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IDENTIFICATION OF SLOW RUSTING COMPONENTS IN MUSTARD AS A COMPONENT OF INTEGRATED DISEASE MANAGEMENT AGAINST WHITE RUST

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White rust caused by *Albugo candida* is an important disease affecting mustard crops globally. Continuous emergence of new races of the pathogen is responsible for breaking down of the resistance in the existing cultivars. So, there is a need for continuous evaluation of mustard cultivars to find resistant sources against the disease. In this regard, 38 mustard genotypes were evaluated for their resistance to white rust in field under natural epiphytotic conditions. Apparent rate of infection (r) and Area under disease progress curve (AUDPC) were calculated for each genotype. Among the 38 genotypes evaluated, three genotypes NPJ 257, PRO 5111 and Pusa MH 126 exhibited moderately resistant reaction with lower AUDPC and r values indicating slow rusting. As an eco-friendly and sustainable approach, the identification of moderately resistant and slow-rusting genotypes is crucial for developing mustard cultivars with enhanced white rust resistance, thereby reducing the excessive and indiscriminate use of fungicides.

Key words: White rust, mustard, genotypes, resistance, slow rusting

Introduction

Oilseeds play a crucial role in global agriculture and food security, serving as a vital source of edible oils, protein-rich meal and biofuels. Mustard (*Brassica juncea* L. Czern. and Coss.) holds a significant position in the global oilseed crop industry. The seeds contain 4.7-13 per cent linolenic acid, 27 per cent oleic acid and 10-12 per cent linoleic acid, which are essential components of a healthy human diet. The oil content of the seed ranges from 38-46 per cent. Moreover, they are rich in glucosinolates (180-200 micro moles) and erucic acid (38-

57%) (Kumar, 2012).

Despite its economic importance, mustard cultivation faces numerous challenges, including biotic and abiotic stresses. White rust caused by *Albugo candida* (Pers.) Kuntze is one of the important diseases of mustard and it appears in severe form in all major mustard growing areas of India (Kolte, 1985). It is world-wide distributed pathogen of mustard and also reported to attack number of host species belonging to family Brassicaceae (Choi *et al.*, 2007). The disease occurs in two phases namely local and systemic infection. Local infection is characterized by the presence of raised pustules on the underside of the leaves during flowering, later extending to stems, inflorescence and pods in severe cases. Systemic infection often leads to staghead formation (Meena *et al.*, 2014). The disease causes substantial yield losses which ranges from 17-34 per cent in India (Yadava *et al.*, 2011; Pandey *et al.*, 2013) and 20-90 per cent worldwide (Mishra *et al.*, 2009). The combined effect of local and systemic infection causes yield losses upto 89.9 per cent (Godika *et al.*, 2001).

Fungicides such as metalaxyl 4 per cent + mancozeb 64 per cent, mancozeb 75 per cent, metalaxyl 35 per cent and copper oxychloride 50 per cent are being used for management of white rust (Saharan, 1984; Gairola and Tewari, 2019). But the high cost of the chemicals and raising environmental concern is limiting the use of these fungicides. The best alternative is the use of resistant varieties which is simple, effective, environmentally friendly and economical. Varieties resistant to white rust of mustard were reported by several researchers (Upadhyay et al., 2021; Bisht et al., 2016) but due to emergence of new races of the pathogen, there is a chance of breakdown of resistance in the existing cultivars. Therefore, there is a need for continuous evaluation and identification of new genotypes resistant to white rust. Hence, the present study was undertaken with an objective to assess the level of resistance in different mustard genotypes against white rust using parameters such as area under disease progress curve (AUDPC) and apparent infection rate (r) which are used to compare the relative susceptibility or resistance of genotypes or varieties.

Materials and Methods

The field experiment was conducted at College of Agriculture, University of Agricultural Sciences, Dharwad, Karnataka to find out the sources of resistance to white rust of mustard. The material consisted a total of 38 genotypes of which 25 genotypes were collected from All India Coordinated Research Project (AICRP) on mustard and 13 genotypes from Baba Atomic Research Centre (BARC). Along with these genotypes a mustard variety Varuna was used as susceptible check. The experiment was conducted in alpha lattice design and each genotype was sown in five rows of 4.5 meters length with a spacing of 45 cm between the rows and 10 cm between the plants during the 2022-23 *rabi* season in the research plots of College of Agriculture, Dharwad, Karnataka, India.

During screening programme, all 38 genotypes along with susceptible check were screened for their reaction against white rust in field under natural epiphytotic conditions. The susceptible cultivar Varuna was sown after every ten test genotypes and all along the border. No fungicidal spray was given in order to allow the spread of the disease. The intensity of disease in the field was estimated from five randomly selected plants in each genotype which were tagged with labels. Observation on disease incidence were recorded at 45, 50, 55, 60 and 65 days after sowing by scoring five plants in each genotype using 0-9 rating scale (Conn *et al.*, 1990).

Per cent disease index (PDI) was calculated using a formula given by Wheeler (1969).

$$\frac{\text{Per cent disease}}{\text{index (PDI)}} = \frac{\text{Sum of all numerical rating}}{\text{No. of leaves examined } \times \text{Maximum disease grade}} \times 100$$

With disease observations at different time intervals, the apparent infection rate (r) and area under disease progress curve (AUDPC) for each genotype were calculated using the formulae given by Vander Plank (1963) and Wilcoxen *et al.*, (1975) respectively.

$$r = \frac{2.3}{t2 - t1} \left[\log \frac{X2}{1 - X2} - \log \frac{X1}{1 - X1} \right]$$

where,

r = Apparent rate of infection

 $X_1 =$ Disease severity at time t_1

 X_2 = Disease severity at time t_2

$$AUDPC = \sum_{i=1}^{k} \frac{1}{2} (Si + Si - 1) \times d$$

Where,

 $S_i = Disease$ severity at the end of the week i

 $S_{i-1} =$ Previous observation

k = No. of successive evaluation of disease

d = Interval between two evaluations

Results and discussion

The experimental material consisting of 38 entries along with susceptible check were evaluated for white rust disease under natural epiphytotic conditions during 2022-23 *rabi* season. The weather condition during crop growth period was highly favourable for disease development. The minimum temperature ranged from 13.7 °C to 16.5 °C and the maximum temperature ranged from 29.6 °C to 32.5 °C. The Relative humidity (%) ranged from 63.0 to 76.6 during the crop growth period. The disease appeared at very early stages and developed sufficiently during later stages of crop growth. The disease was scored using 0-9 scale at 5 days interval starting from the onset of disease and results pertaining to study are presented below.

SI. No.	Genotypes	Per cent Disease Incidence (PDI)					D		Mean	Yield
		45 DAS	50 DAS	55 DAS	60 DAS	65 DAS	Reactions	AUDPC	r value	(t/ha)
1	Pusa MH 126	11.11	13.33	15.56	20.00	24.44	MR	333.33	0.0026	1.54
2	BAUM 2022-1	15.56	22.22	26.67	37.78	48.89	S	594.48	0.0023	0.63
3	DRMRHT 1712	24.44	46.67	64.44	75.56	84.44	HS	1205.55	0.0015	1.02
4	ACN 247	20.00	28.89	35.56	40.00	44.44	S	683.35	0.0014	1.04
5	JD 6	17.78	33.33	57.78	73.33	82.22	HS	1072.20	0.0023	1.17
6	PRE 2018-1	42.22	64.44	75.56	86.67	91.11	HS	1466.68	0.0006	1.12
7	DRMRCI 156	20	26.67	31.11	35.56	46.67	S	633.38	0.0015	0.75
8	KMR (E) 22-2	46.67	68.89	84.44	88.89	93.33	HS	1561.10	0.0573	1.10
9	NPJ 257	6.67	8.89	11.11	13.33	17.78	MR	227.78	0.0052	1.74
10	TM 314-1	15.56	53.30	82.22	86.67	91.11	HS	1377.63	0.0028	0.94
11	PM 25	28.89	42.22	77.78	86.67	88.89	HS	1327.80	0.0012	1.43
12	SVJH72	24.44	28.89	46.67	66.67	75.56	HS	961.15	0.0014	2.02
13	RH2199-11	44.44	62.22	73.33	82.22	86.67	HS	1416.63	0.0006	1.18
14	DRMRSJ 272	40.00	64.44	77.78	91.11	95.56	HS	1505.55	0.0007	1.57
15	NPJ 258	33.33	51.11	75.56	82.22	88.89	HS	1350.00	0.0010	1.42
16	KMR (E) 22-1	28.89	46.67	64.44	75.56	84.44	HS	1216.68	0.0012	1.50
17	DRMRIJ 20 117	15.56	24.44	51.11	64.44	73.33	HS	922.18	0.0026	1.51
18	Q80623	20.00	26.67	33.33	40.00	48.89	S	672.23	0.0015	1.56
19	NRCHB 101	24.44	48.89	64.44	77.78	86.67	HS	1233.33	0.0015	1.37
20	ANDM 14-09	20.00	35.56	51.11	60.00	68.89	HS	955.58	0.0018	1.15
21	PRO 5111	8.89	11.11	13.33	15.56	22.22	MR	277.78	0.0005	1.40
22	HUJMI 21-1	20.00	24.44	44.44	68.89	77.78	HS	933.30	0.0019	1.45
23	RH 1997-37	22.22	33.33	55.56	75.56	84.44	HS	1088.90	0.0017	1.62
24	SHIVANI PLUS	26.67	55.56	64.44	75.56	86.67	HS	1261.15	0.0013	1.03
25	PRE 2020-14	28.89	46.67	60.00	71.11	82.22	HS	1166.68	0.0011	0.98
26	PM 25	20.00	44.44	66.67	75.56	82.22	HS	1188.90	0.0020	1.16
27	TM 301-3	26.67	48.89	71.11	80.00	86.67	HS	1283.35	0.0013	1.32
28	TM 310-1	15.56	26.67	48.89	62.22	73.33	HS	911.13	0.0026	1.21
29	TM 307-2	11.11	22.22	28.89	40.00	48.89	S	605.55	0.0037	1.28
30	TM 306-1	15.56	35.56	55.56	66.67	77.78	HS	1022.30	0.0027	1.60
31	TM 316	22.22	40.00	64.44	82.22	91.11	HS	1216.63	0.0017	1.07
32	TM 314	28.89	44.44	68.89	84.44	95.56	HS	1299.98	0.0012	1.02
33	TM 313	24.44	37.78	66.67	80.00	91.11	HS	1211.13	0.0015	1.31
34	TM 309-2	26.67	46.67	71.11	86.67	95.56	HS	1327.83	0.0014	1.00
35	TM 305-1	20.00	35.56	57.78	68.89	82.22	HS	1066.70	0.0020	1.64
36	TM 304-1	13.33	26.67	46.67	53.33	60.00	HS	816.68	0.0031	1.31
37	JD 6	17.78	31.11	53.33	60.00	77.78	HS	961.10	0.0022	0.97
38	NRCHB 101	13.33	28.89	48.89	57.78	73.33	HS	894.45	0.0032	1.13
39	Check (Varuna)	33.33	55.56	71.11	84.44	95.56	HS	1377.78	0.0010	0.95
MR - Moderately Resistant; S – Susceptible; HS – Highly Susceptible										

Table 1: Progress of white rust in different mustard genotypes.

Disease incidence

The results obtained revealed that the genotypes evaluated exhibited various levels of per cent disease incidence at different growth stages ranging from 6.67 at 45 DAS to 95.56 at 65 DAS. Out of the 38 genotypes evaluated, none of them showed immune or resistant reaction to white rust. However, three genotypes namely NPJ 257 (17.78 PDI), PRO 5111 (22.22 PDI) and Pusa MH 126 (24.44 PDI) showed moderately resistant reaction. Five genotypes *viz.*, BAUM 2022-1 (48.89 PDI), ACN 247 (44.44 PDI), DRMRCI 156 (46.67 PDI), Q80623 (48.89 PDI) and TM-307-2 (48.89 PDI) showed susceptible reaction. While, the remaining thirty genotypes showed highly susceptible reaction (Table 1, 2 & Fig. 2). Similar results were obtained by Awasthi *et al.*, (2012) who observed from his experiment that only a few lines

Disease grade	Disease reaction	Per cent leaf infection	Genotypes	No. of genotypes
0	Ι	0	-	Nil
1	HR	\$	-	Nil
3	R	6-10	-	Nil
5	MR	11-25	Pusa MH 126, NPJ 257, PRO 5111	3
7	S	26-50	BAUM-2022-1, ACN 247, DRMRCI-156, Q80623, TM-307-2	5
9	HS	>50	DRMRHT-1712, JD 6, PRE-2018-1, KMR (E) 22-2, TM 314-1, PM 25, SVJH-72, RH 2199-11, DRMRSJ 272, NPJ 258, KMR (E) 22-1, DRMRIJ 20-117, NRCHB-101, ANDM 14-09, HUJMI 21-1, RH 1997-37, SHIVANI PLUS, PRE-2020-14, PM-25, TM-301-3, TM-310-1, TM-306-1, TM-316, TM- 314, TM-313, TM-309-2, TM-305-1, TM-304-1, JD-6, NRCHB-101	30

Table 2: Reaction of different mustard genotypes against white rust.

were resistant and majority of the important varieties cultivated in India were susceptible to the white rust.

Area under disease progress curve (AUDPC)

The Area Under Disease Progress Curve is a crucial measure of disease severity over time. AUDPC values which are obtained by summation of values calculated at several intervals during disease progress are used for comparing slow rusting ability. The genotypes varied significantly in AUDPC values in all five observation dates. AUDPC values increased with time of observation in all the genotypes. The genotype KMR (E) 22-2 displayed the maximum AUDPC value of 1561.10, indicating early onset of disease with more initial disease incidence and faster disease progression leading to maximum terminal disease severity. Conversely, the genotypes NPJ 257, Pro 5111 and Pusa MH 126 have recorded the lowest AUDPC values of 227.78, 277.78 and 333.33 indicating lower disease severity and slower disease progression in this genotype though on set of disease was in the same week (Table 1). These variations in AUDPC values observed among the genotypes signified the differences in their susceptibility and ability

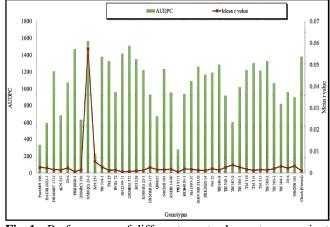


Fig. 1: Performance of different mustard genotypes against white rust.

to resist or tolerate the disease. The higher AUDPC values indicated susceptibility of the genotypes to the white rust disease whereas the lower AUDPC values indicated resistance in the genotypes evaluated (Table 1 & Fig. 1).

Apparent rate of infection (r)

The mean r-value ranged from 0.0005 to 0.0573 which represents the rate at which the disease spreads within a particular genotype, Genotype KMR (E) 22-2 had the highest mean r-value of 0.0573, suggesting a more rapid spread of the disease in this genotype, while PRO 5111 had the lowest infection rate of 0.0005, indicating a slower rate of disease progression in this genotype. These results

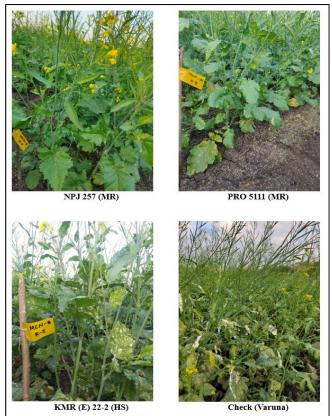


Fig. 2: Response of mustard genotypes to white rust.

indicate the variability in disease progression among the genotypes. These findings are in line with Upadhyay *et al.*, (2021), who observed a significantly low infection rate and AUDPC value in genotypes such as DRMRJ 127 indicating resistance to the disease (Table 1 & Fig. 1).

The cultivars with slow disease development are becoming popular nowadays in many crops. Slow rusting is known as the slow rate of development of white rust disease, indicated by a lower AUDPC and r value when compared to susceptible varieties under the same conditions. In the current study, the moderately resistant genotypes NPJ 257, PRO 5111 and Pusa MH 12 with lower AUDPC and r values were identified as slow rusters.

Conclusion

The results obtained from the current study on identification of slow rusting genotypes in mustard against white rust help us to understand the importance of screening and evaluation of genotypes for their reaction to white rust. The identification of moderately resistant, slow rusting genotypes such as NPJ 257, PRO 5111 and Pusa MH can pave the way for breeding programs aimed at developing resistant varieties. These genotypes could be used for farmers' adoption and also as sources of resistance in breeding program for varietal development. As the experiment was conducted for only one year, to confirm the disease reaction of the tested genotypes, verification testing at least for one more year is required.

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Conflict of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical approval

The submitted work is original and has not been published elsewhere in any form or language.

References

- Awasthi, R.P., Nashaat N.I., Kolte S.J., Tewari A.K., Meena P.D. and Bhatt R. (2012). Screening of putative resistant sources against Indian and exotic isolates of *Albugo* candida inciting white rust in rapeseed-mustard. J Oilseed Brassica., 3(1), 27-37.
- Bisht, K.S., Tewari A.K. and Upadhyay P. (2016). Screening of *Brassica* germplasm against *Albugo candida* (White rust disease) on *Brassica* species (Rapeseed-mustard).

Journal of Applied and Natural Science., 8(2), 658-662.

- Choi, Y.J, Shin H.D., Hong S.B. and Thines M. (2007). Morphological and molecular discrimination among *Albugo candida* materials infecting *Capsella bursa*pastoris world-wide. *Fungal Diversity.*, 27, 11-34.
- Conn, K.L., Tewari J.P. and Awasthi R.P. (1990). A disease assessment key for Alternaria black spot in rapeseed and mustard. *Can Pl Dis Surv.*, **70**, 19-22.
- Gairola, K. and Tewari A.K. (2019). Management of white rust (Albugo candida) in Indian mustard by fungicides and garlic extract. Pestic Res J., 31(1), 60-65.
- Godika, S., Pathak A.K. and Jain J.P. (2001). Integrated management of Alternaria blight (Alternaria brassicae) and white rust (Albugo candida) diseases of Indian mustard. Indian Journal of Agricultural Sciences., 71, 733-735.
- Kolte, S.J. (1985). Disease of Annual Edible Oilseed Crop: Rapeseed Mustard and Sesame Disease, CRC Press, Boca Raton, Florida, U.S.A.
- Kumar, A. (2012). Integrated Pest Management in rapeseedmustard, IARI regional station. *Punjab Agricultural University.*, 848, 1-6.
- Meena, P.D., Rathi A.S., Vinod Kumar and Dhiraj Singh (2014). Compendium of Rapeseed-Mustard Diseases: Identification and Management. Directorate of Rapeseed-Mustard Research (ICAR), Bharatpur, Rajasthan, India.
- Mishra, K.K., Kolte S.J., Nashaat N.I. and Awasthi R.P. (2009). Pathological and biochemical changes in *Brassica juncea* (mustard) infected with *Albugo candida* (white rust). *Plant Pathology*, **58**, 80-86.
- Pandey, P., Tewari A.K. and Awasthi R.P. (2013). Morphological and pathogenic variability of *Albugo candida* isolates causing white rust in rapeseed-mustard. *The Bioscan.*, 3, 835-838.
- Saharan, GS. (1984). A review of research on rapeseed mustard pathology in India. In: Annual workshop AICORPO, Jaipur, Rajasthan, India.
- Upadhyay, P., Tewari A.K., Dev D. and Daniel GR. (2021). Evaluation of different *Brassica* genotypes under field conditions against white rust (*Albugo candida*) of rapeseed-mustard for resistant sources. *Int. J. Chem. Stud.*, 9(1), 2694-2699.
- Vanderplank, J.E. (1963). Plant Diseases: Epidemics control. Academic press, New York.
- Wheeler, B.E.J. (1969). An Introduction to Plant Diseases. John Wiley and Sons Ltd, New York.
- Wilcoxen, R.D., Skovnand B. and Atif A.N. (1975). Evaluation of wheat cultivar for their ability to retard development of stem rust. Ann. Appl. Biol., 80, 275-281.
- Yadav, D.K., Vignesh M., Sujata V., Yadav A.K., Mohapatra T. and Prabhu K.U. (2011). Characterization of Indian mustard (*B. juncea*) indigenous germplasm line BIOYSR for white rust resistance. *Indian J. Pl. Gen. Reso.*, 24, 400-442.