Influence of Flowering Event Factors in Cytoplasmic Male Sterile Lines and F_1 Hybrids on Infection by *Claviceps fusiformis* in Pearl Millet

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ABSTRACT

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The influence of flowering event factors, such as length of protogyny period, and time between full protogyny (FP) and anthesis initiation (AI) on infection by *Claviceps fusiformis* was studied in seven cytoplasmic male sterile (CMS) lines (A lines), their corresponding maintainer lines (B lines), nine pollinator lines (R lines), and their 64 F_1 hybrids (A × R and B × R) of pearl millet (*Pennisetum glaucum*). Three of the seven A lines had significantly longer protogyny than their corresponding B lines. The protogyny periods in R lines (range 38–84 hr) were shorter than in B lines (range 45–135 hr). Significant positive correlations were found between protogyny, the FP-AI period, and ergot severity in susceptible A or B lines and their F_1 hybrids, regardless of the susceptibility or resistance of R lines, but not in resistant A, B, and R lines and their hybrids. Seed set was generally more in B × R hybrids than in A × R hybrids, indicating poor restoration ability of R lines. Stigmas were longer in susceptible lines and their hybrids than in resistant lines and hybrids. The susceptibility of F_1 hybrids was closely associated with flowering event factors of susceptible A lines. No such relationship was found in resistant lines. This indicates that there may be a factor or factors independent of flowering events governing resistance in A lines and their hybrids.

Flowering in pearl millet (*Pennisetum glaucum* (L.) R. Br.) is protogynous, i.e., stigmas emerge and mature before the stamens (3). Infection of pearl millet florets by the ergot pathogen (*Claviceps fusiformis* Loveless) occurs through fresh stigmas, and rapid pollination prevents infection by inducing stigmatic constriction and withering (7,10). F_1 hybrid plants, because of their genetic uniformity, have synchronous tillering and flowering and are generally more susceptible to ergot than the open-pollinated cultivars, which are heterogenous and asynchronous (9).

Thakur et al (4) established a positive association of cytoplasmic male sterility (CMS) with ergot susceptibility of F_1 hybrids in pearl millet. Regardless of the ergot reaction of male parents, F_1 hybrids made on ergot-susceptible CMS lines (A lines) were always susceptible. In our earlier study (5) with some fertile lines of pearl millet, flowering events, such as length of protogyny period (time between initiation of stigma emergence and anther emergence on a panicle), time between full protogyny (>75% stigmas emerged = ergot inoculation stage) and anthesis initiation (FP-AI period), and stigma length were positively correlated to ergot severity. Our objective was to determine the relationship between A lines and their F_1 hybrids for the flowering event factors that influence ergot susceptibility.

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MATERIALS AND METHODS

Pearl millet lines and F_1 hybrids. In three field experiments at ICRISAT Center, we evaluated seven A lines (six susceptible to ergot [>10% ergot severity] and one resistant [<10% ergot severity]), their corresponding maintainer lines (B lines), and nine pollinator or restorer lines (R lines) (six susceptible and three resistant) and their 64 F_1 (A × R and B × R) hybrids (Table 1).

In the first experiment, conducted during the 1988 rainy season (July–September), six A lines (852A, 5054A, 81A, 834A, 843A, and 841A) and their corresponding B lines, all susceptible to ergot; eight R lines, two resistant (ICMPES 1 and ICMPES 2) and six susceptible to ergot (J 104, ICMP 851009, ICMP 85417, ICMP 451, ICMP 423, and ICMP 501), and their 36 F_1 hybrids were evaluated. In the second experiment, conducted during the 1989 dry season (January–March 1989), three A lines (852A, 81A, and 841A), their corresponding B lines, and five R lines (three susceptible and two resistant from the first experiment) and their 16 F_1 hybrids were evaluated. In the third experiment, also conducted during the 1989 dry season, one ergot-resistant A line (ER-A), one susceptible line (81A), their corresponding B lines, and three R lines (two susceptible [ICMP 423 and ICMP 451] and one resistant [ICMPES 23]), and their 18 F_1 hybrids were evaluated.

Planting and experimental design. The experiments, in a randomized block design, were conducted in the ergot nursery during both seasons. Each entry was grown in a two-row plot 4 m long in two replications. Rows were 75 cm apart with 15 cm between plants.

Ergot inoculation. Panicles, at the full protogyny stage, were inoculated by spraying with an aqueous conidial suspension (1×10^6 conidia per milliliter) of *C. fusiformis* (8). Conidia were obtained from honeydew produced on previously inoculated panicles of a susceptible cultivar in a greenhouse. The panicles were protected from external pollen by covering them with parchment paper selfing bags before and after inoculation. In each plot, five panicles were inoculated. High relative humidity (>80%) was maintained by sprinkler irrigations twice a day.

Observations. Panicles were scored for ergot severity 20 days after inoculation using an assessment key (7). An entry was called susceptible when mean ergot severity was 10% or more. Observations on protogyny period, FP-AI period, and seed set were recorded for parental lines and hybrids, in each plot, on five randomly selected panicles that were bagged at the boot-leaf stage. For observations on protogyny and FP-AI periods, panicles were examined daily between 0900 and 1000 hours and 1530 and 1630 hours by briefly removing the bags.

Measurements of stigma length were taken on pot-grown plants of 81A/B, ER A/B (81A × ICMPE 134-6-9 [BC_6]/81B × ICMPE 134-6-9 [BC_6]), ICMPES 23, and their four F_1 hybrids. Detached florets were spread on white paper on a lab bench and the length of stigma protruding out from the glume was measured with a ruler under a magnifying glass. Fifty florets from five main panicles representing upper, middle, and lower parts of the panicle were sampled from each entry for stigma length measurement. Florets were sampled between 0830 and 0900 hours to obtain fresh stigmas.

Data analysis. Estimates of cytoplasmic effects on protogyny, FP-AI period, and ergot severity were determined by computing differences between A and B lines and between A × R and B × R hybrids. Contributions of A and B lines to their F_1 hybrids were also estimated

Table 1. Periods of protogyny (hr)[z] in pearl millet cytoplasmic male sterile (A) lines, their maintainer (B) and pollinator (R) lines, and their F_1 (A × R and B × R) hybrids grown in field experiments at ICRISAT Center

Cross (A/B × R)	Line or hybrid						
	A	B	R	A−B	A × R	B × R	(A × R) − (B × R)
Experiment 1: 1988 rainy season							
852A/B × ICMPES 1	85	94	41	−9	99	60	39
× ICMPES 2			52		103	75	28
× ICMP 851009			38		83	69	14
5054A/B × ICMPES 1	91	54	41	37	107	58	49
× ICMPES 2			52		86	48	38
× J 104			44		74	59	15
81A/B × ICMPES 1	103	106	41	−3	88	70	18
× ICMPES 2			52		88	66	22
× ICMP 451			47		98	62	36
834A/B × ICMPES 1	69	45	41	24	64	35	29
× ICMPES 2			52		69	39	30
× ICMP 501			67		63	53	10
843 A/B × ICMPES 1	119	71	41	48	91	48	43
× ICMPES 2			52		85	43	42
× ICMP 85417			84		84	76	8
841A/B × ICMPES 1	70	67	41	3	61	53	8
× ICMPES 2			52		61	44	17
× ICMP 423			56		107	50	57
LSD ($P = 0.05$)	16.4	12.3	12.1	20.6	15.7	10.5	17.6
Experiment 2: 1989 dry season							
852A/B × ICMP 851009	124	108	58	16	106	87	19
852A/B × ICMPES 1			46		124	71	53
81A/B × ICMP 451	126	133	53	−7	118	82	36
81A/B × ICMPES 1			46		93	80	13
81A/B × ICMPES 2			45		91	80	11
841A/B × ICMP 423	93	83	65	10	111	75	36
841A/B × ICMPES 1			46		78	79	−1
841A/B × ICMPES 2			45		75	73	2
LSD ($P = 0.05$)	13.6	17.2	14.3	20.9	13.9	11.4	19.6
Experiment 3: 1989 dry season							
81A/B × ICMP 423	129	135	62	−6	99	101	−2
81A/B × ICMP 451			55		118	86	32
81A/B × ICMPES 23			43		111	70	41
ER-A/B × ICMP 423	71	52	62	19	86	53	33
ER-A/B × ICMP 451			55		71	53	18
ER-A/B × ICMPES 23			43		78	48	30
LSD ($P = 0.05$)	23.0	11.0	11.2	24.4	14.8	11.3	17.5

[z] Based on mean of five panicles from each of two replications.

Table 2. Comparison of protogyny periods (hr) of pearl millet F_1 (A × R and B × R) hybrids with their midparental (A + R/2 and B + R/2) values

A/B line[x]	Resistant R line (46)[w]		Susceptible R line (51)[x]	
	F_1	Midparent	F_1	Midparent
852A (104)[y]	107	75	94	77
852B (101)[y]	70	73	78	76
81A (119)[z]	93	82	105	85
81B (125)[z]	73	85	83	88
841A (81)[y]	68	63	109	66
841B (75)[y]	62	60	62	63
5054A (91)	96	68	74	71
5054B (54)	53	50	59	52
834A (69)	66	57	63	60
834B (45)	37	45	53	48
843A (119)	89	82	84	85
843B (71)	47	58	76	61
ER-A (71)	78	58	79	61
ER-B (52)	48	49	53	51

[v] Values in parentheses are protogyny periods of parental lines.
[w] Mean of three resistant lines (ICMPES 1, ICMPES 2, and ICMPES 23) from three experiments.
[x] Mean of six susceptible lines (ICMP 851009, J 104, ICMP 451, ICMP 501, ICMP 432, and ICMP 85417) from three experiments.
[y] Mean of two experiments.
[z] Mean of three experiments.

by computing the midparental values and comparing these with the corresponding F_1 values for each trait. Relationships of A lines with A × R hybrids for protogyny, FP-AI period, and ergot severity were indicated by plotting the mean values of A lines against the mean values of A × R hybrids with reference to a diagonal line drawn on a 1:1 basis. This is based on the assumption that A > A × R when points lie above the diagonal line, and A < A × R when points lie below the diagonal line (2).

RESULTS

Effect of CMS on protogyny period. Of the seven A/B pairs, protogyny periods in three A lines (5054A, 834A, and 843A) were significantly longer than their corresponding B lines (Table 1). Most R lines had shorter protogyny than A or B lines. A × R hybrids usually had longer protogyny than B × R hybrids. Of the 32 A/B × R hybrids, 20 A × R hybrids had significantly longer protogyny than their corresponding B × R hybrids. In 81A/B lines, with similar protogyny between A and B lines, six of the nine 81A × R hybrids had longer protogyny than 81B × R hybrids. Similar results were obtained for 852A/B × R, 841A/B × R, and ER-A/B × R hybrids. Pooled data from three experiments showed strong dominance influence of A lines on F_1 (A × R) hybrids. In all (except one) F_1 hybrids made using resistant or susceptible R lines, protogyny periods of hybrids were longer than the midparent values and were close to or more than the highest values of A lines, but the same did not hold for B lines and B × R hybrids (Table 2).

Effect of CMS on FP-AI period. In four of the seven A/B pairs (5054A/B, 834A/B, 843A/B, and 81A/B), A lines had significantly longer FP-AI periods than their corresponding B lines (Table 3). In 81A, the FP-AI period was significantly longer than in 81B in the second and third experiments conducted during the cool, dry months of January–March but not in the rainy season. In 19 of the 32 A/B × R hybrids, the FP-AI periods were longer in A × R than in B × R hybrids. The FP-AI periods of F_1 (A × R) hybrids (except two) were longer than respective R lines or the midparent values and were close to A line values, but the same did not hold for B lines and B × R hybrids (Table 4).

Effect of CMS on ergot severity. Ergot severity in susceptible A lines varied from 83 (81A) to 99% (5054A) and in B lines from 51 (834B) to 94% (852B), whereas in the resistant A and B lines, it was 4 and <1%, respectively (Table 5). The susceptible R lines showed ergot severity of 22 (ICMP 423) to 88% (J 104), whereas two resistant R lines showed 1–9% in different experiments. In three of the six susceptible A/B pairs, ergot severity in

A lines was significantly greater than in their corresponding B lines. Although 841A showed significantly greater ergot severity than 841B in the rainy season, it did not in the dry season.

A × R hybrids had more ergot than B × R hybrids, with 14 of the 32 crosses showing more ergot in A × R than in B × R hybrids. Of 17 crosses involving resistant R lines, 11 showed more ergot in A × R hybrids than in B × R hybrids, and ergot severity of the hybrids exceeded the respective midparental values. In crosses with resistant female parents (A/B lines) and susceptible male parents (R lines), the hybrids were also susceptible, but such hybrids showed significantly less ergot than those having both parents susceptible. In a cross where both parents were resistant, the hybrid was also resistant (Table 5, experiment 3). With few exceptions, all F_1 hybrids (A × R or B × R) had higher ergot than the midparent values or R lines and were either close to or higher than the most susceptible A line parents (Table 6).

Effect of CMS on seed set. All of the A lines were nearly true breeding with 0.0–0.4% seed set under selfing. In B lines, seed set varied from 26 to 93%; in 81B, it was 64% in the rainy season and only 14% in the dry season (Table 7). R lines generally had good seed set of 58–99%. When two ergot-resistant lines, ICMPES 1 and ICMPES 2, were used as R lines, the hybrids on 852A, 5054A, 81A, and 843A produced very little seed, indicating that these may be poor restorers to these A lines, but produced good seed on 834A and 841A. The poor restoration ability of these R lines was more evident during the dry season than in the rainy season. Similarly, an ergot-resistant ICMPES 23 also behaved as a poor restorer on 81A and ER-A during the dry season. B × R hybrids generally produced much more seed than A × R hybrids.

Effect of CMS on stigma length. In the ER A/B pair, stigma length of the A line (2.3 mm) was significantly greater than its corresponding B line (1.2 mm), but the same was not true with the 81A/B pair. Stigmas of a susceptible 81A/B pair (3.9/4.0 mm) were significantly longer than its resistant derivative ER-A/B pair (1.2/2.3 mm), and ICMPES 23 had the shortest stigma length (0.9 mm). A × R hybrids (81A × ICMPES 23 and ER-A × ICMPES 23) had significantly longer stigmas than their corresponding B × R hybrids.

Relationship between flowering event factors. Highly significant, positive correlations were found between protogyny and FP-AI periods ($r = 0.84$, $P \leq 0.01$), protogyny and ergot severity ($r = 0.63$, $P \leq 0.01$), and FP-AI period and ergot severity ($r = 0.60$, $P \leq 0.01$). Significant negative correlations were found between protogyny and seed set ($r = -0.63$, $P \leq 0.01$) and ergot severity and seed

Table 3. Time period (hr)[z] between ergot inoculation stage (full protogyny = FP) and anthesis initiation (AI) in pearl millet cytoplasmic male sterile (A) lines, their maintainer (B) lines and pollinator (R) lines, and their F_1 (A × R and B × R) hybrids in field experiments at ICRISAT Center

	Line or hybrid						
Cross (A/B × R)	A	B	R	A−B	A × R	B × R	(A × R) − (B × R)
Experiment 1: 1988 rainy season							
852A/B × ICMPES 1	80	71	36	9	82	52	30
× ICMPES 2			35		72	43	29
× ICMP 851009			30		60	52	8
5054A/B × ICMPES 1	79	50	36	29	75	36	39
× ICMPES 2			35		67	29	38
× J 104			45		59	46	13
81A/B × ICMPES 1	88	76	36	12	73	59	14
× ICMPES 2			35		73	51	22
× ICMP 451			39		70	35	35
834A/B × ICMPES 1	49	34	36	15	56	30	26
× ICMPES 2			35		47	33	14
× ICMP 501			51		43	37	6
843A/B × ICMPES 1	95	53	36	42	65	30	35
× ICMPES 2			35		72	29	43
× ICMP 85417			49		80	55	25
841A/B × ICMPES 1	55	42	36	13	60	50	10
× ICMPES 2			35		68	51	17
× ICMP 423			51		65	37	28
LSD ($P = 0.05$)	12.6	8.2	8.4	14.5	13.0	9.0	15.5
Experiment 2: 1989 dry season							
852A/B × ICMP 851009	75	81	41	−6	77	49	28
852A/B × ICMPES 1			31		88	62	26
81A/B × ICMP 451	104	84	47	20	93	62	31
81A/B × ICMPES 1			31		69	59	10
81A/B × ICMPES 2			34		65	53	12
841A/B × ICMP 423	72	55	42	17	75	61	14
841A/B × ICMPES 1			31		53	59	−6
841A/B × ICMPES 2			34		52	55	−3
LSD ($P = 0.05$)	13.0	11.6	8.6	18.2	10.9	10.7	15.8
Experiment 3: 1989 dry season							
81A/B × ICMP 423	108	78	43	29	89	87	2
81A/B × ICMP 451			42		89	66	23
81A/B × ICMPES 23			33		91	51	40
ER-A/B × ICMP 423	41	29	43	12	58	35	23
ER-A/B × ICMP 451			42		60	49	11
ER-A/B × ICMPES 23			33		54	35	19
LSD ($P = 0.05$)	13.3	13.6	10.1	20.8	11.2	10.6	16.2

[z] Based on mean of five panicles from each of two replications.

Table 4. Comparison of full protogyny-anthesis initiation (FP-AI) periods (hr) of pearl millet F_1 (A × R and B × R) hybrids with their midparental (A + R/2 and B + R/2) values

	Resistant R line (46)[w]		Susceptible R line (51)[x]	
A/B line[y]	F_1	Midparent	F_1	Midparent
852A (77)[y]	81	55	68	60
852B (76)[y]	52	55	50	60
81A (100)[z]	74	67	85	72
81B (79)[z]	53	56	62	61
841A (63)[y]	66	48	70	53
841B (48)[y]	55	41	49	46
5054A (79)	71	56	59	61
5054B (50)	32	42	46	47
834A (49)	51	41	43	46
834B (34)	32	34	37	39
843A (95)	68	64	80	69
843B (53)	30	43	55	48
ER-A (41)	54	37	59	42
ER-B (29)	35	31	42	36

[y] Values in parentheses are protogyny periods of parental lines.
[w] Mean of three resistant lines (ICMPES 1, ICMPES 2, and ICMPES 23) from three experiments.
[x] Mean of six susceptible lines (ICMP 851009, J 104, ICMP 451, ICMP 501, ICMP 432, and ICMP 85417) from three experiments.
[y] Mean of two experiments.
[z] Mean of three experiments.

Table 5. Ergot severity (%)[z] of pearl milet cytoplasmic male sterile lines (A), their maintainer lines (B) and pollinator lines (R), and their F_1 (A × R and B × R) hybrids in field experiments at ICRISAT Center

	Line or hybrid						
Cross (A/B × R)	A	B	R	A−B	A × R	B × R	(A × R) − (B × R)
Experiment 1: 1988 rainy season							
852A/B × ICMPES 1	84	94	3	−10	93	76	17
× ICMPES 2			<1		89	73	16
× ICMP 851009			30		85	79	6
5054A/B × ICMPES 1	99	76	3	23	94	64	30
× ICMPES 2			<1		96	50	46
× J 104			88		99	95	4
81A/B × ICMPES 1	97	90	3	7	92	61	31
× ICMPES 2			<1		78	26	52
× ICMP 451			60		81	85	−4
834A/B × ICMPES 1	87	51	3	36	82	38	44
× ICMPES 2			<1		83	30	53
× ICMP 501			75		62	72	−10
843A/B × ICMPES 1	90	94	3	−4	89	15	74
× ICMPES 2			<1		96	48	48
× ICMP 85417			93		97	96	1
841A/B × ICMPES 1	87	68	3	19	94	50	44
× ICMPES 2			<1		82	46	36
× ICMP 423			63		97	84	13
LSD ($P = 0.05$)	13.2	10.9	12.7	18.2	9.6	14.4	18.1
Experiment 2: 1989 dry season							
852A/B × ICMP 851009	80	87	65	−7	81	78	3
852A/B × ICMPES 1			9		85	82	3
81A/B × ICMP 451	86	94	28	−8	69	73	−4
81A/B × ICMPES 1			9		83	48	35
81A/B × ICMPES 2			1		84	75	9
841A/B × ICMP 423	91	80	45	11	95	86	9
841A/B × ICMPES 1			9		80	81	−1
841A/B × ICMPES 2			1		91	76	14
LSD ($P = 0.05$)	9.1	9.4	14.3	13.4	7.9	12.3	14.6
Experiment 3: 1989 dry season							
81A/B × ICMP 423	83	95	22	−12	91	92	−1
81A/B × ICMP 451			45		86	81	5
81A/B × ICMPES 23			1		78	35	43
ER-A/B × ICMP 423	4	<1	22	4	68	24	44
ER-A/B × ICMP 451			45		45	30	15
ER-A/B × ICMPES 23			1		9	1	8
LSD ($P = 0.05$)	9.2	2.6	14.1	9.8	8.5	11.3	13.5

[z] Based on mean of five panicles from each of two replications.

Table 6. Comparison of ergot severity (%) of pearl millet F_1 (A × R and B × R) hybrids with their midparental (A + R/2 and B + R/2) values

	Resistant R line (46)[w]		Susceptible R line (51)[x]	
A/B line[v]	F_1	Midparent	F_1	Midparent
852A (82)[y]	89	42	83	70
852B (90)[y]	77	46	78	74
81A (89)[z]	83	46	82	74
81B (93)[z]	49	48	83	76
841A (89)[y]	87	46	96	74
841B (74)[y]	63	38	85	66
5054A (99)	95	50	99	79
5054B (76)	57	39	95	67
834A (87)	82	45	62	73
834B (51)	34	27	72	55
843A (90)	92	46	97	74
843B (94)	31	48	96	76
ER-A (4)	9	3	56	31
ER-B (1)	1	2	27	30

[v] Values in parentheses are protogyny periods of parental lines.
[w] Mean of three resistant lines (ICMPES 1, ICMPES 2, and ICMPES 23) from three experiments.
[x] Mean of six susceptible lines (ICMP 851009, J 104, ICMP 451, ICMP 501, ICMP 432, and ICMP 85417) from three experiments.
[y] Mean of two experiments.
[z] Mean of three experiments.

set ($r = -0.56$, $P \leq 0.05$). A lines ha strong dominance influence on their × R hybrids for protogyny, FP-AI pe riod, and ergot severity. Across thre experiments, the mean ergot severity o eight of the 11 A × R hybrids was eithe significantly more than or close to tha of A lines (Fig. 1).

DISCUSSION

The roles of pollen (7,10) and CMS (4) in ergot susceptibility of F_1 hybrid in pearl millet are well established. I relation to ergot infection in pearl millet pollination-fertilization is the end poin in a time sequence of flowering event that include stigma emergence, full pro togyny (the ergot inoculation stage) anthesis initiation, and pollination-fer tilization. The time factors in the se quence of flowering, such as lengths o protogyny and FP-AI periods, are crucial for ergot infection or seed set. On a bagged panicle, the time duration of flowering events dictates the extent of ergot severity or seed set (5).

It is evident from the results that longer protogyny and FP-AI periods, which are associated with ergot susceptibility, are heritable from A lines to their hybrids. A × R hybrids have longer protogyny and FP-AI periods and are mostly more susceptible than their corresponding B × R hybrids. The extent of variability for these traits in different A/B pairs and their close association with hybrids indicate the existence of genetic differences among them. Highly significant r values based on data from three experiments indicate the stability of traits and can be generalized for similar environments elsewhere. Very high r values for ergot severity are attributable to discontinuous clustered distribution among the lines.

The length of FP-AI period is critical in an inoculated, bagged panicle for infection. In susceptible lines, when anthesis and pollination occur within 16 hr after inoculation, ergot infection is greatly reduced, but when pollination is delayed, ergot infection is increased (7). In all A, B, and R lines and their hybrids, the FP-AI periods were greater than 16 hr; consequently, all were susceptible to ergot, except for the ergot-resistant A, B, and R lines. The hybrids ER-A/B × R (susceptible) were susceptible, but ER-A × R showed more susceptibility than ER-B × R hybrids. When both A/B and R lines were resistant, the hybrids were also resistant. This confirms our earlier observations that to incorporate ergot resistance in hybrids, additive genetic factors from both parents are essential (1,6,8). The association of stigma length to ergot infection is evident, although from limited observations in this study. Under wet weather, which is less favorable for pollen production, susceptible hybrids with longer stigmas expose greater surface area to ergot spores than

Table 7. Seed set (%)[1] in selfed panicles of pearl millet cytoplasmic male sterile lines (A), their maintainer (B) and pollinator lines (R), and their F_1 (A × R and B × R) hybrids in field experiments at ICRISAT Center

Cross (A/B × R)	Line or hybrid				
	A	B	R	A × R	B × R
Experiment 1: 1988 rainy season					
852A/B × ICMPES 1	0.3	26	90	2	99
× ICMPES 2			92	14	99
× ICMP 851009			99	97	99
5054A/B × ICMPES 1	0.01	63	90	35	97
× ICMPES 2			92	1	96
× J 104			91	54	92
81A/B × ICMPES 1	0.1	64	90	6	99
× ICMPES 2			92	3	88
× ICMP 451			93	83	95
834A/B × ICMPES 1	0.3	77	90	96	97
× ICMPES 2			92	64	83
× ICMP 501			67	98	99
843A/B × ICMPES 1	0	93	90	18	99
× ICMPES 2			92	<1	99
× ICMP 85417			79	99	99
841A/B × ICMPES 1	0.4	87	90	49	96
× ICMPES 2			92	23	93
× ICMP 423			59	29	98
LSD ($P = 0.05$)	0.31	16.9	14.7	22.3	7.5
Experiment 2: 1989 dry season					
852A/B × ICMP 851009	0.1	55	60	7	71
852A/B × ICMPES 1			90	0	95
81A/B × ICMP 451	0	14	92	70	97
81A/B × ICMPES 1			90	0	98
81A/B × ICMPES 2			89	0	91
841A/B × ICMP 423	0	34	65	0	69
841A/B × ICMPES 1			90	<1	95
841A/B × ICMPES 2			89	1	96
LSD ($P = 0.05$)	0.2	24.7	16.2	12.8	13.1
Experiment 3: 1989 dry season					
81A/B × ICMP 423	0	14	73	0	42
81A/B × ICMP 451			96	80	96
81A/B × ICMPES 23			58	1	97
ER-A/B × ICMP 423	0	91	73	0	92
ER-A/B × ICMP 451			96	32	87
ER-A/B × ICMPES 23			58	1	84
LSD ($P = 0.05$)	0.0	7.8	13.1	10.4	13.5

[1] Based on mean of five panicles from each of two replications.

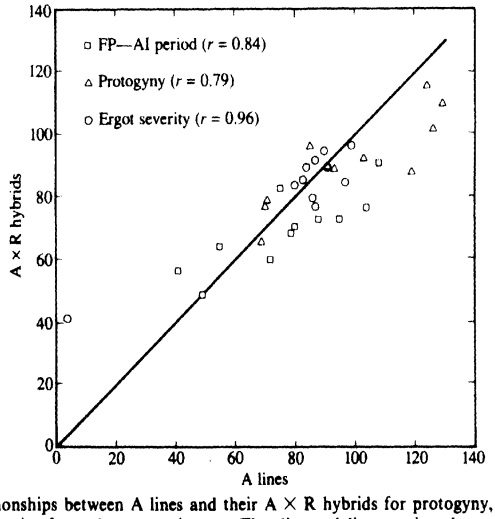

Fig. 1. Relationships between A lines and their A × R hybrids for protogyny, FP-AI period, and ergot severity from three experiments. The diagonal line passing through the origin is drawn on a 1:1 basis. Heritability of traits is considered high when the points are located above or close to the line and low when points are located below or away from the line (based on 11 observations from three experiments, involving seven A lines and their hybrids).

resistant hybrids having shorter stigmas. For example, a hybrid (81A × ICMPES 23) with a stigma length of 3.9 mm was highly susceptible (78% ergot) compared with a hybrid (ER-A × ICMPES 23) with a stigma length of 3.1 mm, which was resistant (9% ergot).

Seed set under selfing was generally higher in B × R hybrids than in A × R hybrids, indicating poor restoration ability, possibly including stigma withering attributable to aging before anthesis. High ergot resistance and poor seed set in the ER-A × ICMPES 23 hybrid indicates good combining ability for ergot resistance but poor restoration for seed set. The results clearly indicate that in susceptible A, B, and R lines, protogyny, FP-AI period, and stigma length are associated with ergot susceptibility and are easily inherited by hybrids from their A lines, thus making hybrids highly susceptible. They also indicate that in contrast, in resistant A, B, and R lines and their hybrids, protogyny and FP-AI period were not related to ergot susceptibility; therefore, resistance in these lines and their hybrids may not be based on the pollination-stigma withering mechanism. Finally, the results indicate that resistant hybrids can be produced if both parents have high combining ability for resistance and fertility restoration in the F_1.

At the ICRISAT Center, some progress has been made in breeding an ergot-resistant A line by incorporating resistance in the established A line, 81A, through backcross breeding (K. N. Rai, ICRISAT, *personal communication*). Its F_1 hybrids are being evaluated for ergot reaction and agronomic traits. The finding that ergot resistance is also based on a factor or factors other than protogyny length and pollination (although only from a single set of A, B, and R lines and their F_1 hybrids), opens up new areas of research on genetics and mechanisms of resistance.

LITERATURE CITED

1. Chahal, S. S., Gill, K. S., Phul, P. S., and Singh, N. B. 1981. Effectiveness of recurrent selection for generating ergot resistance in pearl millet. Soc. Adv. Breed. Res. Asia Oceania 13:184-186.
2. Rai, K. N., Andrews, D. J., and Rao, A. S. 1986. Hybrid potential of 81A—A dwarf male-sterile line of pearl millet. Indian J. Genet. Plant Breed. 46:295-303.
3. Thakur, R. P., and King, S. B. 1988. Ergot disease of pearl millet. Int. Crops Res. Inst. Semi-Arid Trop. (ICRISAT) Inf. Bull. 24. 24 pp.
4. Thakur, R. P., Rao, V. P., and King, S. B. 1989. Ergot susceptibility in relation to cytoplasmic male sterility in pearl millet. Plant Dis. 73:676-678.
5. Thakur, R. P., Rao, V. P., and King, S. B. Factors associated with ergot resistance in pearl millet. Indian Phytopathol. In press.
6. Thakur, R. P., Talukdar, B. S., and Rao, V. P. 1983. Genetics of ergot resistance in pearl millet (Abstr.) Page 737 in: Int. Congr. Genet., 15th Part 2. Oxford and IBH Publishing Company New Delhi.
7. Thakur, R. P., and Williams, R. J. 1980. Pollination effects on pearl millet ergot. Phytopath

ology 70:80-84.
8. Thakur, R. P., Williams, R. J., and Rao, V. P. 1982. Development of ergot resistance in pearl millet. Phytopathology 72:406-408.
9. Thakur, R. P., Williams, R. J., and Rao, V. P. 1983. Control of ergot in pearl millet through pollen management. Ann. Appl. Biol. 103:31-36.
10. Willingale, J., Mantle, P. G., and Thakur, R. P. 1986. Postpollination stigmatic constriction, the basis of ergot resistance in selected lines of pearl millet. Phytopathology 76:536-539.