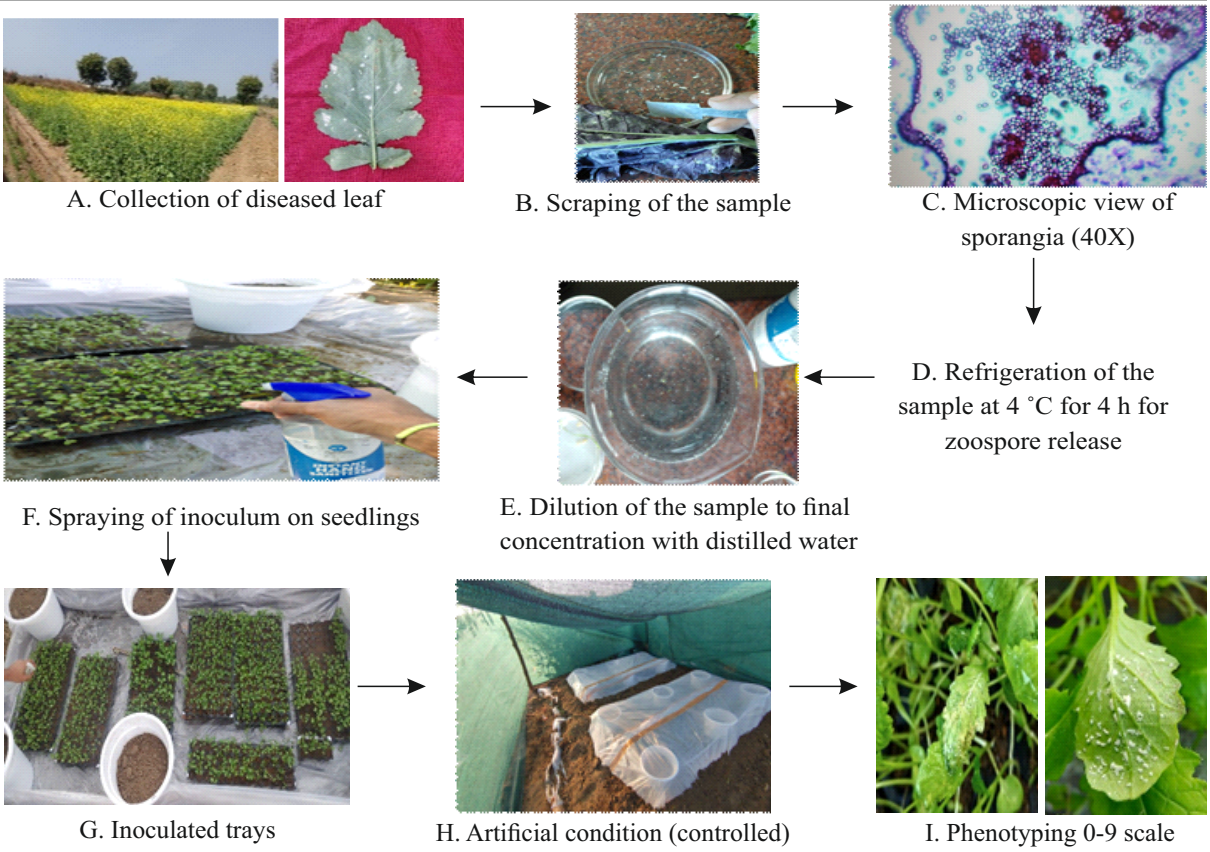

Fig. 1. Schematic illustration of phenotyping of white rust resistance under artificial epiphytotic condition

***Brassica* germplasm against white rust under natural conditions**

In the present study, 208 Indian mustard germplasm were screened against white rust under natural conditions during *Rabi* 2022-23. Out of 208 genotypes studied none of the genotypes were found highly resistant (PDI = 0 %). These results are in accordance with Shrivastava *et al*. (2023b) while screening 75 genotypes of Indian mustard reported that none of the genotypes showed immune reactions. Similarly, Magar *et al*. (2016) found no single genotypes as resistance out of eight genotypes studied. However, our results are not in the agreement with the findings of Chand *et al*. (2022) where they reported six Indian mustard genotypes as highly resistant against white rust while evaluating 25 mustard genotypes with same disease indexing parameters. Twenty-two genotypes *viz*., DRMR-132, IC-766313, EC766134, IC-320378, EC-766141, EC-766576, EC-766292, EC-766234, EC-766069, EC-766470, EC-766594, EC-766555, EC-766312, DRMR-136, DRMR-611, DRMR-692, DRMR-710, RC-371, RCQR-9901, EC-766204, EC-766325 and IC-766214 showed resistant reaction with minimum disease severity (PDI= 0-5 %). The genotypes which showed moderately resistant (PDI= 5-10 %) are DRMR-582, AJ-11, DTM-50, EC-766557, EC-766115, EC-766616, IC-766029, DRMR-360, DRMR-624, DRMR-906, RC-371-1, EM-1, IM-108, PM-67, IC-341108, IC-70302, EC-766234, EC-766557, EC-399308, EC-766498, EC-766489, IC-766029 & DRMR-265. The highly susceptible reaction was observed in the genotypes *viz*., DRMR-13, DRMR-46, DRMR-578, DRMR-680, RC-132, NC-37362, CN-34005, CN-113780, DRMR-2, DRMR-690, IM-170, NDRE-4, EC-766211, DRMR-701, DRMR-904, DRMR-708, DRMR-743 & DRMR-57 with maximum disease severity (PDI= >50 %). Remaining genotypes showed moderately susceptible (PDI= 11-25 %) to susceptible reaction (PDI= 25-50 %) which are shown in the Table 2. Similar findings were reported by while screening 240 germplasm accessions of Indian mustard found IC-326253 and IC-417020 resistant with 5-10 % disease severity. Similarly, Gaur and Meena (2016) reported nine germplasm lines with disease severity less than 5 %. The other findings were done by in which they reported that out of eight varieties/cultivars two were moderately resistant (Jagannath and PCR-10), four were moderately susceptible (Jai kisan, MAUC-7, RH-8812 and RH-30) and two were susceptible (Pusa bold and Varuna) to white rust disease.

Stag head incidence

The staghead incidence indicates the number of plants effected by staghead infection. The staghead incidence was recorded when the crop reached at seed formation stage. Among all genotypes, 95 genotypes got infected with staghead. The maximum staghead incidence was recorded in DRMR-704 (47.8 %) followed by DRMR-689 (40.0 %). It was observed that, genotypes which were showing resistant reaction towards white rust during vegetative stage, showing staghead symptoms. Whereas, the genotypes which were showing highly susceptible reaction got no symptoms of staghead. So, from these observations we can say that there is no association between disease severity and staghead incidence. Similar findings were also reported by they concluded that there is no relationship between foliar disease and staghead incidence across the genotypes tested.

Disease reactions and PDI of *Brassica* germplasm against white rust under artificial epiphytotic conditions

In artificial epiphytotic conditions, 208 genotypes were screened against three white rust isolates collected from Morena, Bharatpur and Jhansi for identifying the variable disease reactions among different isolates and also to confirm the resistance among the genotypes which were screened under natural conditions. The disease scores were recorded at cotyledonary stage and true leaf stage.

In case of Morena isolate, out of 208 genotypes screened three genotypes were found highly resistant against white rust, thirty-six genotypes were found resistant, eighty-two genotypes showed moderately resistant reaction at cotyledonary stage of the seedling and remaining genotypes were shown moderately susceptible to highly susceptible reaction. At true leaf stage it was found that out of 208 genotypes screened, seven genotypes showed highly resistant reaction, seventeen genotypes were resistant and thirty-seven genotypes were found moderately resistant. Remaining genotypes exhibited moderately susceptible to highly susceptible reaction. Similar findings were reported by Yadav *et al*. (2019).

In case of Bharatpur isolate, thirteen genotypes were found highly resistant against white rust, fifty-four genotypes were found resistant and sixty-two genotypes showed moderately resistant reaction at cotyledonary stage of the seedling and remaining genotypes showed moderately susceptible to highly susceptible reactions. At true leaf stage it was found that out of 208 genotypes screened, twenty-five genotypes showed highly resistant reaction, twenty-four genotypes were resistant and forty-six genotypes were found moderately resistant. Remaining genotypes exhibited moderately susceptible to highly susceptible reaction.

For Jhansi isolate, eight genotypes were found highly resistant against white rust, forty-eight genotypes were

Table 2: List of Indian mustard genotypes showing variable disease reactions to white rust under natural condition

Rating scale	Disease reaction	Percent disease index (PDI) (%)	Genotypes
0	Highly resistant (HR)	0	None of the genotypes
1	Resistant (R)	<5%	DRMR-132, IC-766313, EC766134, IC-320378, EC-766141, EC-766576, EC-766292, EC-766234, EC-766069, EC-766470, EC-766594, EC-766555, EC-766312, DRMR-136, DRMR-611, DRMR-692, DRMR-710, RC-371, RCQR-9901, EC-766204, EC-766325 & IC-766214
3	Moderately resistant (MR)	5-10	DRMR-582, AJ-11, DTM-50, EC-766557, EC-766115, EC-766616, IC-766029, DRMR-360, DRMR-624, DRMR-906, RC-371-1, EM-1, IM-108, PM-67, IC-341108, IC-70302, EC-766234, EC-766557, EC-399308, EC-766498, EC-766489, IC-766029 & DRMR-265.
5	Moderately susceptible (MS)	11-25	DRMR-5, DRMR-6, DRMR-9, DRMR-33, DRMR-357, DRMR-361, DRMR-362, DRMR-579, DRMR-610, DRMR-625, DRMR-909, Basanti, RUHR-2-1, RC-132, DRMR-736, DRMR-38, CN-105364, CN-101813, CN-105233, DRMR-56, DJ-12, KDM-49-1, RB-60, PR-2001-42, RGN-34, RNN-505, I-79, IC-206741, IC-589676, IC-589683, IC-49704, IC-422256, IC-481011, EC-766110, EC-766402, DRMR-685, EC-766560, EC-766276, EC-766124, DRMR-707, EC-399293, EC-571648, EC-766564, DRMR-704, EC-766322 & IC-766129.
7	Susceptible (S)	26-50	DRMR-4, DRMR-7, DRMR-11, DRMR-12, DRMR-48, DRMR-137, DRMR-897, DRMR-50, DRMR-47, DRMR-358, DRMR-359, DRMR-364, DRMR-574, DRMR- 576, DRMR-577, DRMR-626, DRMR-55, DRMR-683, DRMR-55, DRMR-683, DRMR-686, DRMR-689, DRMR-691, DRMR-729, DRMR-711, DRMR-588, DRMR-731, DRMR-900, DRMR-732, Pusa Bahar, RC891-1, DRMR-684, NC-660, CN-105257, CN-105312, CN-105309, DRMR-8, CN-34008, CN-105379, AJ-3, DJ-65, RJ-10, DRMR-703, PTJ-3-64, IM-76, IM-152, IM-3, PBR-97, RE-44, RH-30, NPJ-113, PCR-9403, PM-30, CN-101845, IC-122137, IC-11721, IC-372259, DRMR-682, IC-491485, IC-531377, IC-491348, IC-375924, IC-422156, IC-73225, EC-399300, EC-766423, EC-766127, EC-766136, DRMR-586, EC-766611, EC-766571,EC-766391, EC-766381, EC-766335, IC-766334, DRMR-135, EC-766311, EC-766310, EC-766296, EC-766278, EC-766270, EC-766207, EC-766060, EC-766487, EC-766344, EC-766576, EC-399318, DRMR-581, EC-764501, EC-766622, EC-766393, EC-766095, EC-564649-1, IC-766098, IC-766614, IC-766299, DRMR-730, IC-321424, DRMR-706, DRMR-712 & DRMR-45
9	Highly susceptible (HS)	>50	DRMR-13, DRMR-46, DRMR-578, DRMR-680, RC-132, NC-37362, CN-34005, CN-113780, DRMR-2, DRMR-690, IM-170, NDRE-4, EC-766211, DRMR-701, DRMR-904, DRMR-708, DRMR-743 & DRMR-57

0 (0%) HR
-

1(<5%) R
IC-320378

3(5-10%) MR
DRMR-582

5(11-25%) MS
DRMR - 5

7(26-50%) S
DRMR - 7

9(>50%) HS
DRMR - 13

Fig. 2: Leaves of *Brassica juncea* infected by pustules of white rust disease caused by *A. candida* rated from 0-9 scale under natural conditions

(a) (b)

Fig. 3: Comparison between leaves of resistant check, BIOYSR (a) and highly susceptible check, Rohini (b)

found resistant and ninety-one genotypes showed moderately resistant reaction at cotyledonary stage of the seedling. At true leaf stage it was found that out of 208 genotypes screened, twenty-eight genotypes showed highly resistant reaction, sixty genotypes were resistant and eighty-nine genotypes were found moderately resistant. Remaining genotypes exhibited moderately susceptible to highly susceptible reaction.

Under artificial conditions, disease phenotyping observations indicated that out of twenty-two genotypes which were found resistant in natural conditions, only ten genotypes *viz*., IC-320378, EC-766312, EC-766594, EC-766470, EC-766234, EC-766292, EC-766576, EC-766134, IC-766313 and DRMR-132 showed resistant reaction in all three fungal isolates of Morena, Bharatpur and Jhansi with PDI ranging from 0 to 4.8 and results are presented in Table 3.

From the above data it was found that out of 208 germplasm accessions evaluated, it was found that ten accessions *viz*., IC-320378, EC-766312, EC-766594, EC-766470, EC-766234, EC-766292, EC-766576, EC-766134, IC-766313 and DRMR-132 showed resistance reaction (PDI=0 to 5 %) towards white rust disease under both natural and artificial conditions and ten accessions *viz.,* DRMR-582, AJ-11, DTM-50, EC-766141, EC-766557, EC-766069, EC-766115, EC-766616, EC-766555 and IC-766029 showed moderately resistance reaction (PDI=5-10 %) under both natural and artificially inoculated conditions. Here, most of the genotypes which showed resistant to moderately resistant reactions in field conditions failed to show same reactions in artificially inoculated conditions rather they showed moderately to highly susceptible reactions. So, from these observations it is clear that studies under artificial epiphytotics are necessary in order to confirm the resistance of the genotypes. These results are in accordance with Gairola and Tewari (2017). Similar

Table 3: PDI and disease reaction for white rust resistant genotypes of *Brassica juncea* under natural and artificial conditions

Genotypes	Artificial condition												Natural condition	
	Morena				Bharatpur				Jhansi					
	Cotyledonary stage		True leaf stage		Cotyledonary stage		True leaf stage		Cotyledonary stage		True leaf stage		PDI	DR
	PDI	DR	PDI	DR	PDI	DR	PDI	DR	PDI	DR	PDI	DR		
DRMR-132	4.4	R	2.2	R	4.4	R	4.4	R	0	HR	0	HR	4.8	R
IC - 766313	2.2	R	1.5	R	4.4	R	2.2	R	4.4	R	4.4	R	4.8	R
EC -766134	4.4	R	4.4	R	3.1	R	4.4	R	2.2	R	2.2	R	3.2	R
EC -766576	4.4	R	2.2	R	4.4	R	4.4	R	4.4	R	0	HR	3.2	R
EC - 766292	2.2	R	0	HR	0	HR	0	HR	2.2	R	0	HR	3.2	R
EC - 766234	4.4	R	4.7	R	4.4	R	0	HR	2.2	R	2.2	R	3.2	R
EC - 766470	4.4	R	2.2	R	0	HR	0	HR	4.4	R	0	HR	1.6	R
EC - 766594	4.4	R	4.4	R	4.4	R	2.2	R	4.4	R	2.2	R	4.8	R
EC - 766312	4.4	R	4.4	R	4.4	R	3.1	R	4.4	R	2.2	R	3.2	R
IC - 320378	4.4	R	0	HR	4.4	R	0	HR	4.4	R	0	HR	4.8	R

Similarly, out of twenty-three genotypes which were shown moderate resistance in natural conditions only ten genotypes *viz*., DRMR-582, AJ-11, DTM-50, EC-766141, EC-766557, EC-766069, EC-766115, EC-766616, EC-766555 and IC-766029 were found moderately resistant (PDI = 5-10 %). The results are shown in Table 4**.**

Table 4: PDI and disease reaction for white rust moderately resistant genotypes of *Brassica juncea* under natural and artificial conditions

Genotypes	Artificial condition												Natural condition	
	Morena				Bharatpur				Jhansi					
	Cotyledonary stage		True leaf stage		Cotyledonary stage		True leaf stage		Cotyledonary stage		True leaf stage		PDI	DR
	PDI	DR	PDI	DR	PDI	DR	PDI	DR	PDI	DR	PDI	DR		
DRMR -582	8.8	MR	8.8	MR	0	HR	0	HR	8.8	MR	4.4	R	7.9	MR
AJ - 11	6.6	MR	8.8	MR	4.4	R	4.4	R	8.8	MR	0	HR	9.5	MR
DTM - 50	0	HR	0	HR	8.8	MR	8.8	MR	4.4	R	4.4	R	9.5	MR
EC -766141	8.8	MR	7.9	MR	4.4	R	0	HR	8.8	MR	8.8	MR	3.2	R
EC - 766557	4.4	R	2.2	R	4.4	R	2.2	R	2.2	R	0	HR	9.5	MR
EC - 766069	6.6	MR	7.9	MR	8.8	MR	0	HR	8.8	MR	4.4	R	3.2	R
EC - 766115	4.4	R	2.2	R	8.8	MR	8.8	MR	0	HR	0	HR	6.3	MR
EC - 766616	4.4	R	2.2	R	4.4	R	0	HR	4.4	R	0	HR	6.3	MR
EC - 766555	8.8	MR	7.9	MR	4.4	R	2.2	R	4.4	R	8.8	MR	3.2	R
IC -766029	0	HR	0	HR	4.4	R	4.4	R	6.6	MR	2.2	R	9.5	MR

mustard were conducted by Bisht *et al*. (2016) where they reported seven germplasm showing immune reaction while evaluating 70 germplasm of Indian mustard. Other findings like Yadav *et al*. (2017) reported eight accessions *viz.,* IC313380, IC265495, EC766091, EC766133, EC766272, EC766134, EC766192 and EC766230 as highly resistant under artificially inoculated conditions. Chand *et al.* (2022) reported six genotypes of Indian mustard as highly resistant while working on 25 genotypes.

Conclusion

In present investigation, 208 germplasm of *B. juncea* were screened out for white rust resistance under both natural and artificial epiphytotic conditions. Among these only ten genotypes *viz*., IC-320378, EC-766312,

0 (0%) HR DTM - 50 | 1(<5%) R IC - 320378 | 3(5-10%) MR DRMR -582 | 5(11-25%) MS DRMR - 11 | 7(26-50%) S EC - 766555 | 9(>50%) HS IC - 589683

Fig. 4: Leaves of *Brassica juncea* infected by pustules of white rust disease caused by *A. candida* rated from 0-9 scale under artificial conditions at cotyledonary stage

0 (0%) HR DTM - 50 | 1(<5%) R EC - 766312 | 3(5-10%) MR DRMR - 576 | 5(11-25%) MS DRMR - 4 | 7(26-50%) S DRMR-50 | 9(>50%) HS DRMR - 8

Fig. 5: Leaves of *Brassica juncea* infected by pustules of white rust disease caused by *A. candida* rated from 0-9 scale under artificial conditions at true leaf stage

EC-766594, EC- 766470, EC-766234, EC-766292, EC-766576, EC-766134, IC-766313 and DRMR-132 were found resistant in both the conditions. Whereas ten genotypes *viz.,* DRMR-582, AJ-11, DTM-50, EC-766141, EC-766557, EC-766069, EC-766115, EC-766616, EC-766555 and IC-766029 were found moderately resistant. The contrasting genotypes (HR like DTM-50 and HS like DRMR-8) can be used to develop mapping population to identify and map genes /QTLs responsible for imparting resistance in Indian mustard.

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