Downy Mildew Reactions of Pearl Millet Lines With and Without Cytoplasmic Male Sterility

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ABSTRACT

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A total of 2,976 breeding progenies of pearl millet (*Pennisetum americanum*) from four different sources possessing male-sterile and normal cytoplasm of the A_1 sterile system were evaluated for susceptibility to downy mildew (caused by *Sclerospora graminicola*) employing an effective field-screening technique. Lines carrying male-sterile cytoplasm were found to be not more susceptible than those with normal cytoplasm, indicating that in pearl millet the A_1 male-sterile cytoplasm is not involved in determining susceptibility to downy mildew.

Pearl millet (*Pennisetum americanum*) is an important food crop in the drier regions of many African countries and India, with an annual production of about 13 million metric tons from an estimated 26 million hectares (11). Cytoplasmic male sterility, first observed in 1955 at Tifton, GA (1), and later in India (4), made the commercial production of hybrids in this crop possible. Tift 23A, one of the two sources of male sterility from Tifton (2,3) carrying the A_1 class of male sterility, proved to be the most successful seed parent in developing single-cross grain hybrids in India; it showed 75–100% increases in yield over local varieties (5). Of the five hybrids released from 1965 to 1972, each of which had the same seed parent, the third—23A × J104 (designated HB3 and released in 1968)—rapidly gained popularity. By the early 1970s, hybrids dominated by HB3 were grown on 15% of the 11 million hectares of pearl millet in India (7). However, cultivation of more than 1.6 million hectares of the same homogenous cultivar produced a situation of genetic vulnerability. In 1971, India's pearl millet production suffered an epidemic of downy mildew incited by *Sclerospora graminicola* (Sacc.) Schroet. Combined with drought, this epidemic reduced pearl millet grain production close to 3 million metric tons (9).

The epidemic of downy mildew in India occurred at the same time as the southern leaf blight (*Helminthosporium maydis*) epidemic in the United States (10), where it was established that cytoplasm, in addition to nuclear genes, can be significant for the expression of a specific pathogen (6). It was suggested that susceptibility to downy mildew in pearl millet could be caused by the single source of male-sterile cytoplasm used in all the hybrids (8,9). Evidence is provided in this report to show that in pearl millet

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the widely used A_1 class of male-sterile cytoplasm is not involved in determining susceptibility to downy mildew.

MATERIALS AND METHODS

Downy mildew incidence was evaluated on male-sterile lines (A lines) and the corresponding maintainer lines (B lines), which were near isogenic except for the presence of sterile and normal cytoplasm. Two groups of backcross progenies were used from breeding projects where new seed parents resistant to downy mildew were being developed.

The first group consisted of male-sterile and maintainer progenies derived from Tift $23d_2B$ (isogenic to Tift 23A except for the d_2 dwarfing gene and with the same sterile cytoplasm in the A line) after mutagen treatment. In 1975, dry seed of Tift $23d_2B$, which is highly susceptible to downy mildew in India, was treated with an acute dose of 30 kR gamma rays from a ^{60}Co gamma cell with a view to inducing downy mildew resistance. Three-hundred M_1 generation seeds were planted at ICRISAT Center and all surviving plants selfed. These were sown as head-rows in the January–April off-season in 1976 in the downy mildew screening nursery developed at ICRISAT (12). Plants free of downy mildew in these rows were both selfed and test crossed onto Tift $23d_2A$. This process of backcrossing and selection only of maintainer plants free of downy mildew was repeated for five further generations, two generations per year both grown in the downy mildew nursery, up to the sixth backcross.

The second group of material consisted of three sources of maintainer and male-sterile progenies in A_1 sterile cytoplasm, each at the fourth backcross stage. The sources were Casady Dwarf 5 (CD 5) and Casady Dwarf 67-1 (CD 67-1) (from Uganda and incorporating a U.S. dwarf source) and a semitall segregant—3/4 Heine Kheri-207 (HK 207)—from the dwarf population 3/4 Heine Kheri from Niger.

Both groups of material were grown in a downy mildew screening nursery, the establishment and operational details of which were described recently (12). Essentially, the nursery consists of inoculated infector rows of NHB-3 (the hybrid that became highly susceptible to downy mildew in 1971) planted in every fifth row 3 wk prior to planting the test rows (entries to be evaluated for disease incidence) in the intervening rows. Indicator rows were planted with NHB-3 among the test rows. The plot size for each progeny and indicator row was a single 4-m row (with rows 0.75 m apart); within-row spacing was 10-15 cm between plants. Incidence of downy mildew was recorded as percentage of plants infected at 45 days after planting, which included a score at 30 days to detect any seedlings killed by the disease that might subsequently disintegrate and be lost. The progenies derived from Tift $23d_2A$ and B plant × plant crosses were evaluated in the rainy season of 1979. Progenies of the second group were tested in the following off-season of 1980. A chi-square test using actual numbers of progenies in each class interval was used to compare the relative distributions of downy mildew incidence between the A and B progenies within each source.

Table 1. Downy mildew incidence on sterile and fertile sixth-backcross progenies derived from mutagen-treated Tift $23d_2B$

Incidence class (%)	Relative frequency (%) A lines (sterile cytoplasm)	B lines (normal cytoplasm)	Indicator[a]
0–10	61.5	62.8	0
11–20	27.6	28.6	0
21–30	7.3	5.3	4.5
31–40	2.4	1.3	4.5
41–50	0.5	1.0	0
51–60	0.3	0	9.1
61–70	0.3	1.0	13.6
71–80	0.1	0	27.3
81–90	0	0	18.2
91–100	0	0	22.7
Chi-square value	3.76[b]		
Probability range	0.75–0.50		
Number of progenies	1,142	301	22

[a] Susceptible hybrid NHB3.
[b] Numbers of progenies in the 41–60% incidence range were combined, as were those in the 61–80% range.

Table 2. Downy mildew incidence on sterile and fertile fourth-backcross progenies of three potential cytoplasmic male steriles

Incidence class (%)	Relative frequency (%) Casady A[a]	Casady B[b]	Dwarf-5 A	Dwarf-67-1 B	3/4 Heine Kheri-207 A	3/4 Heine Kheri-207 B	Indicator[c]
0–10	22.0	22.7	90.7	91.0	87.1	57.1	2.6
11–20	11.9	9.3	2.7	5.4	12.9	33.3	5.1
21–30	7.3	6.1	5.3	1.8	0	9.5	2.6
31–40	7.9	5.0	1.3	1.8	0	0	7.7
41–50	7.7	6.8	0	0	0	0	15.4
51–60	6.2	4.4	0	0	0	0	5.1
61–70	8.5	8.8	0	0	0	0	5.1
71–80	8.9	9.9	0	0	0	0	5.1
81–90	7.5	12.2	0	0	0	0	15.4
91–100	12.1	14.8	0	0	0	0	35.9
Chi-square value	18.77[d]		1.83		5.91[e]		
Probability range	0.05–0.025		0.75–0.50		0.025–0.010		
Number of progenies	826	525	75	55	31	21	39

[a] Sterile cytoplasm.
[b] Normal cytoplasm.
[c] Susceptible hybrid NHB-3.
[d] Chi-square value for 0–80% incidence classes = 7.44, $P = 0.5–0.25$.
[e] Numbers of progenies in 11–20% and 21–30% incidence classes were combined.

RESULTS

The incidence in the 22 indicator rows grown among the test progenies averaged 74% (sum of incidence class × frequency), but the majority of the A and B progenies selected from the $23d_2B$ mutagen-treated seed source recorded less than 10% downy mildew incidence (Table 1). A chi-square test ($\chi^2_5 = 3.76$, $P = 0.75–0.50$) indicated that the disease incidence frequency distributions of the A and B lines were not different.

In the second group tested, different levels of disease incidence were recorded between sources (Table 2). Sixty percent of the indicator rows showed 61% or more downy mildew incidence. In the CD 67-1 and HK 207 sources, a high proportion of the progenies recorded less than 10% incidence; in CD 5, however, a substantial number of the progenies ranged from 81 to 100% incidence. When all the CD 5 progenies were compared, the chi-square test indicated that the incidence distributions of the A and B lines were probably different.

Of more practical significance is the comparison of incidence among the more resistant progenies, as with the other sources used. When the comparison was made omitting the progenies in the two highest incidence classes (81–100%), the chi-square test showed that the incidence

distributions in the remaining CD 5 A and B progenies are unlikely to be different. The chi-square test indicated that a difference may exist between the A and B disease incidence frequencies in the HK 207 source progenies, with higher levels of resistance in the A-lines. However, the number of progeny in this source is relatively small, and most are below the level of 20% incidence. In the CD 67-1 source, the chi-square test gave no evidence to indicate a difference in incidence between the A and B progenies.

DISCUSSION

The high incidence levels of downy mildew recorded on the indicator rows showed that adequate disease pressure was exerted on the material under test in both seasons. The comparison of distribution of disease incidence between the A and the B progenies in three of the four sources studied (using 0–80% incidence data from CD 5) provided no evidence to show that disease levels on male-sterile and normal cytoplasm lines were different. In CD 5, the differences between incidence on A and B progenies in the 81–90% and 91–100% frequency classes were relatively small and in the direction of more resistance in A line progenies. The evidence from the HK 207 source is of less consequence than from the other sources because of the smaller number of progeny involved, particularly in the B lines; here again, however, the difference was in the direction of less susceptibility in lines with male-sterile cytoplasm.

These results indicate that sterile cytoplasm of the A_1 class in pearl millet did not confer higher susceptibility to downy mildew, and it is thus likely that nuclear genes alone were responsible for the observed resistance or susceptibility. Support for this conclusion may also be inferred from the improved levels of downy mildew resistance exhibited by more recent hybrids now being grown in India that still utilize seed parents carrying A_1 sterile cytoplasm. Although on general principles efforts should be made to exploit other classes of sterile cytoplasm, there is currently no evidence to suggest that the A_1 class of sterile cytoplasm should not continue to be used in pearl millet to make new hybrid combinations, provided that adequate genetic resistance is incorporated.

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