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Gene effects for phenol content in Indian mustard (*Brassica juncea* L. Czern & Coss.)

Ramesh Kumar*, N. K. Thakral, D. Singh and R. K. Behl

Oilseeds Section, Department of Plant Breeding, CCS HAU, Hissar 125 004

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Abstract

Higher phenol content, in general, is considered as an important factor to impart disease resistance in *Brassica* species. Therefore, gene effects involved in governing total phenols were studied in six generations i.e. P1, P2, F1, F2, BC1, and BC2, of three crosses in Indian mustard. Both, additive as well as non-additive gene effects figured important. Also, epistatic effects were prominent in most of the crosses studied. Findings of the present study for advocated for inter-mating in segregating generations to accumulate favorable alleles responsible for the genetic control of phenol content. Selection of desirable types in advance segregating generations would be useful in improving phenol content.

Key words: *Brassica juncea,* phenols, gene effects, white rust resistance

Introduction

Indian mustard [*Brassica juncea* (L.) Czern & Coss.] is the most important oilseed *Brassica* species in Indian subcontinent. It possess inherently high yield potential and relative tolerance to biotic and abiotic stresses as compared to other cultivated *Brassica* species *viz*., forms of *B. nigra* and oliferous. However, Indian mustard is vulnerable to number of diseases such as alternaria blight, white rust, downy mildew and powdery mildew. Among these white rust has been found to cause severe losses up to 54.5 per cent in yield under late sown conditions [1]. All the prevailing high yielding varieties of Indian mustard are susceptible to this disease. Although, the losses due to white rust can be mitigated to some extent by seed treatment and sprays of fungicides, but the use of fungicides is quite expensive and environmentally unsafe. This warrants for breeding disease resistant genotypes with durable resistance for sustainable production and environmental safety.

Studies on disease resistance mechanisms at biochemical level have revealed the importance of phenolic compounds, protein, reducing as well as total sugars in different crops [2-4]. The biochemical basis of resistance clearly indicated that higher amount of phenols is important for enhancing the level of resistance [3]. Significant genetic variability for phenol content has been reported by various workers in Indian mustard [5]. Genotypes RH 8113, RC 781, UDN 69 are reported sources for white rust resistance whereas; RH9624, Varuna and Sarita are susceptible ones. RH 8113 is a released variety for Haryana state as white rust tolerant genotype; RC 781 [6] and UDN 69 are identified donor sources for white rust resistance [7]. Indian mustard white rust resistant genotypes possess higher content of phenols as compared to the susceptible ones [5]. However, there is scanty information available on the mode of inheritance of this trait in Indian mustard. The knowledge of gene effects governing phenol content would be pre-requisite to initiate a sound breeding programme to develop white rust resistant cultivars in this crop. Considering available information the present investigation was undertaken to study the gene effects for phenol content in three crosses of Indian mustard involving genetically diverse parents.

Materialand methods

Genetically diverse Indian mustard genotypes differing in their response to white rust *viz*., RH8113, RC781, UDN69 (resistant), RH9624, Varuna and Sarita (susceptible) were involved in crosses to develop six generations (P1, P2, F_1, F_2, BC_1, and BC_2) in respect of three crosses *viz*., RH8113 x RC781 (R x R), UDN69 x RH9624 (R x S) and Varuna x Sarita: (S x S). These six

*Corresponding author's present address: PPV&FR Authority, 2nd Floor NASC Complex, D.P. Shastri Marg, New Delhi 110 012

generations in each of the three crosses were developed during 1999. The experiment to study gene effects for phenol content was conducted in a compact family block design replicated thrice under two environments i.e. normal (E1-21st Oct., 2000) and late (E2-23rd Nov., 2000) sown at research area of Department of Plant Breeding, CCS HAU, Hisar.

The second environment (E2) was created by delayed sowing because under normal sown conditions the temperature remain high with low humidity so the chances of inoculum build up of this disease are very less, whereas, under late sown conditions low temperature accompanied with high humidity provide better chances for the growth of fungus. Seeds of each of the non-segregating generations (P1, P2, F_1), backcrosses (BC_1, BC_2) and F_2's were sown in one, two and eight row plots of 4 meter length in each replication, respectively, The row-to-row and plant-to-plant distances were maintained as 30 cm and 15cm, respectively. The random diseased and healthy leaf samples were collected at vegetative (35DAS) and siliquae formation stage (65 DAS) and analyzed for total phenols as per Swain and Hillis [8]. The data was subjected to generation mean analysis to estimate the gene effects following the methods suggested by Cavalli [9] and Jinks and Jones [10] to judge that how best the model fit well in each cross.

Results and discussion

The results obtained in present studies (Table 1) revealed that phenol content was, in general higher in E1 as compared to E2 at 35 DAS as well as 65 DAS irrespective of resistance or susceptibility of parents to white rust. The reduction in phenolic compounds was more at 65 DAS than at 35 DAS.

The phenolic constituents were generally observed higher in resistant cultivars than susceptible ones [11]. The susceptible cultivar Varuna in E1 had shown 34.56 mg/g phenols which were at par with the resistant/tolerant cultivar. However, after the infection of disease considerably more reduction in the phenolic constituents was observed in susceptible cultivars including Varuna as compared to resistant genotypes [12]. This is also in corroborative of the results [13], wherein it was reported that different temperature regimes caused marked differences in phenol content in *Sitica spruce,* but clonal characteristics generally overrode treatment differences. High temperature favoured synthesis of polymeric polyphenols. We found in the same experiment that the disease incidence was higher in E2 at 65 DAS as compared to E1 and at 35 DAS. Considering E2 as most favourable environment and 65 DAS as most appropriate plant growth stage for the disease development, a comparative evaluation for phenolic content among parents revealed interesting results. Phenolic content in most parents was at par with each other at 65 DAS in E2. However, after infection susceptible parents like RH 9624, Varuna and Sarita exhibited 31.07 to 38.03% reduction in phenol content incidentally these genotypes were also at par with each other for phenol-content at 65 DAS post infection. The resistant parents, however, revealed much lower reduction in phenol content (4.73 to 13.36%) at 65 DAS post infection. Similar results were reported [14] in muskmelon against disease caused by *Sphaerotheco faliginea* and in case of Alternaria blight of mustard [15]. They also reported that magnitude of post infection reduction in phenolic compounds was more in susceptible cultivars than the resistant ones. Therefore, the genotypes which have the ability to maintain the values of phenolic compounds after the incidence of disease are more important for white rust resistance. The F_1s exhibited in general, either intermediate values of parental genotypes for phenol content or tended towards the parent possessing low phenol contents. However, the crosses involving both or at least one of the resistant genotypes, the F_1s at 65 DAS showed considerably higher phenol content.

The results presented in Table 2 revealed that additive-dominance model holds adequate for the crosses RH 8113 x RC 781 at vegetative stage and for UDN 69 x RH 9624 at siliqua formation stage under healthy conditions (E2). For other crosses additive–dominance model was found adequate, indicating the presence of non-allelic interactions. Therefore, the data was subjected to work out the digenic non-allelic interactions.

The additive as well as non-additive gene effects were found significant in all three crosses under both the situations. The dominance gene effects had higher magnitude than the additive gene effects. The additive x additive type of interactions were significant in crosses RH 8113 x RC 781 in E1 and E2 under both the stages; UDN 69 x RH 9624 under diseased (D) conditions only and in the cross Varuna x Sarita across the environments and stages except at siliqua formation stage in healthy leaves under late sown conditions. Additive x Additive kind of interactions being fixable in nature and can be exploited for further improvement through simple selection. The additive x dominance type

Table 1. Mean performance of six generations of three crosses for total phenols in Indian mustard

Cross	Stage	Environment	Total Phenols (mg/g) over generations				B_1	B_2
			P1	P2	F_1	F_2		
RH 8113 x RC781	Vegetative (35 DAS)	E1	37.65±0.24	56.57±0.34	47.11±0.26	38.87±1.55	38.80±0.84	48.08±1.06
		E2	24.96±0.16	22.63±0.07	29.87±0.09	24.21±1.13	21.56±0.96	21.24±0.43
	Siliqua formation (65 DAS)	E1	19.37±0.27	27.78±0.34	22.22±0.20	29.93±1.20	20.41±0.56	30.29±0.53
		E2 (H)	21.17±0.19	19.23±0.16	26.42±0.05	20.31±1.04	14.71±0.71	17.22±0.46
		E 2(D)	18.34±0.11	8.32±0.07	21.21±0.15	18.76±0.55	19.14±0.27	21.26±0.21
UDN 69 x RH9624	Vegetative (35 DAS)	E1	33.96±0.43	23.77±0.26	24.65±0.16	24.39±1.00	27.34±0.46	23.15±0.52
		E2	22.47±0.14	18.57±0.17	18.82±0.10	21.30±0.86	21.06±0.57	20.47±0.38
	Siliqua formation (65 DAS)	E1	26.49±0.47	17.55±0.17	17.61±0.14	21.05±1.26	21.78±0.83	21.24±0.78
		E2(H)	19.75±0.16	18.34±0.03	24.03±0.06	15.44±0.53	14.36±0.38	14.47±0.34
		E2(D)	18.56±0.07	12.64±0.13	19.79±0.25	19.52±0.45	19.15±0.25	17.77±0.30
Varuna x Sarita	Vegetative (35 DAS)	E1	34.56±0.43	29.63±0.83	29.84±0.39	27.11±1.10	28.45±0.80	31.02±0.73
		E2	28.26±0.37	31.98±0.38	28.73±0.14	33.80±0.99	26.63±0.50	30.01±0.44
	Siliqua formation (65 DAS)	E1	17.43±0.41	21.88±0.22	18.53±0.17	16.36±0.73	19.65±0.45	17.43±0.51
		E2(H)	20.65±0.18	20.01±0.13	20.79±0.06	20.12±1.04	20.60±0.61	19.19±0.37
		E2(D)	12.77±0.06	13.00±0.04	11.44±0.07	10.34±0.36	12.58±0.31	12.27±0.17

Table 2. Estimates of gene effects on 3-parameter model for total phenols in Indian mustard

Crosses	Stage	Environment	Gene effects			
			(m)	(d)	(h)	(χ^2)
RH8113 x RC781	Vegetative (35 DAS)	E1	46.87±0.21	9.41±0.21	-0.15±0.34	**54.42
		E2	**23.79±0.09	**-1.16±0.09	-3.91±0.13	5.14
	Siliqua formation (65 DAS)	E1	24.14±0.21	4.75±0.21	-1.53±0.30	**11.82
		E2 (H)	20.14±0.12	-0.90±0.12	-3.72±0.13	**33.15
		E2 (D)	18.32±0.16	0.06±0.06	-3.07±0.15	**53.72
UDN 69 x RH9624	Vegeative (35 DAS)	EI	28.40±0.23	-4.78±0.23	-3.98±0.13	**21.84
		E2	20.64±0.11	-1.85±0.11	-2.13±0.14	**30.89
	Siliqua formation (65 DAS)	E1	22.07±0.24	-4.42±0.24	-4.49±0.28	**21.84
		E2 (H)	**15.53±0.07	**-0.19±0.07	-1.50±0.09	3.86
		E2(D)	18.56±0.07	-0.98±0.07	0.86±0.23	*11.10
Varuna x Sarita	Vegetative (35 DAS)	E1	32.67±041	-1.59±0.41	-7.05±0.59	**23.77
		E2	29.97±0.25	2.03±0.25	-1.29±0.30	**33.11
	Siliqua formation (65 DAS)	E1	19.70±0.21	1.86±0.21	-1.29±0.28	**53.06
		E2 (H)	20.27±0.12	-.038±0.12	0.50±0.13	** 11.42
		E2(D)	13.0O±0.11	-0.01±0.11	-1.53±0.13	**11.64

*,**Significant at 5% and 1% level, respectively.

of interactions were found significant in crosses RH 8113 x RC 781 at siliqua formation stage in both the environments; UDN 69 x RH 9624 at vegetative stage under late sown conditions and in E1 at siliqua formation stage; Varuna x Sarita across the environments and stages except at siliqua formation stage under late sown conditions. The dominance x dominance types of interactions were found to be significant for crosses RH 8113 x RC 781 at siliqua formation stage under late sown conditions (healthy and diseased); UDN 69 x RH 9624 under diseased conditions and Varuna x Sarita in both the environments at vegetative stage and at siliqua

Table 3. Estimate of gene effects of generation means on three/six parameters model for total phenols in Indian mustard

Cross	Stage	Environment	Gene effects for total phenols						Type of epitasis	Joint test (χ^2)
			(m)	(d)	(h)	(i)	(j)	(l)		
RH 8113	Vegetative	E1	28.83±6.82	**9 .46±0. 21	21.87±14.95	** 18.28±6.81	–0.36±2.76	3.58±8.32		**54.42
x	(35 DAS)									
RC781		E2	-	-	-	-	-	-		5.14
	Siliqua formation (65 DAS)	E1	41.89±5.05	**4.20±0.22	**-28.18±10.70	**–18.32±5.05	**I1.34±1.59	8.50±5.74		**118.24
		E2 (H)	36.55±4.52	**0.96±0.12	**-44.86±9.85	**–16.37±4.52.	**7.96±1.76	**24.75±5.44	D	**33.15
		E2 (D)	12.56±2.33	0.0 1±0.06	**16.12±4.92	*5.76±2.32	**4.25±0.71	**–7.47±2.64	D	**53.72
UDN 69	Vegetative (35 DAS)	E1	25.44±4.27	**5.09±0.25	–3.42±9.12	3.41±4.27	1.80±1.47	2.62±4.94		**21.84
RH9624		E2	22.75±3.70	**1.95±0.11	–1.48±1.02	–2.23±3.70	*2.81±1.38	–2.84±4.41		**30.89
	Siliqua formation (65 DAS)	E1	20.18±5.66	**4.46±0.25	6.13±12.55	1.82±5.65	**7.86±2.45	–8.82±7.05		**21.84
		E2 (H)	-	-	-	-	-	-	-	3.86
		E2 (D)	22.83±2.00	**0.96±0.07	**-10.22±4.39	*4.23±2.00	-0.83±0.80	**7.1 7±2.49	D	*11.10
Varuna	Vegetative	E1	21.29±4.96	**2.46±0.46	18.11±11.10	*10.49±4.94	** 10.07±2 .36	*14.16±6.32		**23.77
x	(35 DAS)									
Sarita		E2	52.04±4.18	**1.85±0.27	**–49.65±8.90	**–21.92±4.17	*3.04±1.43	**26.34±4.80	D	**33.11
	Siliqua formation (65 DAS)	E1	10.93±3.25	**2.2 2±0. 23	*14.16±7.21	**8.72±3.24	**–8.89±1.45	–6.50±4.06		**53.06
		E2 (H)	28.02±4.43	*0.31±0.12	–17.59±9.42	–7.68±4.42	–2.18±1.44	*10.33±5.07		**11.42
		E2 (D)	16.54±10.24	0.11±0.12	*–7.72±3.61	*–3.66±1.61	–0.84±0.75	2.61±2.05		**11.64

*, ** Significant at 5% and 1% level, respectively,
- Denotes Additive-Dominance model

formation stage under late sown conditions (healthy leaves). Duplicate type of epistasis was also found in crosses RH 8113 x RC 781 at siliqua formation stage under late sown conditions, UDN 69 x RH 9624 under diseased conditions, whereas, Varuna x Sarita under late sown conditions at vegetative stage. Similar results were reported in Indian mustard [5] and in guar [16]. However, a supplementary study on a larger number of genotypes and these crosses is needed to establish a correlation between phenol content and disease resistance overtime and space so as to chalk out a coherent strategy for breeding white rust resistance in oilseed *Brassicas*. Considerable proportion of additive as well as additive x additive gene effect for phenol content in the crosses RH 8113 x RC 781 in both stages and environments, whereas, UDN 69 x RH 9624 under diseased condition only, suggests use of simple pedigree selection for further improvement. On the other hand intermating in advance segregating generations followed by delayed selection will in general, be useful to improve any trait, when additive and non-additive gene effects with epistatic effects are significant. This kind of breeding approach will be helpful in accumulating favorable alleles responsible for the genetic control of phenol content.

The higher amount of phenol content as well as its stability across the environments is of utmost interest to plant breeders for breeding white rust resistant genotypes in oilseed Brassicas. Therefore, the efforts should be made to further enhance phenol content to a desired level with its stability under heavy disease pressure. Based upon the genetic information generated in the present study, it is advocated that intermating in segregating generations of selected stable plant progenies followed by selection in advance generations would help to enhance the level of phenolic constituents and their stability with resistance to white rust.

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