Biological Forum – An International Journal 15(4): 268-272(2023)

ISSN No. (Print): 0975-1130
ISSN No. (Online): 2249-3239

Screening of Indian Mustard Genotypes against White Rust Disease based on Disease Indexing

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(Received: 09 February 2023; Revised: 09 March 2023; Accepted: 15 March 2023; Published: 20 April 2023)
(Published by Research Trend)

ABSTRACT: Mustard is an important oilseed crop in India. Various biotic factors are responsible for yield reduction as well as seed quality in mustard. Among different biotic stresses, white rust causes yield loss and quality in mustard up to a great extent. White rust not only degrades seed quality but also significantly lowers its oil content. As it is distinguished that, among various disease management tactics, use of resistant varieties is the best choice owing to cost-effectiveness and environment friendly approach. However, till now only few resistant sources against this disease has been reported. Therefore, in the present investigation, 75 Indian mustard genotypes have been evaluated in field under epiphytotic conditions during *Rabi* 2021-22. Some of the genotypes showed resistance against white rust disease. These resistance sources may be helpful to develop superior cultivar (s) for managing white rust disease where mustard cultivation is dominant.

Keywords: Resistance, White rust, disease indexing, biotic stress, Indian mustard.

INTRODUCTION

Indian mustard (*Brassica juncea* L. Czern. & Coss) is the most imperative crop of oilseed Brassica group, which is a natural amphidiploid (2n = 36, AABB genome), often cross-pollinated and with genome size of 920 Mb (Barfa *et al*., 2017; Shyam and Tripathi 2019; Baghel *et al*., 2020; Rajpoot *et al*., 2020; Sharma *et al*., 2022; Yadava *et al*., 2022). It is being grown around the globe for its oil, condiment along with for leafy vegetable in some parts of the world (Shyam *et al*., 2020; Shyam *et al*., 2021a; Sharma *et al.,* 2022). It is the most important oilseed crop of India having significant economic, nutritional, and industrial applications (Tripathi *et al.,* 2015; Thakur *et al.,* 2020). It is the most significant and widely cultivated species of rapeseed mustard crops in India, accounting for 90% of the crop's area (9.168 million ha) and production (11.75 MT), with a productivity of 1178 kgha^{-1} in 2021–2022 (Ministry of Agriculture and Farmers Welfare, GoI (2022).

The vulnerability of crop plants including Indian mustard to various biotic (Verma *et al.,* 2021; Tripathi *et al.,* 2022; Makwana *et al*., 2023), abiotic stresses (Asati *et al.,* 2022; Yadav *et al.,* 2022a; Yadav *et al.,* 2022b), nutritional quality (Shyam *et al.,* 2021b; Shyam *et al.,* 2021c; Shyam *et al.,* 2022a; Shyam *et al.,* 2022b; Shyam *et al.,* 2022c; Tomar *et al*., 2022) and presence of low levels of genetic diversity in the population (Rajpoot *et al.,* 2022; Shyam *et al.,* 2021d; Shyam *et al.,* 2022d; Ningwal *et al*., 2023) are the major drawbacks for its improvement. This is a thoughtful anxiety for breeding as higher genetic variability guarantees better selections and supports in realizing genetic gains. Moreover, the identification and selection of genetically assorted parents are the most vivacious criteria for hybrid breeding programmes (Banga *et al.*, 2015).

White rust is caused by a biotrophic oomycetes *Albugo candida* (Pers.) Kuntze in mustard and this disease is a dreadful disease of Indian mustard (Kamoun *et al.,* 2015; Behera *et al*., 2016). Phenotypically, it is characterized by the presence of distinct white blister/pustules on cotyledon, inflorescence, pustules on abaxial side of leaf and the base of leaf petiole/stem. In severe infestation there is formation of the stag-head on plant. The conditions which are congenial for pathogen is cold night, warm days, and rains. According to the findings of Awasthi *et al*. (2012) almost all the commercially released Indian mustard varieties are susceptible to white rust disease. Depending upon disease severity and environmental conditions during the season, the yield loss varies from 10-70% and may sometimes reaches upto 90% due to prevailing favourable conditions for disease to occur (Lakra and

Saharan 1989). Moreover, Indian gene pool of *B. juncea* is highly susceptible to *A. candida* as compared to the east European gene pool (Awasthi *et al.,* 2012).

As a common disease management practice, use of fungicides has been followed by most of the mustard growers, which ultimately affects the environment. However, it is of a well-known fact that the availability of resistant varieties is one of the cheapest and environment friendly options. Therefore, the present investigation was carried out with the objectives to screen Indian mustard genotype (s) by means of disease indexing under field conditions against white rust disease.

MATERIALS AND METHOD

The current investigation was undertaken on a total of 75 Indian mustard genotypes (1) acquired from the Zonal Agricultural Research Station, Morena, Rajmata Vijayaraje Scindia Krishi Vishwa Vidyalaya (RVSKVV), Gwalior, M.P., India (AICRP on Rapeseed and Mustard). All the genotypes were grown in randomized block design with two replications in Rabi 2021 at the experimental field of Department of Genetics & Plant Breeding, College of Agriculture, RVSKVV, Gwalior, India. Each genotype was planted in a plot of one row of 2-meter length with an arrangement of 30 cm apart between rows and 15 cm plant to plant. The observation on incidence of white rust disease was monitored and documented. Observations on the occurrence of white rust for analyzing the percent disease index (PDI) were taken from 10 randomly selected plants in each line of each block at 8-day intervals during the vegetative as well as true leaf stage, *i.e.,* 42 days after sowing (DAS) under natural field conditions. The disease incidence was recorded following a 0–9 scale as following Table 1.

Table 1: Disease rating scale for white rust in rapeseed mustard on leaf.

Rating Scale	Leaf area covered (%)	Disease Reaction
0	0	Immune
1	<5%	Highly Resistant (HR)
3	5-10%	Resistant (R)
5	11-25%	Moderately Resistant (MR)
7	26-50%	Susceptible (S)
9	>50%	Highly Susceptible (HS)

The intensity was calculated with the help of formulae

$$PDI = \frac{\text{Sum of total numerical rating}}{\text{Total no. of observation}} \times \frac{100}{\text{Max. grade}}$$

RESULTS AND DISCUSSION

During *Rabi* 2021-22, screening of 75 Indian mustard genotypes was carried out against white rust disease. Out of 75 genotypes screened against white rust, none of the genotype was found free from white rust. However, our results are not in the agreement with the findings of Chand *et al.* (2022) where they reported six mustard genotypes as highly resistant against white rust while evaluating 25 mustard genotypes with same disease indexing parameters. In some of the other studies, including Singh *et al.* (2021) found 12 genotypes, of *B. juncea* having immune type response at the cotyledonary stage. Similarly, in a separate investigation, five genotypes of *B. juncea* were reported highly resistant under field conditions. In our study, three mustard genotypes *viz.*, WRR-15, WRR-25 and JMWR-908-1 showed resistance (R) reaction with 5.55% disease incidence. However, 28 Indian mustard germplasm accessions with resistance reactions were reported by Yadav *et al.* (2018) in screening at Hisar, Ludhiana, and Pantnagar under field conditions. Eight of these were found to be highly resistant to the "Delhi isolate" of *A. candida* at the cotyledonary and true leaf stages under artificial circumstances.

In our Investigation, twenty-six mustard genotypes were found moderately resistant including Vasundhara, Pusa Jagannath, Kiran, PM-27, JMM-991, WRR-5, WRR-7, WRR-11, WRR-12, WRR-14, WRR-16, WRR-17, WRR-19, WRR-26, WRR-27, WRR-29, WRR-31, WRR-32 Maya, L-4, China, GSL-1, GSC-7, PC-5, PC-6 and RP-9. It was also found that most of the genotypes were susceptible including RB-50, Pusa Bold, Rohini, RH-725, Vardan, Swarn Jyoti, PusaJaiKisan, Albeli, Sej-2, Shraddha, DMH 1, RGN-73, NRC-HB-101, RVM-3, RH-749, NRC DR-2, JTC-1,JM-1, JM-2, JM-3, RVM-2, PM-26, PM-30, RMM-10-01-01, RMM-12-01-18, WRR-6, WRR-8, WRR-9, WRR-10, WRR-13, WRR-18, WRR-20, WRR-21, WRR-22, WRR-28 and WRR-30 against white rust. Whilst ten genotypes displayed highly susceptible reactions namely: Varuna, Kranti, PM-25, DRMR IJ-31, RVM-1, PM-28, Pusa Vijay, JMM-927 and RMM-12-03-18 under field conditions as PDI was more than 50% (Table 2-3 and Fig. 1).

The spread of the *A. candida* is influenced by several variables; including the aggressiveness of a race, the quantity of initial inoculums available, the timing of the disease's onset, and the local climate. The first kind is a localized infection of the white rust disease on leaves, while the second type is a systemic infection on inflorescence. A systemic infection causes abnormal inflorescence growth, distortion, and floral sterility, which are frequently referred to as a "stag head" shape created by hypertrophy and hyperplasia. In the present investigation among all the genotypes screened against white rust only three genotypes showed resistant reaction against the disease. The results are in accordance with Lakra and Saharan (1989); Gairola and Tewari (2017). However, Yadav and Singh (1999) screened 74 Indian mustard (*B. juncea*) germplasm lines for a resistance against white rust disease and none of the genotype was found to be resistant. Awasthi *et al.* (2012) reported that almost all the important varieties of *B. juncea* being grown in India were susceptible to white rust. The broad variety in how different genotypes react to pathogens in terms of susceptibility to disease may be caused by the varied expression of resistance gene(s) and genetic background of genotypes that influences genotype-pathogen interaction (Singh *et al*., 2021). The dynamics of host-pathogen interaction are greatly influenced by both macro and micro environment, which in turn has an impact on the severity of the disease (Tamang *et al.,* 2022).

Table 2: Genotypic response against white rust in a set of Indian mustard genotypes.

Sr. No.	Genotypes	White Rust		Sr. No.	Genotypes	White Rust	
		PDI	Reaction			PDI	Reaction
1.	RB-50	33.3333333	S	39.	PM-25	55.5555556	HS
2.	Pusa Bold	27.7777778	S	40.	PM-26	44.4444444	S
3.	Varuna	55.5555556	HS	41.	PM-27	22.2222222	MR
4.	Rohini	27.7777778	S	42.	PM-28	55.5555556	HS
5.	Kranti	55.5555556	HS	43.	PM-30	44.4444444	S
6.	RH- 725	33.3333333	S	44.	Pusa Vijay	55.5555556	HS
7.	Maya	16.666667	MR	45.	JMM-927	55.5555556	HS
8.	Vardan	27.7777778	S	46.	JMM-991	22.2222222	MR
9.	Vasundhara	16.6666667	MR	47.	RMM-10-01-01	44.4444444	S
10.	Swarn Jyoti	38.8888889	S	48.	RMM-12-01-18	44.4444444	S
11.	Pusa Jagannath	16.6666667	MR	49.	RMM-12-03-18	55.5555556	HS
12.	PusaJaiKisan	44.4444444	S	50.	WRR-5	16.6666667	MR
13.	Albeli	33.3333333	S	51.	WRR-6	33.3333333	S
14.	Sej-2	44.4444444	S	52.	WRR-7	22.2222222	MR
15.	Shraddha	38.8888889	S	53.	WRR-8	44.4444444	S
16.	DMH 1	33.3333333	S	54.	WRR-9	38.8888889	S
17.	L-4	22.2222222	MR	55.	WRR-10	33.3333333	S
18.	JMWR-908-1	5.55555556	R	56.	WRR-11	11.1111111	MR
19.	RGN-73	33.3333333	S	57.	WRR-12	16.6666667	MR
20.	NRC-HB-101	33.3333333	S	58.	WRR-13	27.7777778	S
21.	NRC-HB-506	22.2222222	MR	59.	WRR-14	22.2222222	MR
22.	RVM-3	44.4444444	S	60.	WRR-15	5.55555556	R
23.	RH-749	44.4444444	S	61.	WRR-16	11.1111111	MR
24.	NRC DR-2	44.4444444	S	62.	WRR-17	16.6666667	MR
25.	DRMR IJ-31	55.5555556	HS	63.	WRR-18	33.3333333	S
26.	CHINA	16.666667	MR	64.	WRR-19	22.2222222	MR
27.	GSL-1	22.2222222	MR	65.	WRR-20	44.4444444	S
28.	GSC-7	11.111111	MR	66.	WRR-21	38.8888889	S
29.	PC-5	5.55555556	R	67.	WRR-22	33.3333333	S
30.	PC-6	16.666667	MR	68.	WRR-25	5.55555556	R
31.	RP-9	11.111111	MR	69.	WRR-26	22.2222222	MR
32.	KIRAN	16.6666667	MR	70.	WRR-27	16.6666667	MR
33.	JTC-1	33.3333333	S	71.	WRR-28	27.7777778	S
34.	JM-1	44.4444444	S	72.	WRR-29	16.6666667	MR
35.	JM-2	33.3333333	S	73.	WRR-30	33.3333333	S
36.	JM-3	44.4444444	S	74.	WRR-31	22.2222222	MR
37.	RVM-1	66.6666667	HS	75.	WRR-32	16.6666667	MR
38.	RVM-2	33.3333333	S				

Table 3: Categorizations of reactions of Indian mustard genotypes against white rust.

Severity (%) category	Disease reaction	Numbers of genotypes	Name of genotypes
0	Immune	-	
<5	Highly Resistant	-	
5.0 – 10	Resistant	3	WRR-15, WRR-25, JMWR-908-1
10.1 – 25	Moderately Resistant	26	Vasundhara, Pusa Jagannath, Kiran, PM-27, JMM-991, WRR-5, WRR-7, WRR-11, WRR-12, WRR-14, WRR-16, WRR-17, WRR-19, WRR-26, WRR-27, WRR-29, WRR-31, WRR-32 Maya, L-4, China, GSL-1, GSC-7, PC-5, PC-6, RP-9
25.1 – 50	Susceptible	36	RB-50, Pusa Bold, Rohini, RH-725, Vardan, Swarn Jyoti, Pusa JaiKisan, Albeli, Sej-2, Shraddha, DMH1, RGN-73, NRC-HB-101, RVM-3, RH-749, NRC DR-2, JTC-1, JM-1, JM-2, JM-3, RVM-2, PM-26, PM-30, RMM-10-01-01, RMM-12-01-18, WRR-6, WRR-8, WRR-9, WRR-10, WRR-13, WRR-18, WRR-20, WRR-21, WRR-22, WRR-28, WRR-30
>50.1	Highly Susceptible	10	Varuna, Kranti, PM-25, DRMR IJ-31, RVM-1, PM-28, PM-25, Pusa Vijay, JMM-927, RMM-12-03-18

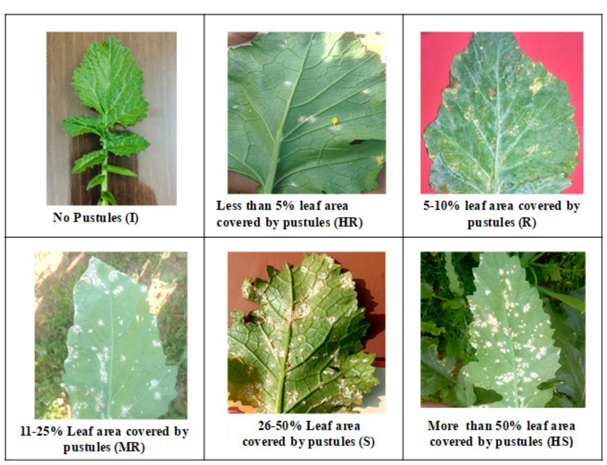

Fig. 1. Categorizations of reactions of Indian mustard genotypes against white rust.

CONCLUSIONS

It has been determined that the germplasm lines of Indian mustard exhibited resistant to moderately resistant response in field screening trial against white rust disease. It is possible that these germplasm lines could be used in future breeding programs to develop resistant cultivars, which could then be commercialized for cultivation in farmer's fields. In order to combat the constantly changing diseases, it is crucial to identify a variety of resistance genes in any crop species. With the long-term goal of diversifying the current cropping system, significant efforts have recently been made in India to horizontally increase the area under mustard cultivation in the non-traditional locations. Therefore, it is necessary to always have access to donor parents which have a high level of resistance against white rust disease.

FUTURE SCOPE

This goal could be achieved by conducting controlled laboratory tests on a wide collection of assorted genotypes/germplasm accessions for the disease. Moreover, it is required that resistance must be confirmed in glasshouse under controlled artificial inoculation conditions and employing disease resistant gene-linked molecular markers followed by testing under field conditions. as some times disease escaped and plant showed resistant reactions. As the pathogen needs more virulent genes to surpass the host's resistance level due to its low fitness and re-productivity, more resistance genes in the host would prevent the emergence of new pathogen races.

Conflict of Interest. None.

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How to cite this article: Aditi Shrivastava, M.K. Tripathi, Sushma Tiwari, Purnima Singh, Niraj Tripathi, Prakash Narayan Tiwari, Prerana Parihar, J. Singh and Shailja Chauhan (2023). Screening of Indian Mustard Genotypes against White Rust Disease based on Disease Indexing. *Biological Forum – An International Journal, 15*(4): 268-272.