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Determination of slow white rusting response of Indian mustard genotypes to white rust

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Abstract

Slow rusting resistance to white rusting (*Albugo candida*) in twenty two Indian mustard (*Brassica juncea*) genotypes was determined through two epidemiological parameters, disease severity (%) and area under disease progress curve (AUDPC) and four components of resistance (incubation period, latent periods, number of pustules per leaf and pustule size) under artificial inoculation in field. A considerable range of variations for slow rusting and its components were found among all the twenty two Indian mustard genotypes inoculated with *Albugo candida* during *rabi* 2017-18 and 2018-19. Out of these nine genotypes *viz.*, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 showed lesser size of pustules in range of 0.83 to 1.58 mm and less numbers of pustules per leaf in a range of 24.17 to 51.50 indicating slow white rusting development. Genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 also showed improved resistance against white rust disease as these genotypes had low level of disease severity in range of 5.28 to 10.00 per cent and also low values of AUDPC ranging between 12.07 to 30.64 cm^2 indicating slow white rusting behavior.

Keywords: *Albugo candida, slow white rusting, Indian mustard, white rust*

Introduction

Rapeseed-mustard (Oilseed Brassicca) are the second most important edible oilseed crop in India after groundnut, accounting nearly 30% of the total oilseed produced in the country (Priyamedha *et al.*, 2021). Rapeseed-mustard area, production, and productivity are 7.99 million hectares, 11.96 million tons, and 1497 kg per hectare, respectively, and it is primarily grown in India during the *rabi* season (Anonymous, 2024). In Haryana area, production, and productivity are 0.1714 million hectare, 1.366 million tons, and 1914 kg per hectare (Anonymous, 2024). Among all seven cultivated *Brassicca* species [*Brassica juncea* (L.) Czern and Coss.], *Brassica* napus L., *Brassica napus* spp. yellow sarson, *Brassica rapa* L. spp. brown sarson and *Brassica rapa* spp. toria, *Brassica carinata* Braun and *Brassica nigra* (L.) Koch, *Brassica juncea* is an important oilseed crop in India contributing about 70-80% of the area as well as production obtained from Rapeseed-mustard (Anonymous 2020). The major constraints in realization of higher yield of oilseed Brassicas is its susceptibility to biotic and abiotic stresses, which causes substantial qualitative and quantitative yield losses (Meena *et al.*, 2010). White rust caused by the biotrophicoo-mycete *Albugo candida* (Pers. ex. Lev.) Kuntze is a major disease of oilseed and vegetable crops belonging to the genus *Brassica* (Kamoun *et al.*, 2015). Sangeetha and Siddaramaih (2007) have reported that a maximum temperature of 26 to 29°C, a minimum temperature of 14 to 15°C and average relative humidity of more than 65% favour the development of these diseases. The disease appears as white pustules containing zoospores on the abaxial leaf surface of susceptible plants, while upper surface facing pustules show yellowing; these pustules become more prominent as the disease progresses (Meena *et al.*, 2014; Saharan *et al.*, 2014). The infection can spread systemically to the reproductive parts of the infected plant and stimulates deformities like hypertrophy and/or hyperplasia at the flowering resulted in 'stageheads formation' or 'floret infection' that contain oospores (Saharan *et al.*, 2014; Meena *et al.*, 2014). Staghead are usually co-infected with downy mildew caused by oomycete *Peronospora* speices. The Indian gene pool lines of *Brassica juncea* (oilseed mustard) are highly susceptible to *A. candida*.

Among various management practices, breeding for resistant varieties is one of the most eco-friendly, economic and effective method for the control of plant

diseases. Sources of complete resistance having monogenic or oligogenic inheritance against white rust in Indian mustard are available with the mustard breeders and breeding for vertical resistant varieties through conventional method is in progress in India. However, in past vertical resistance in many temperate countries has been known to be surrendered due to the occurrence of new races that realized the attention towards more stable form of resistance. Use of horizontal resistance is an alternative to vertical resistance as a potential means of reducing damage to plant diseases but it has not been widely used. This type of horizontal resistance called as slow rusting particularly in wheat. In slow rust genotypes a slow epidemic build up despite a high infection type indicating a compatible host-pathogen relationship. Slow rusting is a form of partial resistance in which host genotypes retard or delay rust development by various means. It is both race non-specific and durable (Priyamvada *et al.*, 2011). This type of resistance allows some disease to develop; resulting in reduced selection pressure for the preferential development of undetected virulent strains. It has been observed that cultivars with this type of resistance confer protection for longer periods than complete or hypersensitive type of resistance. However, this phenomenon has been used in few host pathogen systems like leaf and stem rust of wheat, crown rust of oats and slow mildewing in case of powdery mildew of wheat. So, there is a need to identify genotypes with slow white rusting attributes to curb the epidemic development of white rust, in the field. Several researchers (Upadhyay *et al*., 2021; Bisht *et al.*, 2016) reported varieties resistant to white rust of mustard but due to emergence of new races of the pathogen, there is a chance of breakdown of resistance in the existing cultivars.

The objective of this study was to evaluate some of Indian mustard genotypes for resistance to white rust using the components and parameters of slow rusting resistance under field conditions. Slow white rusting resistance to *A. candida* in crucifer genotypes can be identified through lower infection frequency, longer latent periods, longer incubation periods, smaller pustule size and less number of pustules per leaves. Obviously these are the components which minimize primary inoculum and secondary spread of the disease and ultimately determine the final disease build up on the plant.

Materials and Methods

Twenty two Indian mustard (*Brassica juncea*) genotypes *viz.*, RH 1400, RH 1556, RH 1566, RH 1569, RH 1573, RH 1590, RH 1658, RH(OE) 1701, RH(0E) 1705, RH(OE) 1706, RH 1799-2, RH 1799-30, RH 1799-59, RH 1799-63, RH 8701, PHR 2, EC 322091, EC 399301, DOMO 4 and ZEM 2 along with one complete resistance (BIOYSR) and one highly susceptible check (Rohini) were sown at Oilseeds Research Area of CCS Haryana Agricultural University, Hisar during *rabi* 2017-18 and 2018-19, to find out the slow white rusting behaviour of genotypes under artificial inoculation conditions. Fresh zoosporangial suspension (10^5/ml) from leaves collected from early or timely sown mustard fields was sprayed at the siliquae formation. Slow white rusting components *viz.*, incubation and latent periods, number of pustules per leaf, size of pustule, disease severity and AUDPC were chosen for the study. Disease severity was calculated by using 0-6 disease rating scale with slight modification.

Area under disease progressive curve (AUDPC)

For comparative study of disease progression over a time in 22 Indian mustard cultivars, area under disease progressive curve was also computed. By plotting the disease severity values on Y- axis and time interval of observations on X- axis a curve is obtained. By plotting slopes to the curve at regular intervals, area under disease progressive curve can be obtained, which is calculated by the formula of Vander Plank (1963).

$$\text{AUDPC} = \sum_{i=1}^{n-1} \left(\frac{y_i + y_{i+1}}{2} \right) (t_{i+1} - t_i)$$

Where "t" is time in days of each reading, "y" is the percentage of affected foliage at each reading and "n" is the number of readings.

Incubation period

The time period between the inoculation and date of first appearance of the symptom on each genotype was recorded as incubation period (days).

Latent period

The period between the inoculation and full development of symptom and sporulation (pustules) on each genotype was recorded as latent period (days).

Number of pustule/leaf

Number of pustule/leaf was recorded on ten tagged leaves of each genotype on every 5th day and final observation was taken at maximum pustules development.

Size of pustule

The size of pustule was recorded on ten tagged leaves of each genotype on every 5th day and final observation was taken at maximum pustules size development (mm).

Statistical analysis

The data from the experiments were analyzed by using

statistical package of programs OPSTAT (Sheoran *et al.*, 1998). Angular transformation was done for analysis of the percent data wherever required. Analysis of variance (ANOVA) in one way for the analysis of the data was used to calculate the critical difference (CD) and coefficient of variations (CV) for the significance of the treatments.

Results and Discussion

Slow rusting is based on hormonous system of coexistence in which neither of two components (host and pathogen) disturbs each other. This balance can be achieved by evaluation of different component of slow rusting which impart longer life to the cultivar. Host-genetic resistance is still the most effective method for controlling plant diseases, especially rust diseases (Said and Taher 2020). A considerable range of variations for slow rusting and its components was found among all the twenty two Indian mustard genotypes inoculated with *Albugo candida* during the two successive seasons. The incubation period was shortest of 4 days in the highly susceptible genotype (fast rusting) rohini and longest of 9 days in the resistant (slow rusting) genotypes such as EC 322091. Whereas, in some genotypes *viz*., PHR-2, EC 399301, DOMO-4, ZEM 2 and RH 1400 incubation period found between the range of 7 to 9 days (Table 1). Furthermore, latent period varied between 8 to 15 days in all the genotypes. Shortest latent period of 8.5 day was recorded in Rohini as compared to longest latent period of 15 days in RH 1400. Genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 showed latent period in a range of 13 to 15 days indicating slow white rust development. Rate of infection or disease spread is influenced by incubation periods and latent periods of *A. candida* in its compatible host. In white rust disease, the sporangia become visible after the host epidermis is ruptured as a white powdery mass which can readily be dispersed by wind or rain drops to cause secondary infection. In rapeseed, white rust pustules become visible in 5-6 days after inoculation (Liu *et al*., 1989), while in cabbage symptoms appear in 8 days after inoculation. In *B. Juncea* cvs. Rajat and RC 781, incubation and latent periods of 11/14, and 11/15 days have been observed; similarly, in *B. Rapa* cvs. Candle, Tobin and Span, longer incubation and latent periods of 11/15, 15/18 and 11/18 days respectively have been observed (Gupta and Saharan, 2002).

Table 1: Component of slow rust on selected genotypes of Indian mustard after artificial inoculation in field during 2017-18 & 2018-19

Genotypes	Incubation period (days)			Latent period (days)		
	2017-18	2018-19	Mean	2017-18	2018-19	Mean
RH 1400	9.0	8.0	8.50	15.0	15.0	15.00
RH 1556	7.3	7.0	7.17	13.7	13.0	13.33
RH 1566	6.7	6.7	6.67	10.5	11.4	10.95
RH 1569	8.0	7.3	7.67	14.0	13.3	13.65
RH 1573	8.0	7.3	7.67	14.0	13.3	13.65
RH 1590	7.7	7.0	7.33	11.0	11.0	11.00
RH 1658	5.3	5.0	5.17	9.6	9.9	9.75
RH(OE) 1701	6.3	6.0	6.17	9.0	9.0	9.00
RH(0E) 1705	6.7	6.7	6.67	10.0	10.0	10.00
RH(OE) 1706	7.0	7.0	7.00	12.7	11.9	12.28
RH 1799-2	7.3	7.0	7.17	11.9	12.7	12.30
RH 1799-30	7.3	8.0	7.67	13.1	12.9	13.00
RH 1799-59	7.0	6.0	6.50	9.7	9.0	9.35
RH 1799-63	6.3	5.7	6.00	10.0	9.7	9.85
RH 8701	6.3	6.0	6.17	10.0	9.3	9.65
PHR 2	8.3	8.0	8.17	14.7	14.3	14.50
EC 322091	9.0	9.0	9.00	14.0	14.0	14.00
EC 399301	8.7	8.0	8.33	14.0	14.0	14.00
DOMO 4	8.0	8.3	8.17	13.3	14.0	13.65
ZEM 2	8.0	8.3	8.17	14.5	13.5	14.00
Rohini	6.0	4.7	5.33	9.0	8.0	8.50
BIOYSR	0.0	0.0	0.00	0.0	0.0	0.00

Table 2: Component of slow rust on selected genotypes of Indian mustard after artificial inoculation in field during 2017-18 & 2018-19

Genotypes	Size of pustule before coalseing (mm)			Pustules/leaf (no.)		
	2017-18	2018-19	Mean	2017-18	2018-19	Mean
RH 1400	0.67	0.83	0.75	35.33	50.67	43.00
RH 1556	1.50	1.67	1.58	25.33	45.67	35.50
RH 1566	2.00	2.33	2.17	32.33	51.00	41.67
RH 1569	1.50	1.33	1.42	41.00	62.00	51.50
RH 1573	1.50	1.17	1.33	46.00	66.00	56.00
RH 1590	1.67	2.33	2.00	40.33	61.00	50.67
RH 1658	2.33	2.00	2.17	33.00	51.33	42.17
RH(OE) 1701	3.00	3.67	3.33	60.00	81.33	70.67
RH(0E) 1705	2.67	3.00	2.83	42.67	62.00	52.33
RH(OE) 1706	2.00	2.33	2.17	35.67	56.00	45.83
RH 1799-2	1.83	2.17	2.00	53.67	73.00	63.33
RH 1799-30	1.17	1.50	1.33	15.33	33.00	24.17
RH 1799-59	2.50	2.50	2.50	54.67	74.67	64.67
RH 1799-63	2.67	3.00	2.83	33.67	52.00	42.83
RH 8701	1.67	2.00	1.83	63.33	88.33	75.83
PHR 2	1.67	1.50	1.58	27.33	48.00	37.67
EC 322091	0.83	1.00	0.92	15.00	32.33	23.67
EC 399301	0.67	1.00	0.83	15.00	32.33	23.67
DOMO 4	1.33	1.33	1.33	31.33	50.33	40.83
ZEM 2	0.50	1.00	0.75	16.00	35.00	25.50
Rohini	5.00	6.00	5.50	129.67	154.0	141.83
BIOYSR	0.00	0.00	0.00	0.00	0.00	0.00

Pustule size is also considered to be one of the important components of slow rusting trait and associations of small pustule size with slow rusting has been reported in wheat and beans. In present investigation smallest size of pustules 0.75 mm was recorded on genotype RH 1400 and ZEM 2, while largest size of pustules 5.55 mm on genotype Rohini (Table 2). However, medium size pustules ranging from 0.83 to 1.58 mm were observed on the genotypes *viz.*, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2. On the rest of genotypes pustules size ranged from 1.83 to 3.33 mm. The number of pustules per leaf is also genetical response of genotypes to pathogen which shows effective range of resistance and susceptibility. This study indicate that out of twenty two genotypes minimum numbers of pustules 23.67 per leaf were observed on the genotypes EC 322091 and EC 399301 as compared to maximum numbers of pustules 141.83 per leaf on the genotype Rohini. Numbers of pustules were also significantly lower in a range of 24.17 to 51.50 on the genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2. However, on the rest of genotypes numbers of pustules per leaf ranged significantly medium in numbers 41.67-70.67. In a similar study, Mehta *et al*., (2009) reported the progression of powdery mildew in nine mustard varieties *viz.*, RH-30, RH-9801, RH-8812, RH-9901, RH-9304, RC-781, Purple mutant, GSL-1 and HC-9603 for components of slow mildewing. The disease appeared late in all varieties except HC-9603 which was free from disease. Only variety GSL-1 showed less progression on the basis of number of specks/leaf, size of specks, number of conidia per speck, incubation period, disease progression and disease intensity among all the tested varieties of mustard. In our study, some genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 showed improved resistance against white rust disease. Minimum disease severity of 3.89 per cent was observed on the genotype EC 322091 followed by 5.00 per cent on genotype EC 399301 as compared to maximum disease severity of 50.69 per cent on Rohini (Table 3). Genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 showed low level of disease severity in range of 5.28-10.00 per cent indicating slow white rusting behavior. However, rest of the genotypes showed moderate level of disease severity in range of 11.11 to 30.28 per cent.

Table 3: Component of slow rust on selected genotypes of Indian mustard after artificial inoculation in field during 2017-18 & 2018-19

Genotypes	Disease severity (%)			AUDPC (cm^2)		
	2017-18	2018-19	Mean	2017-18	2018-19	Mean
RH 1400	5.00	6.11	5.56	14.93	18.58	16.76
RH 1556	9.17	9.72	9.44	24.14	28.82	26.48
RH 1566	12.50	11.11	11.81	35.76	37.68	36.72
RH 1569	8.89	8.61	8.75	22.05	25.69	23.87
RH 1573	10.28	11.95	11.11	31.42	34.72	33.07
RH 1590	12.78	13.61	13.20	36.11	41.66	38.89
RH 1658	11.95	13.61	12.78	33.16	43.23	38.19
RH(OE) 1701	29.72	30.83	30.28	91.14	95.66	93.40
RH(0E) 1705	14.44	15.00	14.72	43.93	47.92	45.92
RH(OE) 1706	11.94	13.61	12.78	35.24	41.14	38.19
RH 1799-2	12.50	13.33	12.92	33.16	41.36	37.26
RH 1799-30	5.00	5.56	5.28	14.07	17.18	15.63
RH 1799-59	19.72	21.11	20.42	57.89	60.94	59.41
RH 1799-63	19.17	21.57	20.37	56.08	64.69	60.38
RH 8701	15.28	15.00	15.14	44.10	46.35	45.23
PHR 2	10.00	9.72	9.86	29.51	32.47	30.99
EC 322091	3.33	4.44	3.89	10.08	14.06	12.07
EC 399301	3.89	6.11	5.00	15.63	20.14	17.88
DOMO 4	10.00	10.00	10.00	29.69	31.60	30.64
ZEM 2	3.89	6.67	5.28	15.28	19.10	17.19
Rohini	45.56	55.83	50.69	129.5	166.8	148.2
BIOYSR	0.00	0.00	0.00	0.00	0.00	0.00

Based upon the progression of disease at different intervals AUDPC was statistically analyzed and presented in Table 3. It has been found that, AUDPC was lowest of 12.07 cm^2 in genotype EC 322091 followed by 15.63 cm^2 in genotypes RH 1799-30 as compared to maximum values of AUDPC 148.2 cm^2 in genotype Rohini that showed susceptible reaction. However, some genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1573, RH 1799-30, PHR-2, EC 322091, EC 399301, DOMO-4 and ZEM 2 showed improved resistance against white rust disease as it evident from low values of AUDPC ranged between 12.07-30.64 cm^2. In rest of the genotypes values of AUDPC ranged between 33.07-93.04 cm^2. It was found that value of AUDPC was lowest in genotypes showing slow white rusting behavior. These genotypes showed low level of disease severity also show low values of AUDPC indicating slow development disease in relation to time and space as AUDPC is reliable parameter to assess slow rusting (Raza *et al.*, 2016; Hei, 2017). SanjanaVeni *et al*. (2025) evaluate 38 genotypes of mustard, three genotypes NPJ 257, PRO 5111 and Pusa MH 126 exhibited moderately resistant reaction with lower AUDPC values of 227.78, 277.78 and 333.33 indicating lower disease severities and slower disease progression and hence these three genotypes (NPJ 257, PRO 5111 and Pusa MH 126) indicating slow rusting.

Wheat varieties DWR 39, HD 2189, HI 977, Keerti, Sonalika, WH 147 and WH 416 identified as slow leaf rusters on the basis of values of AUDPC. Hence, these slow rusters can be cultivated in fields for sustainable wheat production, and can be included in further breeding programs to produce durable rust resistant varieties. Mishra *et al*. (2009) also reported that components of disease reaction, including longer incubation period, smaller size of pustules and lower number of zoosporangia per pustule showed lowest disease index in genotypes of *B. juncea viz.*, EC-399296, EC-399299, EC-399313, EC-399301 as compared to Varuna and Kranti with *A. candida.* Whereas, genotypes Varuna and Kranti showing significantly smaller incubation period, larger size pustules, higher number of zoosporangia had higher disease index. The genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1799-30, PHR-2, DOMO-4, ZEM 2, EC 399301 and EC 322091 showed slow white rusting behavior.

Conclusion

The information on slow white rusting in Indian mustard seems limited but there is scope to exploit this nature of

disease resistance. The completely resistant genotypes against disease are available with mustard breeders and work for vertical resistance breeding is continuous progress. However, polygenes or minor genes are in combination with major genes known to play durable resistance and avoids epidemics. The genotypes *viz.*, RH 1400, RH 1556, RH 1569, RH 1799-30, PHR-2, DOMO-4, ZEM 2, EC 399301 and EC 322091 showed slow white rusting behavior on the basis of longer incubation and latent periods, smaller pustule size and less number of pustules per leaves and less AUDPC. Since, these are the components which determine the fast built up of secondary spread of this polycyclic pathogen.

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