Identification of Stable Resistance to Ergot in Pearl Millet

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ABSTRACT

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More than 2,500 germ plasm accessions and about 7,300 advanced breeding lines were screened to identify resistance to ergot in pearl millet. All advanced breeding lines were highly susceptible and only very low levels of resistance were detected in a few germ plasm accessions from India, Nigeria, and Uganda. Ergot-resistant lines, developed by intermating relatively less-susceptible plants and pedigree selecting for several generations under high disease pressure, were evaluated through a cooperative international multilocational testing program, the International Pearl Millet Ergot Nursery (IPMEN), to determine their resistance stability. Seven inbred lines (ICMPE numbers) and six sib-bulk populations (ICMPES numbers) evaluated at eight to 12 locations for 2–3 yr in India and West Africa showed consistently high levels of ergot resistance. Mean ergot severities in these lines across locations ranged from <1 to 7% compared with 30–65% in the susceptible check. These lines were also resistant to smut and downy mildew at ICRISAT Center, Patancheru.

Ergot, caused by *Claviceps fusiformis* Loveless, is a serious, widespread disease of pearl millet (*Pennisetum americanum* (L.) Leeke). The disease is particularly serious in commercial F_1 hybrids in India, because several characteristics including highly synchronous tillering and flowering and genetic homogeneity make the hybrids more vulnerable than open-pollinated varieties (14). Ergot is a serious health hazard to humans and animals because of the neurotoxic alkaloids contained in the sclerotia of the causal fungus (1,5–8). Downy mildew (*Sclerospora graminicola*) and smut (*Tolyposporium penicillariae*) are also important diseases that cause substantial reductions in grain yield of pearl millet in the semiarid tropics (9,17).

Pearl millet is grown largely by resource-poor farmers in the subsistence agriculture of the semiarid tropics, and ergot is a soilborne and airborne disease (11). Therefore, methods of disease control other than host-plant resistance are not likely to be technically and economically viable means of control. Thus stable (time and environments) sources of resistance must be identified and used in breeding disease-resistant hybrids and varieties. During the past 7 yr at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, a large number of germ plasm accessions and breeding lines have been screened, and from several relatively less susceptible plants selected from a few germ plasm accessions, lines with high levels of ergot resistance have been developed. The stability of resistance of these lines was determined through a cooperative multilocational testing program, the International Pearl Millet Ergot Nursery (IPMEN) in India and countries in West Africa. In this paper, we describe the progress made in developing and identifying stable ergot resistance in pearl millet and discuss its possible use in resistance breeding programs. Part of this work has been published as an abstract (10).

MATERIALS AND METHODS

Screening method. The standard ergot resistance screening method involving bagging the inflorescences at the boot-

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leaf stage, inoculating them at full protogyny (maximum fresh stigma emergence), rebagging, sprinkler irrigating to increase relative humidity, and scoring for ergot severity 15–20 days after inoculation (15) was used to screen germ plasm accessions and breeding lines. Screening was done during the rainy and postrainy seasons at Patancheru.

Screening germ plasm accessions. More than 2,500 germ plasm accessions from nine African and two Asian countries were screened between 1977 and 1983 (Table 1). In the initial screening, 10 randomly selected plants (inflorescences) were inoculated in a single-row plot 4 m long. Plants that developed relatively lower percentages of ergot-infected florets (up to 10%) and produced good seed (>80% selfed) were selected for further evaluation. In the advanced screenings, head-to-row progenies were grown and 20–40 plants were screened in a two-row plot 4 m long. Plants that showed increased levels of ergot resistance (≤5% florets infected per inflorescence) in advanced screening 1 were selected either for evaluation at advanced screening 2 or for use in crosses to develop lines with higher levels of ergot resistance.

Screening breeding lines. More than 7,300 breeding lines from the All India Coordinated Millet Improvement Project (AICMIP) and ICRISAT were screened between 1976 and 1983. These included hybrids, population progenies, synthetics, male steriles, and inbreds. Ten to 20 plants were screened in each entry.

Development of lines with increased ergot resistance. Individual plants that showed relatively less ergot susceptibility (up to 10% severity) during initial and advanced screenings were intermated to accumulate resistance genes in their progenies. By pedigree selection, ergot-resistant plants were selected at each generation (up to F_8) under high disease pressure (15). Lines that consistently showed high ergot resistance (≤5% severity) in testing at Patancheru were tested multilocationally in the IPMEN to determine their resistance stability. To further improve the levels of resistance and widen the genetic base of resistance, ergot-resistant F_5–F_8 lines from diverse crosses were intermated and resistance selection was repeated for several generations to obtain lines with uniformly high ergot resistance. Four to 10 phenotypically uniform ergot-resistant sister lines (designated by ICMPE numbers) were sib-mated (pollen collected from 10–20 plants from each line, bulked, and used to pollinate plants of the sister lines in the same population) to constitute sib-bulks (designated by ICMPES numbers).

Evaluation of stability of resistance. Stability of resistance was determined through a cooperative international multilocational program, the IPMEN.

Test locations. The locations selected were mainly those where ergot occurs every year. The first IPMEN in 1977 was tested at 12 locations in four countries: India, Nigeria, Burkina Faso (Upper Volta), and Senegal. The number of locations varied from eight to 13 in different years with the maximum number always being in India. The locations are in the major pearl millet-growing regions of the semiarid tropics varying from latitudes of 11°11′ N (Samaru, Nigeria) to 30°56′N (Ludhiana, India). Mean temperatures during flowering at these locations varied from 17 to 33 C. Because of limited resources and reliance on favorable weather conditions during the crop season at most locations, useful data were not received from all locations every year.

Test entries. Each year, an IPMEN set included 21–32 test entries and a susceptible check, and since 1979, a local resistant and a local susceptible check were also used at each location. Until 1980, entries in the IPMEN were mainly relatively less susceptible selections from germ plasm and breeding lines. ICMPE lines have been included in the IPMEN since 1981, and ICMPES populations, since 1982. Seed for individual entries for each year were drawn from original stocks to eliminate variation in ergot reaction resulting from different seed stocks.

Nursery management. The nursery was coordinated from the ICRISAT Center. Each year, seed and a nursery book with instructions and data record sheets were despatched to IPMEN cooperators in May for planting in the rainy season, which normally begins in June or July. Each entry was planted in two 4-m row plots with two replicates. Interrow and interplant spacings varied from 75 × 20 cm to 45 × 10 cm at different locations. Single inflorescences from 10 plants per row were evaluated. At Patancheru only, sprinkler irrigation was provided for 30 min twice each day (noon and evening) on rainfree days from inoculation to disease evaluation. Inoculated plants were scored using the standard ergot severity assessment key (14). Data received from cooperators were analyzed for each location to determine the mean and range of ergot severity for individual entries, and a detailed report was prepared for each year's IPMEN.

Screening for multiple disease resistance. During the 1981–1983 crop seasons, IPMEN entries were planted in two 4-m row plots with two replicates in the Patancheru downy mildew nursery (18). Rows were spaced at 75 cm and plants at 10–20 cm within a row, which provided 40–80 plants per plot. Counts were taken of total and infected plants 30 and 45 days after emergence to determine percent downy mildew incidence. Two tillers from 20 downy mildew-free plants per entry were inoculated, one with *C. fusiformis* (15) and one with *T. penicillariae* (12), to assess levels of resistance to ergot and smut, respectively.

RESULTS

Resistance in germ plasm accessions. In the initial screening, many lines were identified (except from Cameroon and Lebanon) that had relatively less susceptible plants (up to 10% ergot severity), but in the advanced screening, only 12 lines originating from India, 8 from Nigeria, and 3 from Uganda provided ergot-resistant plants (≤5% severity) (Table 1).

Resistance in breeding lines. Of 7,318 breeding lines screened, more than 90% showed high susceptibility (>30% ergot severity) in the initial screening. A few lines that showed less severity in the initial screening developed more ergot (>30% severity) in subsequent advanced screenings.

Stability of resistance. During 1977–1980, the IPMEN entries, which included less susceptible selections from germ plasm/breeding lines, showed variable reactions (6–22% ergot severity) across locations over years. No entry had an across-location mean severity of less than 10% in 1977. Only two of 26 entries each in 1978 and 1979 and five of 32

Table 1. Summary of pearl millet germ plasm accessions screened for ergot resistance at Patancheru during 1977–1983

Country of origin	Initial screening (10 plants/line)		Advanced screening (20–40 plants/line)	
	Lines screened	Lines with less susceptible plants (≤10% severity)	Lines screened	Lines with resistant plants (≤5% severity)
Cameroon	45	0	0	0
Ghana	123	16 (26)[a]	26	0
Mali	93	16 (20)	20	0
Niger	398	8 (43)	43	0
Nigeria	446	14 (208)	208	8 (32)
Senegal	123	2 (3)	3	0
Tanzania	129	18 (26)	26	0
Togo	178	36 (127)	127	0
Uganda	38	3 (40)	40	3 (15)
India	937	38 (144)	144	12 (34)
Lebanon	14	0	...	...

[a] In parentheses is number of plants selected.

entries in 1980 showed an across-location mean severity of less than 10%. With the inclusion of ergot-resistant ICMPE and ICMPES lines in the IPMEN from 1981, a large proportion of entries showed across-location mean ergot severity of less than 10%. Ergot pressure, as indicated by across-location mean severity on the susceptible check, varied from 30% in 1979 to 65% in 1982 and 1983.

Our attempt to select individual plants for increased levels of resistance from within a line did not prove effective. Four lines that were selected and evaluated for four to seven generations did not show increased levels of resistance. However, it became possible to extract ergot-resistant lines in the progenies from crosses between less susceptible plants from several such lines. Seven ICMPE lines in 30 tests over 3 yr had mean ergot severities in the range of <1–7% and the six ICMPES lines in 20 tests over 2 yr between <1 and 3% compared with 30–65% in the susceptible check (Table 2).

Mean ergot severities of entries at seven locations for 2–3 yr are presented in Table 3. Disease pressure (severity in the susceptible check) varied at and across locations over years. Patancheru provided the maximum ergot pressure (90–99% severity) followed by Aurangabad, Samaru, Ludhiana, Jamnagar, New Delhi, and Mysore (33–62% severity). Despite variations in ergot pressures, all 13 entries showed consistently high ergot resistance at and across locations over years.

Multiple disease resistance. Thirteen ergot-resistant lines also were resistant to downy mildew in four tests and to smut in three tests at Patancheru (Table 4).

DISCUSSION

The lack of adequate levels of ergot resistance in the large, varied germ plasm accessions and breeding lines of pearl millet that we have screened so far probably indicates the absence of major genes for ergot resistance in this crop. Recent studies on inheritance of ergot resistance have indicated that resistance is recessive and polygenically controlled (13). Our success in building higher levels of resistance to ergot in the progenies of crosses between relatively less susceptible plants through a process of resistance accumulation (gene-pyramiding) also supports the polygenic nature of ergot resistance.

The absence of undetectable major genes for resistance to ergot in pearl millet can be explained by examining some of the epidemiological aspects of the disease. The pearl millet-growing

Table 2. Mean ergot severity (%)[a] of pearl millet genotypes across locations in multilocational testing during 1977–1983

	Year (no. of locations)								
Genotype[b]	1977 (12)	1978 (13)	1979 (11)	1980 (9)	1981 (10)	1982 (8)	1983 (12)	Mean	Range
J 797	20	14	16	16	–[c]	–	–	16.5	14–20
SC-2(M)5-4-E-8	18	15	9	7	11	14	–	12.3	7–18
J 2238-2-E-4-1	14	16	13	14	15	22	14	15.4	13–22
700708-1-E-1	–	11	15	22	22	13	6	14.8	6–22
ICMPE 134-6-9	–	–	–	–	2	<1	<1	1.0	<1–2
ICMPE 134-6-11	–	–	–	–	2	1	<1	1.0	1–2
ICMPE 134-6-41	–	–	–	–	2	1	<1	1.0	1–2
ICMPE 134-6-34	–	–	–	–	2	1	1	1.3	1–2
ICMPE 134-6-25	–	–	–	–	2	1	1	1.3	1–2
ICMPE 13-6-27	–	–	–	–	6	2	1	3.0	1–6
ICMPE 13-6-30	–	–	–	–	7	2	2	3.7	2–7
ICMPES 1	–	–	–	–	–	1	1	1.0	1–1
ICMPES 2	–	–	–	–	–	1	<1	1.0	<1–1
ICMPES 23	–	–	–	–	–	2	1	1.0	1–2
ICMPES 27	–	–	–	–	–	1	<1	1.0	<1–1
ICMPES 28	–	–	–	–	–	3	1	2.0	1–3
ICMPES 32	–	–	–	–	–	2	3	2.5	2–3
Susceptible check	42	42	30	35	44	65	65	46.0	30–65

[a] Based on 20–40 inoculated inflorescences in two replicates at each location.
[b] Origin of genotypes: SC = Serere Composite from Uganda; J = Jamnagar, India; 700 = breeding line from Kano, Nigeria; ICMPE = ICRISAT Millet Pathology ergot-resistant lines; ICMPES = ICMPE sib-bulks.
[c] Entries not tested.

Table 3. Performance of the 13 IPMEN entries in 1981, 1982, and 1983 at Samaru, Nigeria, and at six Indian locations

	Ergot severity (%)[b]																	
	Samaru		Aurangabad		Jamnagar			Patancheru			Ludhiana			New Delhi			Mysore	
Entry	1982	1983	1982	1983	1981	1982	1983	1981	1982	1983	1981	1982	1983	1981	1982	1983	1982	1983
ICMPE[a] 134-6-9	<1	<1	1	<1	<1	<1	0	<1	<1	<1	5	<1	1	1	2	0	<1	<1
ICMPE 134-6-11	<1	1	5	<1	<1	<1	<1	<1	1	<1	2	<1	2	1	2	0	<1	0
ICMPE 134-6-41	<1	1	5	<1	<1	<1	<1	<1	1	<1	1	<1	1	4	<1	<1	<1	0
ICMPE 134-6-34	<1	1	2	1	1	<1	<1	<1	1	<1	1	<1	5	1	2	<1	<1	0
ICMPE 134-6-25	1	<1	1	1	<1	0	<1	<1	<1	<1	3	<1	3	1	1	<1	2	0
ICMPE 13-6-27	6	<1	1	2	7	1	1	5	1	1	5	2	3	5	<1	1	3	0
ICMPE 13-6-30	1	1	1	1	4	1	<1	3	2	2	7	<1	8	4	4	2	1	1
ICMPES 1	1	0	1	1	–[c]	1	2	–	2	1	–	1	3	–	1	1	1	0
ICMPES 2	<1	1	2	1	–	0	<1	–	<1	<1	–	<1	2	–	3	0	<1	<1
ICMPES 23	0	1	2	1	–	0	1	–	3	2	–	5	2	–	2	<1	1	0
ICMPES 27	0	<1	1	1	–	0	<1	–	1	<1	–	<1	2	–	1	<1	3	0
ICMPES 28	<1	<1	3	2	–	<1	1	–	7	1	–	<1	2	–	3	2	1	0
ICMPES 32	1	1	4	16	–	<1	2	–	2	1	–	1	2	–	<1	1	1	1
Susceptible check	83	89	97	91	41	58	44	99	98	90	67	65	66	63	23	52	33	62

[a] ICMPE(S) = ICRISAT Millet Pathology ergot-resistant (sib-bulk) lines.
[b] Based on 20–40 inoculated inflorescences per entry in two replicates.
[c] Entry not included.

Table 4. Ergot, smut, and downy mildew (DM) reactions of ergot-resistant pearl millet lines during the rainy (R) and summer (S) seasons at Patancheru

Lines[a]	Ergot severity (%)[b]				Smut severity (%)[b]			DM incidence (%)[c]			
	1981	1982		1983	1981	1982	1983	1981	1982		1983
	R	R	S	R	R	R	R	R	S	R	R
ICMPE 134-6-9	<1	1	0	<1	0	0	0	0	1	0	0
ICMPE 134-6-11	<1	0	<1	<1	0	0	0	0	0	5	9
ICMPE 134-6-41	<1	<1	<1	<1	0	0	1	0	1	0	0
ICMPE 134-6-34	3	0	1	<1	0	0	0	4	1	0	3
ICMPE 134-6-25	<1	0	<1	<1	0	0	0	0	0	0	0
ICMPE 13-6-27	5	0	0	1	0	0	0	0	0	0	0
ICMPE 13-6-30	<1	–[d]	2	2	–	0	0	0	–	0	0
ICMPES 1	–	–	2	1	–	0	0	–	–	2	4
ICMPES 2	–	–	<1	<1	–	0	0	–	–	3	2
ICMPES 23	–	–	3	2	–	0	0	–	–	0	1
ICMPES 27	–	–	1	<1	–	0	0	–	–	6	0
ICMPES 28	–	–	7	1	–	0	0	–	–	0	1
ICMPES 32	–	–	2	1	–	0	0	–	–	0	4
BJ 104 (check)	83	67	94	91	54	48	82	32	5	51	34

[a] ICMPE(S) = ICRISAT Millet Pathology ergot-resistant (sib-bulk) lines.
[b] Mean of 20–40 inoculated inflorescenes in two replicates, both ergot and smut inoculations, made on two tillers of the same plant free of downy mildew.
[c] Mean of two replicates in the ICRISAT center downy mildew nursery.
[d] Entry not included/data not available.

environment, which is generally characterized by low and erratic rainfall and high temperature, and ergot infection and development, which is favored by relatively high humidity and low temperature (20–25 C), are mutually contrasting. Ergot becomes more severe when the crop flowers during rainy days. In addition, the complex nature of the disease, including the short period of susceptibility (2–3 days of fresh stigma stage), the restricted infection site, and the strong negative interaction between pollination and ergot infection (14), preclude a strong natural selection either for resistance in the host or for virulence in the pathogen. This supports the host-pathogen coevolution hypothesis that the extreme susceptibility and virulence of epidemic situations are not the norm over a long-term evolution (3). It would appear that for a long time, the evolution of pearl millet-*C. fusiformis* system has been in a state of epidemiological equilibrium within the local land races of pearl millet composed of heterogeneous plant populations. Only recently have genetically near-homogeneous populations, such as F_1 hybrids in India, been grown on a commercial scale, and the disease, once considered to be of minor importance, has become an important production constraint in the hybrid crop.

In the strategy of controlling a disease through host-plant resistance, identification of stable resistance sources is the first step. Several of our ICMPE and ICMPES lines have shown stability of resistance across diverse locations in India and West Africa for 2–3 yr, although the time scale and number of test environments have been limited, particularly in Africa.

Since resistance to ergot is recessive and polygenically controlled, transferring this resistance would be more difficult and time consuming than if it were dominant and single-gene controlled. Polygenic resistance, however, may have its own merit in terms of conferring stability of resistance. It is generally believed (with exceptions, of course) that resistance governed by polygenes is more stable and durable (time and area) than resistance governed by major genes (2,4,16).

Since only a limited number of germ plasm accessions (2,500) of the total world collections of about 17,000 from 30 countries (ICRISAT Genetic Resource Unit, *personal communication*) have been screened, there are possibilities of identifying sources with major resistance genes.

It is encouraging to note that 13 of the ergot-resistant lines also showed resistance to smut and downy mildew at Patancheru, but wider testing is needed in India and Africa to confirm the stability of multiple resistance in these lines.

In the ICRISAT Center pearl millet improvement program, ergot resistance is being incorporated into hybrid seed parents and pollinator parents using a back-cross program and in population improvement breeding through recurrent selection. We will make available on request to any scientist information on lines and small quantities of seed of ergot-resistant lines described in this paper.

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