## GENETIC ANALYSIS OF DOWNY MILDEW IN PEARLMILLET

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#### **SUMMARY**

The genetics of downy mildew was studied in 144 hybrids developed by crossing six A- and their six B-lines with 12 R-lines of pearl millet in a line x tester mating design. The six A-lines 81A, and 8A, (A<sub>1</sub>), Pb 313A (A<sub>2</sub>), Pb 402A (A<sub>3</sub>), 81A<sub>4</sub> and 81A<sub>5</sub> and their corresponding B- lines represented five different systems of male sterility and were very diverse. The 24 parents and the 144 hybrids were grown separately in contiguous blocks in  $2R \times 2.5 \text{ m} \times 0.45 \text{ m}$  in randomized block design with two replications in six environments under natural condition viz., early sown non-cut crop  $(E_1, E_4)$ , ratio crop  $(E_2, E_5)$  and late sown non-ratooned (E,, E) at Research Farm, CCSHAU, Hisar and two environments in sick plot (SP1, SP2) Department of Plant Pathology, CCS HAU, Hisar. The downy mildew incidence was recorded on all plants in the plot under natural and sick plot after 30 days (stage I) and 60 days (stage II) of sowing. The data on downy mildew incidence (%) were subjected to angular transformation for analysis of variance. The analysis of variance was conducted by developing statistical model involving all genotypeslines (A-, B-), testers (R-lines), A- x R- and B- x R-hybrids, environments and all possible interactions. The combining ability analysis was carried out following line x tester model. The genotypes, parents, lines (A-, B-), testers (R-lines), A- x R- and B- x R-hybrids differed significantly at both the stages but A- vs. Blines and A- x R- vs. B- x R-hybrids contrasts did not differ significantly showing no role of cytoplasm in downy mildew vulnerability. The significant differences among lines, testers and lines x testers (hybrids) indicated parental and hybrids variation for general combining ability (gca) and specific combining ability (sca) variances and effects, respectively, under natural as well as sick plot at both the stages. The fixed effect mean square variances due to general combining ability (gca) and specific combining ability (sca) revealed that magnitude of sca variances exceeded at 30 DAS and that of gca variances excelled sca variances at 60 DAS indicating that final selection should be carried out at latter stage. The lines Pb. 402A3 and Pb. 402B3 with negative gca effects combined significantly better for downy mildew resistance at both the stages. The other lines combined poor to average with most of the non significant gca effects.

Key words: Genetic resistance, downy mildew, pearlmillet, environment

Genetic resistance is the most economic and feasible method for control of DM caused by *Sclerospora graminicola*. The first epiphytotic DM occurred on first popular hybrid HB 3 and caused substantial yield losses particularly in single-cross F<sub>1</sub> hybrids in India. About 50 per cent of the area under pearlmillet cultivation is grown with more than 70 hybrids in India (Rai *et al.*, 2006). The DM incidence has been quit variable on different hybrids, showing more than 90 per cent incidence or even total failure of crop in farmers' fields (Rao *et al.*, 2007). The estimated grain yield losses due to DM are approximately 20-40 per cent (Hash and Witcombe, 2002). The most cost effective management of disease can be obtained by breeding DM resistant hybrids. A

large number of disease resistant hybrids have been developed and deployed and have contributed in arresting the occurrence of widespread DM epidemics since 1990 (Thakur *et al.*, 2006). Several sources of male-sterility inducing cytoplasms e. g. A<sub>1</sub>, A<sub>2</sub>, A<sub>3</sub>, PT732A, ex-Bornu (Gero), *violaceum*, A<sub>4</sub> and A<sub>5</sub> have been reported in pearl millet. But all except two (GHB 316 on A<sub>3</sub>, HHB 216 on A<sub>4</sub> CMS system) hybrids, released in India, are based on A<sub>1</sub> system CMS lines. The use of single source of cytoplasm is risky and has inevitable consequences of conferring "cytoplasmic uniformity" in the hybrid and consequently downy mildew susceptibility of pearlmillet as also argued by Safeeulla (1977). The lines showing stable resistance across the environments non-significant

(year x location) can be useful for understanding the genetic basis of resistance (Thakur *et al.*, 2004) as well as in resistant breeding. The study of genetics of DM using lines-restorers and male sterile lines representing single system of male sterility (A<sub>1</sub>) has been studied and published (Thakur *et al.*, 2004) but the information using different systems of CMS is very scanty. Therefore, the present investigation was carried out to evaluate cytoplasmic effects on mean performance of DM incidence in diverse system of male sterility in natural and sick plot.

### MATERIALS AND METHODS

The material for the present study comprised six male sterile (A-) lines from five systems of cytoplasmic-genic male sterility viz., two male sterile lines from A<sub>1</sub> system (MS 81A<sub>1</sub> and HMS 8A<sub>1</sub>) and one each from  $A_2$  (Pb313 $A_2$ ),  $A_3$  (Pb402 $A_3$ ),  $A_4$  (MS81 $A_4$ ) and A<sub>s</sub> (MS 81A<sub>s</sub>), their corresponding maintainer (B-) lines 81B<sub>1</sub>, HMS8B<sub>1</sub>, Pb313B<sub>2</sub>, Pb402B<sub>3</sub>, 81B<sub>4</sub> and 81B<sub>5</sub> and 12 restorer (R-) lines viz., H90/4-5, H77/833-2, G73-107, 77/245, 77/273, CSSC 46-2, ISK48, ICR161, 77/ 180, 78/711, 77/28-2 and Raj 42. While two A-lines (Pb 313A<sub>2</sub> and Pb 402A<sub>2</sub>) were resistant and other four Alines (MS 81A<sub>1</sub>, HMS 8A<sub>1</sub> MS81A<sub>4</sub> and MS 81A<sub>5</sub>) were susceptible to DM. Four R-lines (G73-107, CSSC 46-2, ICR161 and 77/28-2) were highly resistant, another four lines were resistant (H90/4-5, 78/711, ISK48 and 77/ 180), three were susceptible (H77/833-2, 77/245 and 77/273)) and one R-line (Raj 42) was highly susceptible to DM.

Six male sterile lines and their corresponding six maintainer lines were crossed with 12 restorer lines in a line  $\times$  tester mating design at ICRISAT, Hyderabad, during off season. The 144 hybrids thus produced and their parents were grown separately in contiguous blocks in randomized block design with two replications in eight artificially created environments, six under normal condition viz., early sown non-cut crop ( $E_1$ ,  $E_4$ ), ratoon crop ( $E_2$ ,  $E_5$ ) and late sown non-ratooned ( $E_3$ ,  $E_6$ ) at Research Farm, Bajra Section, Department of Plant Breeding, and two in multiple disease sick plot (SP<sub>1</sub>, SP<sub>2</sub>) at Department of Plant Pathology, CCSHAU, Hisar. The ratoon crop ( $E_2$ ,  $E_5$ ) was cut at a height of approximately 12 cm after 40 days of sowing and left to regenerate.

The plot size was  $2R \times 2.5 \text{ m} \times 0.45 \text{ m}$  with 10 cm intra-row spacing. All the recommended agronomic

practices were followed to raise a good crop. The DM incidence was recorded on all plants in the plot under natural as well as sick plot condition at 30 and 60 days after sowing. The sick plot was created artificially by adding DM susceptible plants with high downy mildew load and has been maintained over the years. In the sick plot, a highly susceptible mixture of 7042S and NHB-3 was grown after every eighth entry as infector row. Fresh sporangia (asexual spores of Sclerospora graminicola) produced on mixture of 7042S and NHB-3 provided enough inoculum load for the infection of test entries. The number of infected plants with downy mildew infection was counted in all environments after 30 and 60 days of sowing except in E<sub>2</sub> and E<sub>5</sub> (30 days of sowing and 30 days after cutting) which was divided by total number of plants in each plot. The data on DM incidence (%) were subjected to angular transformation (Fisher and Yates, 1963) for analysis of variance.

The analysis of variance was carried out in each of the environments according to Federer (1977) and combined analysis of variance was performed according to the model given below:

$$Y_{ijklm} \!=\! \mu \!+\! g_{ij} \!+\! e_{kl} \!+\! (ge)_{ijkl} \!+\! r_{m(lk)} \!+\! \mathring{a}_{ijklm}$$

Further

$$\begin{split} g_{ij} &= p_i + t_j + (pt)_{ij} = pa_i + pb_i + t_j + (pt)a_{ij} + (pt)b_{ij} \\ e_{kl} &= y_k + d_l + (yd)_{kl} \\ (ge)_{ijkl} &= (pe)_{ikl} + (te)_{jkl} + (pt)_{(ij)(kl)} \\ &= (pe)a_{ikl} + (pe)b_{ikl} + (te)_{jkl} + (pt)a_{(ij)(kl)} + (pt)b_{(ij)(kl)} \\ i &= 1, 2, \dots, 12 \text{ (lines)} \\ a &= 1, \dots, 6 \\ b &= 1, \dots, 6 \\ b &= 1, 2, \dots, 12 \text{ (testers)} \\ k &= 1, 2, (years) \\ 1 &= 1, \dots, 4 \text{ (dates)} \\ m &= 1, 2 \text{ (replications)} \end{split}$$

Where, G-genotypes, e-environments, r-replications, p-parents, t-tester, y-year, d-date.

The combining ability analysis was carried out following Kempthorne (1957).

# RESULTS AND DISCUSSION

The genotypes, their component parents and hybrids exhibited significant differences for DM

incidence in all the four treatment environments during both the years at both the observation stages (30 and 60 DAS) Thus, partitioning of the genotypes sum of squares into parents, hybrids and parents vs. hybrids was appropriate. The significant sum of squares due to parents and hybrids also allowed partitioning of these components into lines (A-lines, B-lines, A- vs. B- lines), testers, lines vs. testers and A- x R-hybrids, B- x R-hybrids, A- x R- vs. B- x R-hybrids, respectively. The combined analysis of variance for downy mildew incidence on pooled basis in eight treatments, four each during both the years revealed significant differences for treatments/environments, years, dates, genotypes, parents, hybrids and various other contrasts (Table 1). The non-significance of mean squares due to A- vs. B-

lines, A- x R- vs. B- x R- hybrids and (A- x R- vs. B- x R-) x E at both the observation stages confirms that sterile cytoplasm, of any system, has no specific role to play in DM vulnerability (Kumar and Sagar 2009). The downy mildew incidence in ratoon/regenerated crop was significantly higher than that on early sown non-ratooned or late sown non-ratooned crop during both the years This indicates that the juvenile plant parts of regenerated plants are more vulnerable to this disease. Mohan and Chahal (1989) also reported increased downy mildew incidence in pearlmillet after cutting at 30 days after sowing.

The analysis of variance for combining ability (Table 2) revealed that the mean sum of squares due to lines, testers and lines × testers were highly significant

TABLE 1
Combined analysis of variance for downy mildew incidence in eight environments during two years

Source of	d. f.	Mean sum	of squares
variation		Downy mildew incidence at 30 days (%)	Downy mildew incidence at 60 days (%)
Rep. in environments	8	90.96	103.89
Environments (E)	7	1669.91**	3198.41**
Year	1	883.78**	3251.20**
Date	3	3088.39**	6143.53**
Year × Date	3	513.48**	235.70**
Genotypes (G)	167	485.35**	848.66**
Parents (P)	23	720.60**	1129.85**
Lines	11	483.76**	851.89**
A-lines	5	559.16**	992.12**
B-lines	5	489.54**	879.20**
A- vs. B-lines	1	78.03	14.19
Testers	11	846.11**	1203.50**
Line vs. Tester	1	1945.31**	3377.43**
Hybrids (H)	143	443.39**	799.13**
P vs. H	1	1075.22**	1464.43**
A x R hybrids	71	457.39**	768.04**
B x R hybrids	71	435.62**	840.86**
A x R vs. B x R	1	1.52	43.02
$G \times E$	1169	47.53**	59.58**
$P \times E$	161	69.72**	53.65**
$H \times E$	1001	43.66**	60.09**
$(P \text{ vs. } H) \times E$	7	90.41**	121.32**
$(A \times R) \times E$	497	43.78**	58.08**
$(B \times R) \times E$	497	43.87**	62.77**
$(A \times R \times S \times B \times R) \times E$	7	19.78	13.04
Error	1336	27.11	37.05

<sup>\*\*</sup>Significant at P=0.01 level.

Source of	d. f.	Mean sum of squares								
		$E_{1}$	$\mathbf{E}_{2}$	$E_3$	SP <sub>1</sub>	$E_4$	$E_{5}$	$\mathrm{E}_{_{6}}$	$\mathrm{SP}_2$	
Downy mildew i	ncidence	at 30 days (	0%)							
Lines	11	60.71**	109.11**	47.93*+	238.31**	22.54	45.68	110.51**+	80.90*+	
Testers	11	229.96**	509.96**	386.05**	692.32**	288.65**	490.39**	709.60**	1679.01**	
Lines x testers	121	29.06**	49.19**	28.83**	79.67**	29.04**	34.81**	65.23**	52.53**	
Error	143	18.35	23.08	20.09	38.13	23.22	31.23	25.29	33.03	
gca variance		9.37	26.98	16.44	26.73	9.78	48.02	24.16	38.08	
sca variance		6.98	23.35	8.86	14.80	2.56	8.21	19.36	12.87	
Downy mildew i	incidence	at 60 days (	<sup>9</sup> / <sub>0</sub> )							
Lines	11	92.47**	223.29**	91.37**	366.86**	54.13	147.19*+	160.84**+	105.26*+	
Testers	11	421.64**	1207.20**	775.00**	1066.25**	484.56**	2326.67**	1173.17**	1847.56**	
Lines x testers	121	32.28	68.13**	38.73	75.04**	34.73**	84.48**	87.20**	62.48**	
Error	143	18.31	21.64	20.98	45.44	29.60	68.05	48.48	36.74	
gca variance		4.84	10.85	7.84	16.07	5.27	9.72	14.37	34.48	
sca variance		5.37	13.05	4.37	20.77	2.92	1.79	19.92	9.74	

TABLE 2
Combining ability analysis, gca and sca variances for dowry mildew incidence in natural and sick plot during two years

when tested against the error mean sum of squares in all the environments during both the years showing significant differences among lines, testers and hybrids. However, the mean sum of squares due to lines when tested against interaction (line x tester), the mean squares due to lines exhibited non-signifificance in some of the environments for DM incidence at 30 days (E<sub>3</sub>, E<sub>6</sub>, SP<sub>2</sub>) and DM incidence at 60 days (E<sub>5</sub>, E<sub>6</sub>, SP<sub>2</sub>). This revealed that lines did not show much variation in these environments.

The mean sum of squares due to testers were highly significant for all the traits in all the environments indicating that testers differed more for general combining ability. The highly significant mean sum of squares due to lines × testers suggested significant differences of hybrids for specific combining ability in all environments during both the years. Further, the magnitude of mean sum of squares due to testers was invariably higher as compared to those of lines for most of the traits. This indicated that a large portion of the genetic variability in crosses was accounted by the differences in testers.

The fixed effect variances due to general combining ability (gca) and specific combining ability (sca) presented in Table 3 revealed that the magnitude of gca variances exceeded to that of sca variances for DM at 30 DAS in  $E_3$ ,  $E_4$ ,  $E_5$  and  $SP_5$ ; DM incidence that

at 60 DAS in all the environments. The preponderance of additive variance over non additive variance i. e. higher magnitude of fixed effect gca variance over that of fixed effect sca variance for DM incidence in some of the environments played a significant role in their expression. Singh et al. (1982) and Yadav (1996) also reported similar observations. The fixed effect mean square variances due to general combining ability (gca) and specific combining ability (sca) revealed that magnitude of sca variances exceeded at 30 DAS and that of gca variances excelled sca variances at 60 DAS indicating preponderance of dominance and additive gene effects, respectively. However, the inconsistency of estimates of fixed effect variances in the environments shows that the environment was important in modifying the expression of variances. Deshwal et al. (1998) also reported both additive and non-additive gene effects in the inheritance to DM and also the role of G x E interactions for DM severity.

The estimates of general combining ability effects of parents (lines and testers) for DM in eight environments during two years are presented in Table 3. The negative value of gca effects of this trait implies to combine better for downy mildew resistance. Interestingly the lines 4 (402A), 10 (402B) combined significantly better for downy mildew resistance in most of the environments both at 30 DAS as well as 60 DAS. The other lines

<sup>\*,\*\*</sup>Significant at P=0.05 and P=0.01 levels, respectively.

<sup>+=</sup>Non-significant when tested against line x tester mean sum of squares at P=0.05.

E<sub>1</sub> and E<sub>2</sub>-Unratoon early sown crop, E<sub>3</sub> and E<sub>4</sub>-Ratoon crop, E<sub>3</sub> and E<sub>4</sub>-Unratoon late sown crop, SP<sub>4</sub> and SP<sub>2</sub>-Sick plots.

Estimates of general combining ability effects of lines and testers for downy mildew incidence in natural and sick plots during two years TABLE 3

S. No.	. No. Genotypes		ı	Downy mil	mildew incidence at 30 days (%)	lence at 3	0 days (%				Ď	Downy mildew incidence at 60 days (%)	lew incid	ence at 60	0 days (%	(9)	
		E	$\overline{\mathrm{E}}_{2}$	$ extbf{E}_3$	$SP_1$	E <sub>4</sub>	Ē	E,	$\mathbf{SP}_2$	$\mathbf{E}_{_{1}}$	$\mathbf{E}_{_{2}}$	$\mathbb{E}_{_{3}}$	$\mathbf{SP}_1$	$\mathbf{E}_{_{4}}$	$\mathrm{E_{s}}$	$\mathrm{E}_{_{6}}$	$\mathbf{SP}_2$
	Lines		ı														
1.	81A	-0.01		2.19	2.45	-0.28	-0.22	0.28	0.39	1.87	3.43*	2.07	1.96	-0.59	0.70	-0.21	-1.30
5.	8A	0.15		0.24	1.97	-0.03	1.74	2.27	-1.69	1.08	1.86	0.53	2.10	0.27	0.43	3.45	-1.00
3.	313A	-0.61		-0.13	-2.97	-1.54	-0.32	-1.52	0.84	-1.26	-2.92*	-0.87	-3.76	-2.03	0.12	0.58	0.32
4.	402A	-2.21		-2.67*	-5.04*	1.12	-1.15	-1.83	-1.80	-2.90*	-5.02*	-3.44*	-6.17*	2.46	-2.50	-2.15	-2.38
5.	81A4	-0.07		1.24	-0.17	1.06	-0.42	-0.78	-0.61	-0.53	0.64	98.0	0.58	0.28	-1.84	-1.22	-0.11
9.	81A5	0.13		0.98	5.08*	0.30	0.20	-0.32	1.99	0.40	1.97	1.45	5.99*	-0.64	0.55	-0.56	2.01
7.	81B	3.33*		-0.22	1.00	-0.04	-0.19	-3.05*	-0.97	3.37*	3.67*	1.17	0.62	-0.44	-2.18	-3.30	-1.49
8.	8B	0.05	0.97	0.67	2.30	0.28	2.77	5.38*	-0.26	0.17	1.47	1.31	4.68*	1.99	5.17*	6.35*	0.85
9.	313B	0.00		-0.64	-2.37	-2.18	-1.63	0.61	-1.33	-0.70	-0.74	-1.31	-3.36	-2.83	-2.73	0.33	-0.18
10.	402B	-2.22		-2.49	-4.83*	0.47	-2.02	-0.14	-2.43	-3.36*	-5.47*	-3.74*	-5.75*	1.21	-2.49	-1.59	-3.28
11.	81B4	-0.91		0.33	1.88	0.54	-0.10	-0.54	2.96	-0.17	-0.54	0.22	1.50	0.37	1.91	0.47	3.75*
12.	81B5	2.39		0.49	69.0	0.30	1.34	-0.36	2.90	2.03	1.65	1.74	1.60	-0.06	2.85	-0.99	2.82
13.	H90/4-5	2.32		0.35	2.34	-1.16	-1.71	-3.53*	-5.26*	2.45*	4.73*	1.22	2.50	-0.52	-3.10	-5.52*	-3.69*
	S. E. (d)	1.24		1.29	1.78	1.39	1.61	1.45	1.66	1.24	1.34	1.32	1.95	1.57	2.38	2.01	1.75
	Testers																
14.	H77/833-2	1.71		0.94	5.02*	4.07*	4.56*	*68.9	12.96*	2.96*	7.78*	2.56	6.40*	7.18*	14.62*	8.78*	12.30*
15.	G73-107	-2.58*		-3.06	-5.98*	-2.61	-3.78*	-4.36*	-8.43*	-3.72*	-7.18*	-5.20*	-7.93*	-4.08*	*08.6-	-6.91*	-10.91
16.	CSSC46-2	-2.03		-2.40	-4.30*	-2.24	-2.94	-1.56	-5.05*	-2.71*	-3.54*	-4.08*	-4.71*	-3.25*	-6.92*	-3.18	-5.59*
17.	77/245	3.07*		499.7	4.76*	5.60*	1.97	5.17*	4.89*	4.76*	7.64*	82.6	*66.9	5.60*	5.07*	5.89*	5.21*
18.	78/711	-0.64		-1.67	3.06	-0.94	-0.78	0.91	4.03*	-0.26	0.43	-0.78	3.68	-0.15	-3.62	1.37	3.18
19.	77/273	0.03		-0.03	1.90	-0.63	2.84	0.07	5.66*	-0.35	-0.71	0.52	3.85*	-0.77	7.78*	3.83	6.43*
20.	ICR-161	-2.58*		-3.06*	-5.02*	-2.61	-3.78*	-4.42*	-6.98*	-3.72*	-7.18*	-5.20*	-6.66*	-4.08*	-9.24*	-5.55*	-8.57*
21.	ISK-48	-2.58*		-3.06*	-5.98*	-2.61	-3.78*	-3.85*	-8.08*	-3.72*	-7.18*	-5.20*	-8.96*	-4.08*	-8.75*	-5.56*	-7.77*
22.	77/28-2	-2.58*		-3.06*	-5.24*	-2.61	-3.35*	-4.12*	-7.85*	-3.72*	-7.18*	-4.19*	-5.61*	-4.08*	-8.95*	-5.79*	-7.72*
23.	77/180	-1.49		-1.00	-0.94	-1.23	-0.44	-3.56*	-1.00	-1.51	-1.44	-1.04	-0.63	0.44	4.58	-2.22	1.06
24.	Raj-42	7.37*		8.38*	10.37*	6.93*	11.20*	12.34*	15.11*	9.53*	13.81*	11.59*	11.08*	7.80*	18.35*	14.86*	16.07*
	S. E. (d)	1.24		1.29	1.78	1.39	1.61	1.45	1.66	1.24	1.34	1.32	1.95	1.57	2.38	2.01	1.75

\*Significant at P=0.05 level.  $E_1$  and  $E_2$  and  $E_3$ -Ratoon crop,  $E_3$  and  $E_6$ -Unratoon late sown crop,  $SP_1$  and  $SP_2$ -Sick plots.

combined poor to average with most of the non-significant gca effects. The male sterile lines and maintainers representing different sources of cytoplasm showed substantial differences for combining ability as reported by Kumar *et al.* (1996) as they found that none of the male sterile cytoplasmic sources in general was good combiner for all the traits studied by them.

Tester 15 (G73-107), 20 (ICR161), 21 (ISK48) and 22 (77/28-2) exhibited significant negative gca effects in seven of the eight environments at 30 DAS and in all the environments at 60 DAS, thus are considered as best combiners for DM resistance. Tester 16 (CSSC 46-2) was next best combiner for resistance possessing significant negative gca effects in most of the environments at 60 DAS. On the other hand, tester 24 (Raj 42) turned to be the poorest combiner for disease

resistance, having significant positive gca effects in all the environments both at 30 and 60 DAS. The other poor combiners exhibited significant positive gca effects in more than six of the eight environments were 17 (77/245) and 14 (H77/833-2). The estimates of general combining ability effects of a line are important in a crop like pearlmillet where large number of hybrids are developed and tested every year and, therefore, would help in selecting the parents for using them in hybridization.

The specific combining ability effects worked out for different traits in eight environments during the two years are given in Tables 4 and 5. The crosses with negative effects indicate the desirability for this trait i. e. combining resistance for DM. An appraisal of sca effects for DM incidence at 30 DAS revealed that the number

TABLE 4
Specific combining ability effects and *per se* performance (in parentheses) for some selected hybrids for downy mildew incidence at 30 days (%) during two years

F <sub>1</sub> hybrids	$\mathbf{E}_{_{1}}$	$\mathbf{E}_{2}$	$E_3$	SP <sub>1</sub>	$\mathrm{E}_{_{4}}$	$E_5$	$E_{_6}$	SP <sub>2</sub>	Environmental mean
5 x 13	-4.83	-9.51*	-4.65	-8.15	-2.51	-1.64	-0.91	-2.94	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
5 x 21	0.07	-0.80	-1.24	0.17	-1.06	0.42	-0.60	0.11	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
8 x 15	-0.05	-0.97	-0.67	-2.30	-0.2/8	-2.77	-6.26	-0.12	
	(0.00)	(0.00)	(0.00)	(3.55)	(0.00)	(0.00)	(0.00)	(0.00)	(0.44)
8 x 21	-0.05	-0.97	-0.67	-2.30	-0.28	-2.77	-2.66	-0.47	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(2.00)	(0.00)	(0.25)
4 x 24	-3.24	-9.85*	-8.77	-11.31	2.88	-13.84*	-15.74*	-9.58	
	(2.30)	(0.00)	(0.00)	(0.00)	(8.85)	(0.00)	(0.00)	(8.06)	(2.40)
10 x 24	-7.73	-10.64*	-8.95*	-11.51	-0.71	-12.96*	2.23	-7.42	
	(0.00)	(0.00)	(0.00)	(0.00)	(7.16)	(0.00)	(15.76)	(9.41)	(4.04)
2 x 19	10.71*	4.05	2.03	12.94	3.59	8.64	13.42*	1.73	
	(8.71)	(7.16)	(2.95)	(20.62)	(3.15)	(12.51)	(17.52)	(9.80)	(10.30)
6 x 24	7.45	12.13	6.06	19.82*	7.82	0.56	2.68	7.97	
	(13.06)	(27.22)	(14.21)	(50.03)	(13.36)	(12.16)	(16.56)	(37.73)	(23.04)
8 x 24	0.79	2.89	-6.69	9.90	1.18	8.47	14.86*	0.21	(10.20)
	(9.11)	(13.61)	(3.05)	(28.77)	(9.41)	(25.02)	(44.08)	(21.27)	(19.29)
12 x 24	8.36	13.76*	9.97*	5.03	4.99	3.99	5.06	3.48	
	(17.92)	(29.83)	(18.96)	(15.05)	(10.31)	(16.66)	(19.31)	(31.38)	(19.93)
2 x 14	-4.44	2.75	-4.24	-1.37	1.89	-10.08	-14.39*	-7.11	
	(0.00)	(8.10)	(0.00)	(6.80)	(6.25)	(0.00)	(0.00)	(8.56)	(3.71)
3 x 24	5.41	2.61	1.82	2.42	4.82	2.73	12.38*	6.65	
	(9.91)	10.81)	(8.26)	(11.06)	(8.15)	(20.66)	(34.83)	(20.96)	(12.01)
7 x 14	-1.86	-4.59	1.81	9.31	2.24	-4.56	-9.08	-15.85*	
	(3.35)	(3.15)	(3.20)	(17.91)	(6.66)	(1.65)	(0.00)	(2.65)	(4.82)
7 x 24	3.26	-2.31	-0.10	-1.64	-9.50*	9.50	-8.99	9.34	
	(20.58)	(8.90)	(16.47)	(11.01)	(0.00)	(26.07)	(3.15)	(34.72)	(13.25)
9 x 17	0.33	5.01	3.05	-4.48	-6.03	-4.12	-11.01*	-6.84	
	(3.55)	(9.10)	(8.30)	(1.85)	(0.00)	(0.00)	(0.00)	(3.15)	(3.25)
S. E. (d)	4.28	4.80	4.48	6.17	4.82	5.59	5.04	5.75	

<sup>\*</sup>Significant at P=0.05.

Figures in parentheses are original DM incidence (%) values.

 $E_1$  and  $E_2$ -Unration early sown crop,  $E_2$  &  $E_5$ -Ration crop,  $E_3$  and  $E_6$ -Unration late sown crop,  $SP_1$  and  $SP_2$ -Sick plots.

TABLE 5
Specific combining ability effects and *per se* performance (in parentheses) for some selected hybrids for downy mildew incidence at 60 days (%) during two years

F <sub>1</sub> hybrids	E <sub>1</sub>	E <sub>2</sub>	$\mathbf{E}_{3}$	SP <sub>1</sub>	$E_4$	E <sub>5</sub>	$\mathbf{E}_{_{6}}$	SP <sub>2</sub>	Environmental mean
2 x 15	-1.08	-1.86	-0.53	-3.13	-0.27	-1.13	-4.32	0.14	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
2 x 18	-4.53	-9.47	0.35	-9.44	-4.20	2.57	5.83	.012	` ,
	(0.00)	(0.00)	(2.95)	(2.95)	(0.00)	(7.90)	(14.56)	(9.91)	(4.78)
4 x 20	2.90	5.02	3.44	3.87	-2.46	1.23	-0.08	3.48	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(2.15)	(0.27)
4 x 21	2.90	5.02	3.44	6.17	-2.46	.075	-0.07	4.35	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(3.55)	(0.44)
4 x 22	2.90	5.02	2.42	2.83	-2.46	0.95	0.15	-1.67	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
5 x 13	-5.64	-12.55*	-7.28	-6.92	-3.84	2.37	-1.05	-0.97	
	(0.00)	(0.00)	(0.00)	(2.80)	(0.00)	(5.55)	(0.00)	(4.55)	(1.61)
5 x 15	0.53	-0.64	-0.86	-1.61	-0.28	1.14	0.35	-0.75	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
6 x 13	-6.57	-9.57*	-7.87	-2.59	1.51	2.20	-1.71	-5.99	
	(0.00)	(2.15)	(0.00)	(10.01)	(2.25)	(8.25)	(0.00)	(2.00)	(3.10)
6 x 23	2.81	6.52	11.16*	10.23	0.55	1.31	-0.06	8.81	
	(9.47)	(18.27)	(20.81)	(28.59)	(8.49)	(20.95)	(8.99)	(27.70)	(16.54)
6 x 24	3.88	10.80*	6.89	23.09*	6.41	-6.00	-2.15	10.29	
	(21.58)	(37.80)	(29.18)	(53.16)	(21.70)	(27.45)	(23.98)	(44.19)	(29.33)
7 x 14	-1.17	-2.49	4.25	7.59	3.31	-15.55	-13.27	-14.92*	
	(6.66)	(12.21)	(8.71)	(21.27)	(9.57)	(4.95)	(0.00)	(5.25)	(8.58)
8 x 21	-0.17	-1.47	-1.31	4.68	-1.99	-6.91	-4.47	-1.26	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(2.00)	(1.65)	(0.46)
8 x 22	-0.17	-1.47	-2.32	-8.02	-1.99	-6.71	-8.35	-0.47	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(2.00)	(2.25)	(0.28)
10 x 17	-5.12	-9.35*	-5.48	-4.22	5.81	4.73	7.28	3.26	
	(0.00)	(0.00)	(3.35)	(3.55)	(18.26)	(13.46)	(15.56)	(13.16)	(8.42)
10 x 20	3.36	5.47	3.74	3.45	-1.21	1.23	9.53	5.20	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(5.61)	(2.80)	(1.05)
10 x 21	3.36	5.47	3.74	5.75	-1.21	0.74	-0.63	-0.72	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
10 x 22	3.36	5.47	2.73	2.40	-1.21	0.94	-0.41	2.85	
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(1.66)	(0.21)
10 x 24	-9.89*	-9.76*	-8.10	-9.53	-3.81	-6.53	1.71	-7.49	
	(0.00)	(3.35)	(2.65)	(2.50)	(7.16)	(15.91)	(20.32)	(12.50)	(85)
11 x 13	-5.99	-11.37*	-6.64	-12.96	1.81	4.57	-2.73	2.50	
10 10	(0.00)	(0.00)	(0.00)	(0.00)	(3.35)	(9.01)	(0.00)	(9.46)	(2.73)
12 x 13	-8.19	-13.56*	-8.16	-13.06	8.31	2.50	-1.28	5.60	(0.00)
10 01	(0.00)	(0.00)	(0.00)	(0.00)	(7.15)	(11.91)	(0.00)	(12.31)	(3.92)
12 x 24	9.20*	13.23*	9.65*	0.59	3.00	10.82	10.47	6.21	(04.4=)
G F (1)	(22.47)	(40.73)	(27.97)	(19.11)	(10.31)	(51.77)	(34.53)	(42.43)	(31.17)
S. E. (d)	4.28	4.65	4.58	6.74	5.44	8.25	6.97	6.06	

<sup>\*</sup>Significant at P=0.05.

Figures in parentheses are original DM incidence (%) values.

 $E_1$  and  $E_4$ –Unratoon early sown crop,  $E_2$  and  $E_5$ –Ratoon crop,  $E_3$  and  $E_6$ –Unratoon late sown crop,  $SP_1$  and  $SP_2$ –Sick plots.

of  $F_1$  crosses showing significant negative effects was very low. The number of crosses showing significant negative sca effects was 5, 1, 1, 2, 6 and 1 in  $E_2$ ,  $E_3$ ,  $E_4$ ,  $E_5$ ,  $E_6$  and  $SP_2$ , respectively. But in other environments ( $E_1$ ,  $SP_1$  and  $SP_2$ ) none of the crosses exhibited significant sca effects. On the other hand, the number of crosses showing significant positive sca effects was 4, 7, 3, 4,

5, 8 and 3 in  $E_1$ ,  $E_2$ ,  $E_3$ ,  $SP_1$ ,  $E_4$ ,  $E_6$  and  $SP_2$ , respectively. The  $F_1$  cross  $4 \times 24$  involving resistant x susceptible, exhibited significant and negative sca effects in three of the eight environments ( $E_2$ ,  $E_5$  and  $E_6$ ). The  $F_1$  hybrids  $2 \times 14$  ( $E_6$ ),  $3 \times 24$  ( $E_6$ ),  $5 \times 13$  ( $E_2$ ),  $7 \times 14$  ( $SP_2$ ),  $7 \times 24$  ( $E_4$ ),  $9 \times 17$  ( $E_6$ ) and  $10 \times 24$  ( $E_2$ ,  $E_3$ ) exhibited significant negative sca effects in at least one of the eight

environments, while crosses  $1 \times 13$ ,  $2 \times 19$ ,  $3 \times 14$ ,  $6 \times 24$ ,  $9 \times 13$  and  $12 \times 24$  turned to be the poor combination as showing significant positive sca effects at least in two of the eight environments.

The number of crosses exhibiting significant sca effects at 60 days was also low. A very few number of crosses 1, 9, 1 and 1 in E<sub>1</sub>, E<sub>2</sub>, E<sub>6</sub> and SP<sub>2</sub>, respectively, envisaged significant negative sca. The crosses  $2 \times 18$ , 5  $\times$  13, 6  $\times$  13, 11  $\times$  13 and 12  $\times$  13 involving susceptible x resistant exhibited significant negative sca effects in E<sub>2</sub> and average sca in other environments. It shows that at least one resistant parent preferably the pollinator confers resistance in hybrid as also observed in field and proven with downy mildew resistance performance of HHB 50 (MS 81 x H90/4-5) released by CCSHAU, Hisar in 1988. H90/4-5 imparted resistance for a couple of years. Basavaraju et al. (1981) also reported about the role of pollinator in DM resistance. The crosses  $4 \times 20$ ,  $4 \times 21$ ,  $4 \times 22$ ,  $10 \times 20$ ,  $10 \times 21$  and  $10 \times 22$  involving resistant × resistant parents did not show significant negative sca effects in any of the environments; however, all the crosses expressed the resistance to downy mildew. The non significant negative sca effects of these hybrids could be due to greater role of epistasis in mechanism of downy mildew resistance as was reported by Basavaraju et al. (1980). The results show that it is not always necessary only crosses derived from resistant × resistant parents will give significant negative sca effects.

It has been already observed that the cytoplasm is not necessarily a culprit in downy mildew susceptibility in pearl millet as observed by Kumar and Sagar (2009) and Sagar and Kumar (2004). The genetics of downy mildew has been reported to be additive and non-additive with latter's preponderance and environment influences. Thus, there is need to identify downy mildew resistant lines by testing parental lines across the locations. Therefore, the stable downy mildew resistant hybrids can be developed using resistant lines and tested across the environments. The lines G73-107, CSSC 46-2, ICR 161 ISK 48 and 77/28-2 which were highly resistant to DM and also agronomical proved better can be intermated to develop a base population for testing over years and location for development of downy mildew resistant and agronomical superior lines. As the hybrids are highly heterozygous, homogeneous populations probably highly virulent pathogen biotypes, which might have not been virulent on homozygous inbreds would have become virulent on hybrids. Therefore, to maintain resistance in production systems-inbreds, landraces, open pollinated and hybrid populations, there is need to find new resistant genes and deploying them in appropriate cultivars. So, to slow or halt the erosion of available resistant genes, the dynamic multiple population approach with multiple strategies is suggested.

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