J. Oilseeds Res., **19**(1): 17-21 (2002)

Inheritance of components of horizontal resistance to *Alternaria brassicae* (Berk.) Sacc. in Indian mustard, *Brassica juncea* (L.) Czern and Coss.

Lakshmi Kant[1] and S.C. Gulati

Division of Genetics, Indian Agricultural Research Institute, Pusa, New Delhi-110 012

(Received: March, 1999; Revised: February, 2002; Accepted: March, 2002)

Abstract

Two relatively resistant parents of Indian Mustard *Brassica juncea* (L.) Czern and Coss., Bio 8 (3) and Line 81, were selected and crossed to Alternaria susceptible parents Pusa Basant, Pusa Bahar, and Line 113 to produce six crosses. Six generations (P_1, P_2, F_1, F_2, B_1, and B_2) of six crosses were planted under timely sown (TS) and late sown (LS) conditions to find out the genetics of components of resistance to Alternaria. The mean of six generations recorded for time of appearance of Alternaria on leaf (TOAP), rate of disease increase (r), area under disease progress curve (AUDPC), final intensity of Alternaria on plant (FIAP), and final intensity of disease on pods (FIDP) were subjected to scaling tests to detect epistasis and to estimate m, d, h, i, j, and l parameters. Dominance (h) and additive x additive (i) had predominant role in the inheritance of TOAP, r, AUDPC, FIAP, and FIDP, whereas additive x dominance (i) was important for AUDPC, FIAP, and FIDP.

Key words: Mustard, *Brassica juncea*, genetics, *Alternaria brassicae*, resistance

Introduction

Rapeseed mustard constitute an important group of oilseeds second to groundnut in area, production, and productivity in India. Causes of lower yields are a number of biotic and abiotic factors. Estimates of yield losses due to Alternaria blight caused by *Alternaria brassicae* (Berk.) Sacc., varied between 10 to 70% in different species of these crops (Kolte, 1985;1991; Saharan and Chand, 1988). Genetics of resistance to Alternaria blight is governed by a single dominant gene in the cultivar RC 781 of Indian mustard (Tripathi *et al.*, 1978; 1980). However, the resistance of cultivar RC 781 has broken down. There is a lack of information on the genetics of parameters of horizontal disease resistance in mustard, which is essential for development of Alternaria blight resistant genotypes. Therefore, the present investigations were carried out to find the resistant genotypes to Alternaria blight and to study the inheritance of components of its resistance.

Materials and methods

Twelve cultigens of Indian mustard *B. juncea*, tolerant to *Alternaria brassicae*, [DYS-7-1, UDN 23, DYS-25-10, UDN-26, UDN-67, 51=D 418/MHTE 23-3, 81=D 128/D 313, 87=Kranti/D 246, 113=PR 45/D 403//D 326, 174=D 313/HTA-11-1, 348=HC 951/K-133//W 246 and Bio 8 (3)], along with susceptible checks (Pusa Bahar, Pusa Basant and Pusa Bold) were evaluated in a replicated trial for their reaction to *Alternaria* under artificial epiphytotic conditions at the experimental farm of IARI, New Delhi during *rabi* season.

Two resistant parents, Bio 8(3) and Line 81, were selected and crossed as male parents to susceptible female parents, Pusa Basant, Pusa Bahar, and Line 113. Six generations of the six crosses Pusa Basant/Bio 8(3) (C_1), Pusa Basant/Line 81 (C_2), Pusa Bahar/Line 81(C_3), Pusa Bahar/Bio 8(3) (C_4), Line 113/Bio 8(3) (C_5) and Line 113/Line 81 (C_6) were grown in randomised block design with two replications during *rabi* season in rows of 2.25 m long with spacing of 30 x 15 cm between lines and plants. The experiment was planted on two sowing dates i.e., timely and late. An artificial epiphytotic of *Alternaria* blight was created to facilitate the screening of various populations. Besides this infector rows of highly susceptible lines were planted in and around the experimental plots. Inoculum was sprayed twice for screening at 30 days interval.

From the middle rows of each replication five plants each from P_1, P_2 and F_1, 40 plants from F_2 and 20 plants each from B_1 and B_2 were randomly selected in each cross. Observations were recorded on time of appearance of

[1] Scientist (SS), Crop Improvement Division, VPKAS (ICAR), Almora-263 601, UA.

disease (TOAP) in days when lesion first appeared on any leaf of plant from sowing, number of lesion (NOL) by counting total number of lesions on two leaves on which the lesions first appeared in succession of the sampled plants, average size of lesions (mm) (SOL) by measuring the diameter of first two spots in two perpendicular directions and then averaging it, area under disease progress curve (AUDPC) = Time interval [½ (sum of first and last disease scores) + (sum of all in between disease scores)] Pandey *et al.* (1989), rate of increase of disease (r) by dividing the difference between maximum number of lesions and number of lesions at first observation by scoring interval (days), final intensity of disease on plant (FIAP) (0-5 scale) (Kant, 1997) and final intensity of disease on pods (FIDP) (0-3 scale) (Kant, 1997).

The data were subjected to A, B and C scaling tests (Mather, 1949) to detect the presence of epistasis. The adequacy of additive dominance model was tested by the joint scaling test (Cavalli, 1952). The gene effects were studied as per Cavalli (1952) and Hayman (1958).

Results and discussion

The results of the present investigation indicated dominance of alleles for resistance in parents Bio 8 (3) and Line 81, which had lesser numbers of disease lesions (Fig 1) and smaller size of lesions (Fig 2) as against remaining parents.

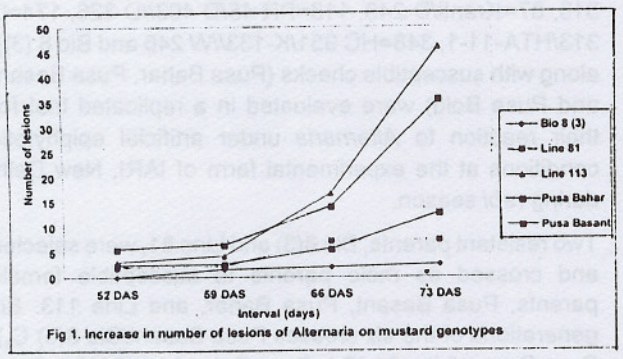

Fig 1. Increase in number of lesions of Alternaria on mustard genotypes

DAS= Days after sowing

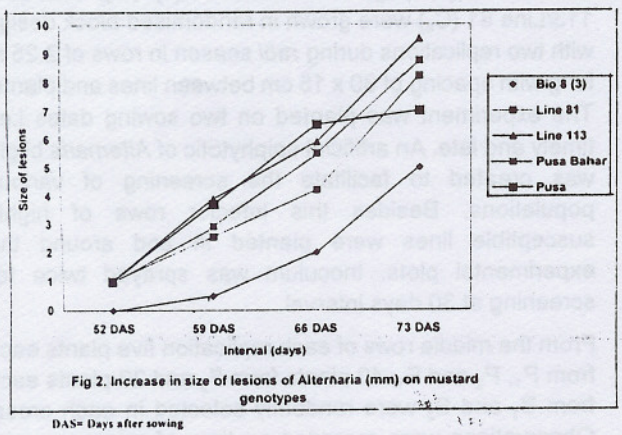

Fig 2. Increase in size of lesions of Alternaria (mm) on mustard genotypes

DAS= Days after sowing

The inheritance of different components of horizontal resistance appeared to be rather complex (Table 1 to Table 3). The inheritance pattern varied with the cross, character and sowing condition under consideration. In most of the cases better estimates of gene effects were found tin (LS) condition, probably due to better expression of disease. A comparison of F_1s with mid parent indicated, in general, a high degree of internal cancellation of gene effects. B_2 means indicated that a second dose of the resistant parent tends to accumulating genes for resistance or lowering expression of disease.

Dominance (h) was found to be more important for the inheritance of TOAP. Cross Pusa Basant/Bio 8(3) and Line 113/Bio 8(3) had both h and i component significant and in desirable direction in (LS) condition. A five parameter model having m = origin, d = additive, h = dominance, i = additive x additive and j = additive x dominance was found adequate to explain the inheritance of TOAP. Therefore, dominance of late appearance of *Alternaria* could be utilized in hybrid development programme. Interaction component i being a fixable component may be exploited in selection programme by transgressive breeding.

Dominance (h) was more important than additive (d) and was in desirable direction for rate of disease increase (r) (Table 1). The rate of increase of *Alternaria* blight indicated multiplication of the disease infection per unit of time and suggested the slow or fast disease reaction of the plants to disease. Lower values would be desirable for selection programme. Negative estimates of h in most of the cases indicated dominance of negative alleles at most of the loci though not able to surpass the lower level in some cases. Pusa Basant/Bio 8(3), Pusa Bahar/Bio 8(3) and Line 113/Bio 8(3) crosses had significant h and i in desirable direction in (LS) condition. Dominance x dominance (l) component was in desirable direction and significant in Pusa Basant/81 and Pusa Bahar/Bio 8(3) crosses. Many workers have demonstrated reduced severity of disease by lower infection rate (r) in different crops. However, low infection rate of resistant cultivars have been reported to be an important component of horizontal resistance in Indian mustard by Saharan and Kadian (1983) and Kadian (1983).

Dominance (h) and additive x additive (i) both were found significant and in desirable direction in Pusa Basant/Bio 8(3) and Line 113/Bio 8(3) crosses in (LS) condition for area under disease progress curve (AUDPC). The h and i components can be exploited through biparental matings or recurrent selections. However, the significance of i component in many cases also suggested that increased manifestation of this character can be achieved through selection programmes. However, additive x dominance (j) was significant and in desirable direction in (TS) condition

in the same crosses. Mani (1991) suggested important role of h and i for AUDPC along with complementary epistasis in case of white rust of Indian mustard. He also found AUDPC as an important parameter in differentiating the genotypes possessing slow and fast rusting behaviour.

Interaction effects were found more important than the main effects for the inheritance of final intensity of *Alternaria* on plant (FIAP) (Table 2). The FIAP is of practical value under field condition to differentiate between resistant and susceptible genotypes. Dominance (h) and additive x additive (i) were found significant and in desirable direction in Pusa Basant/Bio 8(3) and Pusa Bahar/Bio 8(3) crosses. Additive x dominance (j) was important in direction and magnitude in five out of six crosses. The significance of h and i components indicated that biparental mating or recurrent selection could be resorted to improve this character. However, presence of duplicate epistatis may hinder the progress in the selection. Katiyar and Chamola (1995) reported a cultivar selected in the F_7 generation of a cross *Brassica carinata* x *Brassica juncea* showing stable and good resistance to *Alternaria* with only a few small lesions developed on the leaves at completion of fruiting compared with numerous large lesions occurring at flowering on *B. juncea*. Gulati *et al.* (1985) reported final intensity of disease on plant as a criterion for selection under field condition in case of yellow rust of barley. They showed direct relationship between final intensity of disease and AUDPC. Mani (1991) also confirmed these findings in case of white rust of mustard.

Dominance (h) though non-significant was of considerable magnitude and in desirable for final intensity of disease on pods (FIDP) (Table 3). FIDP is of great importance in disease like *Alternaria*, which, when in severe form on pods may lead to shriveling of grains and thereby ultimately leading to reduced oil content. Interaction components were of more importance than main effects. Component i was significant and in desirable direction in Pusa Basant/Bio 8(3) and Pusa Basant/Line 81. However, component j was important in magnitude and direction in Pusa Basant/Line 81 and Line 113/Bio 8(3). Tripathi and Kaushik (1984) reported that intensity of seed infection varied with number of lesions per siliqua in case of rapeseed and mustard. More significance of disease on pods than that of on the leaves was reported by Chahal and Kang (1979). Relation between the infection units on seed pods and loss in seed yield compared with that from healthy pods was reported by Singh and Bhowmik (1985). In such cases it may be possible to expect improvement for this trait in hybrid breeding.

Broadly it can be concluded that dominance (h) had a predominant role in genetic control of later TOAP, lesser r, AUDPC, FIAP and FIDP, whereas additive x dominance (j) was predominant for lesser AUDPC, FIAP and FIDP. Biparental mating or hybrid breeding may be an appropriate strategy to exploit these kinds of gene effects. Additive x additive (i) interaction effects had a major role in later TOAP, lesser r, AUDPC, FIAP and FIDP. This indicated the possibility of selecting for transgressive segregants. In general, crosses with Bio 8(3) showed better expression of resistance and the cross Pusa Basant/Bio 8(3) appeared to be the best cross combination with all the components of horizontal resistance in a desirable direction.

Table 1 Estimates of gene effects ± SE for different components for rate of increase of disease (r)

Cross	Condition	m	d	h	i	j	l	χ^2
C1	TS	0.45**±0.07	0.08±0.04	-0.17±0.11	-0.18*±0.08	-0.14±0.15	-	0.63
	LS	0.88**±0.16	0.08±0.06	-1.38**±0.40	-0.49**±0.15	-0.03±0.15	0.79**±0.24	-
C2	TS	0.36**±0.05	-0.01±0.05	0.13±0.09	-	-	-	4.04
	LS	0.15±0.19	0.09±0.07	0.95±0.52	0.24±0.17	-0.23±0.20	-0.84**±0.33	-
C3	TS	0.37**±0.07	-0.02±0.05	0.08±0.13	-	-	-	1.95
	LS	0.36±0.25	-0.03±0.04	0.08±0.09	-	-	-	4.66
C4	TS	0.17±0.15	0.07±0.05	1.22**±0.41	0.41**±0.14	-0.06±0.15	-0.81*±0.29	-
	LS	0.77**±0.17	0.03±0.04	-1.13**±0.43	-0.43*±0.16	0.52*±0.14	0.70*±0.29	-
C5	TS	0.24**±0.03	0.05±0.03	0.11±0.07	-	-	-	1.75
	LS	0.38**±0.03	0.07±0.03	-0.11±0.06	-	-	-	4.79
C6	TS	0.34*±0.05	-0.05±0.04	-0.06±0.10	-	-	-	-
	LS	0.64±0.10	0.02±0.06	-0.04*±0.14	-0.34**±0.11	-0.03±0.18	-	0.10

C1 = Pusa Basant/Bio 8(3); C2 = Pusa Basant/Line 81; C3 = Pusa Bahar/Line 81; C4 = Pusa Bahar/Bio 8(3); C5 = Line 113/Bio 8(3); C6 = Line 113/Line 81
TS = Timely sown; LS = Late sown *, ** significant at 5% and 1% level

Table 2 Estimates of gene effects ± SE for different components for final intensity of *Alternaria* on plant (FIAP)

Cross	Condition	m	d	h	i	j	l	χ^2
C1	TS	3.70**±0.21	0.71**±0.10	0.15±0.30	0.23±0.23	-2.49**±0.41	-	1.77
	LS	4.70**±0.29	-0.10±0.10	-2.00*±0.82	-0.60*±0.27	0.10±0.30	1.40*±0.56	-
C2	TS	3.58**±0.18	0.35**±0.09	0.91**±0.32	0.67**±0.19	-0.90**±0.28	-	0.01
	LS	3.91**±0.07	0.08±0.07	0.13±0.15	-	-	-	3.76
C3	TS	3.15**±0.34	-0.10±0.16	2.65**±0.97	0.65*±0.30	-0.15±0.41	-1.60*±0.68	-
	LS	4.13**±0.03	0.08±0.08	-0.25±0.23	-0.15±0.12	-0.13±0.12	0.00±0.40	-
C4	TS	4.58**±0.14	0.33*±0.16	-0.94*±0.22	-1.02**±0.21	-0.53±0.47	-	2.59
	LS	4.94**±0.40	0.05±0.10	-2.57*±1.16	-0.69±0.39	-0.96**±0.41	1.73*±0.78	-
C5	TS	4.03*±0.04	0.13±0.17	0.05±0.40	-0.05±0.39	-0.58*±0.20	-0.20±0.74	-
	LS	3.84**±0.05	0.01±0.05	0.68**±0.23	0.76**±0.22	0.13±0.08	-0.73*±0.32	-
C6	TS	4.80**±0.49	0.35**±0.09	-2.20±1.26	-0.55±0.48	-0.85*±0.39	1.70*±0.82	-
	LS	4.10**±0.04	0.10±0.07	-0.03±0.27	-0.38±0.22	-0.04±0.08	0.86±0.46	-

C1 = Pusa Basant/Bio 8(3); C2 = Pusa Basant/Line 81; C3 = Pusa Bahar/Line 81; C4 = Pusa Bahar/Bio 8(3); C5 = Line 113/Bio 8(3); C6 = Line 113/Line 81
TS = Timely sown; LS = Late sown * ** significant at 5% and 1% level

Table 3 Estimates of gene effects ± SE for different components for final intensity of disease on pods (FIDP)

Cross	Condition	m	d	h	i	j	l	χ^2
C1	TS	0.30±0.18	0.28**±0.09	-0.53±0.30	0.55**±0.19	-0.51±0.27	-	2.16
	LS	0.92**±0.14	0.19*±0.08	-0.16±0.23	-0.64**±0.15	0.34±0.25	-	0.35
C2	TS	0.72**±0.13	0.20*±0.09	0.19±0.21	0.19±0.16	-0.69**±0.25	-	0.09
	LS	1.02**±0.17	-0.10±0.10	-0.42±0.25	-0.42±0.21	-0.40±0.30	-	0.10
C3	TS	0.80**±0.04	0/05±0.09	0.30±0.27	0.10±0.25	-0.05±0.13	0.20±0.45	-
	LS	0.89**±0.07	0.05±0.06	-0.04±0.14	-	-	-	3.18
C4	TS	0.59**±0.07	0.28**±0.07	0.18±0.13	-	-	-	4.67
	LS	0.50**±0.06	0.29**±0.05	0.21±0.13	-	-	-	9.06
C5	TS	0.39**±0.05	0.10±0.11	-0.10±0.36	0.35±0.31	-0.15±0.14	-0.50±0.61	-
	LS	0.08±0.19	0.31**±0.07	0.54±0.32	0.35±0.20	-0.77**±0.26	-	1.52
C6	TS	0.83**±0/04	0.03±0.09	0.20±0.29	0.15±0.26	-0.13±0.12	-0.10±0.49	-
	LS	0.75**±0.09	0.00±0.06	0.22±0.18	-	-	-	1.86

C1 = Pusa Basant/Bio 8(3); C2 = Pusa Basant/Line 81; C3 = Pusa Bahar/Line 81; C4 = Pusa Bahar/Bio 8(3); C5 = Line 113/Bio 8(3); C6 = Line 113/Line 81
TS = Timely sown; LS = Late sown *, ** significant at 5% and 1% level

Acknowledgement: Financial support, in the form of Senior Research Fellowship to the first author from IARI, New Delhi is gratefully acknowledged.

References

Cavalli, L.L. 1952. An analysis of linkage in quantitative inheritance. In *Quantitative inheritance* (ed E.C.R. Reeve and C.H. Waddingtion), HMSO, London, pp.135-144.

Chahal, A.S. and Kang, M.S. 1979. Different levels of *Alternaria* blight in relation to grain yield of brown sarson. *Indian Journal of Mycology and Plant Pathology*, 9(2):260-261.

Gulati, S.C., Jain, K.B.L. and Varma, N.S. 1985. Rusting behaviour of some barley varieties to stripe rust. *Cereal Research Communication*, 13(2):223-229.

Hayman, B.I. 1958. The separation of epistatic from additive and dominance variation in generation means. *Heredity*, 12:371-396.

Kadian A.K. 1983. Studies on the *Alternaria* leaf blight of raya. *Thesis abstracts*, 9(1) : 82-83.

Kant, L. 1997. Genetics of resistance to *Alternaria brassicae* (Berk.) Sacc. in Indian mustard (*Brassica juncea* L. Coss), Ph.D. Thesis, IARI, New Delhi.

Katiyar, R.K. and Chamola, R. 1995. Useful end products from *Brassica juncea* x *B. carinata* and *Brassica juncea* x *B. campestris* crosses. *Cruciferae Newsletter*, 17:20-21.

Kolte, S.J. 1985. *Diseases of annual edible oilseed crops.* Vol.II Rapeseed-Mustard and sesame diseases, CRC, Bocartaon Florida, pp.135.

Kolte, S.J. 1991. Diseases. In : *Oilseed Brassicas in Indian Agriculture* (eds. Chopra, V.L. and Shyam Prakash), Vikas Publishing House Pvt. Ltd., New Delhi, pp.198-210.

Mani, N. 1991. Inheritance of white rust [*Albugo candida* (Pers. Ex. Lev.) Kumtzi] resistance in Indian mustard (*Brassica juncea* (L.) Coss.). M.Sc. Thesis, IARI, New Delhi.

Mather, K. 1949. *Biometrical genetics* : *The study of continuous variation*. Methun and Co. Ltd., London.

Pandey, H.N., Menon, T.C.M. and Rao, M.V. 1989. A simple formula for calculating area under disease progress curve. *Rachis*, 8(2) : 38-39.

Saharan, G.S. and Chand, J.N. 1988. *Diseases of oilseed crops*. Haryana Agricultural University, Hisar, pp.268.

Saharan, G.S. and Kadian, A.K. 1983. Analysis of components of horizontal resistance in rapeseed and mustard cultivars against *Alternaria brassicae*. *Indian Phytopathology*, 36(3) : 503-507.

Singh, A. and Bhowmik, T.P. 1985. Persistence and efficacy of some common fungicides against *Alternaria brassicae*, the causal agent of leaf blight of rapeseed and mustard. *Indian Phytopathology*, 38(1) : 35-38.

Tripathi, N.N. and Kaushik, C.D. 1984. Studies on the survival of *Alternaria* brassicae, the causal organism of leaf spot of rapeseed and mustard. *Madras Agricultural Journal*, 71(4) : 237-241.

Tripathi, N.N., Kaushik, C.D., Yadava, T.P. and Yadav, A.K. 1978. Studies on the inheritance of *Alternaria* blight resistance in raya. *Indian Phytopathology*, 31(1) : 127.

Tripathi, N.N., Kaushik, C.D., Yadava, T.P. and Yadav, A.K. 1980. *Alternaria* leaf spot resistance in raya. *Haryana Agricultural University Journal of Research*, 10(2) : 166-168.