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Influence of A_1 cytoplasmic substitution on the downy-mildew incidence of pearl millet

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Abstract. Large-scale cultivation of pearl millet [*Pennisetum glaucum* (L.) R. Br.) F_1 hybrids in India has led to increased incidence of downy-mildew (*Sclerospora graminicola*). There is concern that the A_1 male-sterile cytoplasm used in all the hybrids released so far is responsible for this increase. The influence of A_1 male-sterile cytoplasm on downy-mildew incidence in pearl millet was studied by comparing the disease reaction of 40 pairs of F_1 hybrids, each pair carrying respectively A_1 male-sterile and normal B cytoplasm. Mean downy-mildew incidence was similar in the hybrids carrying either A_1 male-sterile or B cytoplasm. The general combining ability of lines with and without A_1 cytoplasm was found to be similar for downy-mildew incidence. These results indicated that in pearl millet A_1 cytoplasm is not associated with increased downy-mildew incidence. The possible danger of using only one source of cytoplasm has been briefly discussed.

Key words: *Pennisetum glaucum*, Pearl millet – Male sterility – Disease resistance – A_1 cytoplasm – Downy mildew – *Sclerospora graminicola*

Introduction

The commercial production of F_1 hybrids of pearl millet [*Pennisetum glaucum* (L.) R. Br.] in India became possible only after the discovery of cytoplasmic male sterility (Burton 1958) and subsequent release of the male-sterile line Tift 23A carrying the A_1 cytoplasm for male sterility (Burton 1965). As a result of this a quantum jump in pearl millet productivity was realized (Burton and Powell 1968). Since then all the male-sterile lines which have been used in the hybrids released so far for general cultivation in India are based on the A_1 cytoplasm of Tift 23A (Dave 1987), although other sources of cytoplasm, such as A_2, A_3 (Burton and Athwal 1967) and A_4 (Hanna 1989), are available.

Large-scale cultivation of hybrids has resulted in an increased incidence of downy-mildew [*Sclerospora graminicola* (Sacc.) Schroet]. Despite the failure to compare the disease incidence of hybrids with and without A_1 cytoplasm, it was argued (Safeeulla 1977) that the A_1 source of male-sterile cytoplasm used in all the hybrids might be responsible for the increased incidence of downy mildew in them. The influence of A_1 cytoplasm on the downy-mildew incidence of hybrids can be studied by comparing the disease reaction of F_1 hybrid pairs which are identical in their genetic constitution but carry either A_1 or normal cytoplasm. The present investigation was, therefore, undertaken to determine whether the increased downy-mildew incidence of hybrids is indeed due to the A_1 cytoplasm used in all commercial hybrids.

Materials and methods

Development of experimental hybrids

Forty pairs of experimental hybrids for the present investigation were developed by crossing six pollinators (P), which included highly downy-mildew susceptible (J 104 and 7042 S) and resistant (P 310, P 1449, ICMP 85410 and ICMP 423) lines, with nine male-sterile (A) and their corresponding maintainer (B) lines (44 A/B, 47 A/B, 843 A/B, 81 A/B, 841 A/B, 863 A/B, 88004 A/B, 88006 A/B and 89111 A/B). Among these A/B pairs the first three pairs were susceptible and the remaining six were resistant to downy mildew. Both A and B lines of each pair were used as

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female parents in crosses with P lines to produce F_1 hybrids. B × P hybrids were obtained taking advantage of the protogynous nature of flowering in pearl millet (Burton 1980). Pollen from P lines was placed on B lines at full-stigma emergence.

The A and B lines of pearl millet are genetically indentical. The only difference lies in the fact that the former has male-sterile cytoplasm while the later consists of normal cytoplasm. Since the cytoplasm is contributed by the female parent, hybrids with A lines inherited A_1 cytoplasm and those with B lines had normal cytoplasm. The genic constitution of the two groups of hybrids is identical.

Inoculum, inoculation and disease screening

Fresh sporangia (asexual spores of *S. graminicola*) produced on 7042 S were used in inoculation. The concenration of inoculum was kept at 10^5 sporangia per ml.

The seedling inoculation technique (Singh and Gopinath 1985) was used to test the downy-mildew incidence of entries. This stringent greenhouse technique avoids disease escapes in the test material which is common in field screening (Singh and Gopinath 1985). Each entry was evaluated in two replications at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, India. A highly-susceptible control, viz. NHB-3, was also grown after every ten hybrids.

The data on downy-mildew incidence (%) were subjected to angular transformation for an analysis of variance. The influence of A_1 cytoplasm was judged by comparing the downy-mildew incidence of an A × P hybrid with that of a B × P counterpart hybrid. The mean downy-mildew incidence of hybrids carrying male-sterile cytoplasm was compared to that of hybrids having normal cytoplasm by using Students't test.

Results and discussion

The susceptible check, NHB-3, showed more than 95% disease incidence. Some test entries also showed 100% downy-mildew infection. This indicated that there was a good inoculum load during screening.

There was a wide range for downy-mildew incidence in hybrids carrying both sterile (0–100%) and normal cytoplasm (0–90%) (Table 1). Downy-mildew incidence was similar in all but five pairs (Table 1). Among these five pairs two B × P and three A × P hybrids showed a significantly higher incidence of downy mildew than their counterpart hybrid combinations. The mean downy-mildew incidence of hybrids carrying sterile cytoplasm (26%) and that of hybrids with normal cytoplasm (25%) was similar. This indicated that in pearl millet A_1 male-sterile cytoplasm is not associated with a higher downy-mildew incidence. The availability of downy-mildew-resistant lines based on Tift $23A_1$ cytoplasm (Rai and Rao 1990) further substantiates this finding. Anand Kumar et al. (1983) also did not observe any significant difference in the disease reaction of breeding progenies possessing male-sterile and normal cytoplasm of the A_1 sterile system. Similarly, substitution of normal cytoplasm by A_1 cytoplasm has been reported not to confer additional susceptibility to pyricularia leaf spot (Wilson and Hanna 1992) and smut (Yadav et al. 1992) of pearl millet. However, susceptibility differences according to cytoplasm have been observed in ergot of pearl millet (Thakur et al. 1989), ergot of sorghum (Futrell and Webster 1965), Southern corn blight of maize (Scheifele et al. 1970), and ergot of wheat (Reitz and Lucken 1972).

To examine more critically the influence of A_1 cytoplasm on downy-mildew incidence, we extracted a set of 40 crosses (eight females × five males) from the crosses of Table 1 to form a factorial design. The lines

Table 1. Downy mildew incidence (%) of pearl millet hybrids with A_1 male-sterile (A × P) and normal (B × P) cytoplasm

Hybrid	A × P	B × P
841A/B × J104	22.9(28.6)[a]	15.7(23.3)
× 7042S	20.4(26.9)*	78.9(62.7)
× P310	1.8 (7.7)	8.0(16.4)
× P1449	5.0(12.9)	0.0 (0.0)
× ICMP 423	12.8(21.0)	3.6(10.9)
× ICMP 85410	13.7(21.7)	11.6(19.9)
843A/B × J104	74.6(59.7)	85.6(67.7)
× 7042S	100.0(90.0)	98.0(81.9)
× P310	15.2(23.0)	12.8(21.0)
× P1449	17.8(25.0)	25.1(30.1)
× ICMP 423	61.8(51.8)	62.2(52.1)
× ICMP 85410	29.5(32.9)	29.5(32.5)
81A/B × J104	22.8(28.5)	45.1(42.1)
× 7042S	84.5(66.8)*	36.6(37.3)
× P310	7.6(16.0)	4.3(12.0)
× P1449	2.2 (8.5)	12.3(20.5)
× ICMP 423	19.0(25.8)	16.2(23.7)
× ICMP 85410	7.2(15.6)	10.4(18.8)
88006A/B × J104	45.3(42.3)	31.9(34.4)
× 7042S	81.1(64.2)	90.1(71.7)
× P1449	2.2 (8.5)	3.9(11.4)
× ICMP 423	31.2(34.0)	19.2(26.0)
× ICMP 85410	27.9(31.9)	12.8(21.0)
89111A/B × J104	3.3(10.5)	1.9 (7.9)
× 7042S	21.8(27.8)	4.4(12.1)
× P1449	13.1(21.2)*	0.0 (0.0)
× ICMP 423	12.2(20.4)*	0.0 (0.0)
× ICMP 85410	3.8(11.2)	0.0 (0.0)
44A/B × J104	73.7(59.1)	75.3(60.2)
× 7042S	85.1(67.3)	59.8(50.7)
× P1449	11.0(19.4)	9.0(17.5)
× ICMP 85410	6.7(15.0)	10.2(18.5)
88004A/B × J104	1.8 (7.7)	0.0 (0.0)
× 7042 S	14.6(22.5)	10.4(18.8)
× ICMP 423	3.6(10.9)	0.0 (0.0)
863A/B × J104	0.0 (0.0)	0.0 (0.0)
× 7042S	0.0 (0.0)	0.0 (0.0)
× P1449	1.7 (7.5)	0.0 (0.0)
47A/B × J104	29.9(33.2)	33.3(35.2)
× 7042S	51.8(46.0)*	81.9(64.8)
Mean (A/B × P)	26.0(30.7)	25.0(30.0)

[a] Figures in parentheses are arc sine transformations
* Significant difference at $P = 0.05$ between A × P and B × P hybrids

Table 2. General combining ability (GCA) effects of pearl millet lines with A_1 (A lines) and normal (B lines) cytoplasm for downy-mildew incidence[a]

Pair	A line	B line
841 A/B	−12.39*	−11.25*
843 A/B	17.27*	18.25*
81 A/B	−5.27	−6.13
88006 A/B	1.57	−1.71

[a] The GCA effects of A and B lines of all pairs are statistically similar

* Significant GCA effects at $P = 0.01$

841 A/B, 843 A/B, 81 A/B and 88006 A/B were taken as females and the pollinators J 104, 7042 S, P 1449, ICMP 423 and ICMP 85410 as males. The combining-ability analysis was carried out following Kempthorne (1957). The difference between the general combining ability of A and B lines of a pair, if any, could be attributed to either A_1 cytoplasm or its interaction with nuclear genes since A and B lines of a pair differ only with respect to their cytoplasm. The difference between the combining ability of A and B lines of all the pairs was non-significant (Table 2). Both A and B lines of the 841 A/B pair were good general combiners for downy-mildew resistance whereas those of 843 A/B pair were found to be poor combiners. These results further confirmed that A_1 cytoplasm is not associated with increased downy-mildew susceptibility.

The increased incidence of downy mildew in pearl millet hybrids might be due to their genetic homogeneity because hybrids are essentially a single genotype. Such a narrow genetic base has also resulted into epidemics of corn Southern rust caused by *Puccinia polysora* (Futrell 1975).

The perusal of data in Table 1 showed that the degree of downy-mildew incidence of the crosses was in the order of resistant × resistant < resistant × susceptibile < susceptible × susceptible. This indicated the role of additive gene action in the inheritance of downy mildew. Thus simple selection procedures should prove fruitful for improving disease resistance. A role for additive gene action in the inheritance of downy mildew has previously been observed by other workers (Singh et al. 1978; Basavaraj et al. 1980).

The results of the present study clearly showed that A_1 sterile cytoplasm in pearl millet did not confer additional susceptibility to downy mildew. Thus, there seems no need to be alarmed about the vulnerability of such cytoplasm to downy mildew. However, efforts should be continued to exploit other sources like A_2, A_3 and A_4, because large scale and continuous use of a single source involves the risk of the source becoming vulnerable to existing or unforeseen diseases as has been encountered in Southern leaf blight (*Helminthosporium maydis*) of maize in the USA (Tatum 1971).

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