Effects of Gene and Cytoplasm Substitutions in Pearl Millet on Leaf Blight Epidemics and Infection by *Pyricularia grisea*

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ABSTRACT

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Derivatives of the pearl millet inbred Tift 23 with substitutions for various cytoplasms and alleles conferring morphologic or developmental traits were evaluated for differences in leaf blight epidemics in the field and their reactions to infection by *Pyricularia grisea* in the greenhouse. None of the experiments indicated an effect of the B, A_1, or A_4 cytoplasms; the *tr* allele for the trichomeless character; or the d_2 allele for dwarf stature on leaf blight progress or on infection by *P. grisea*. An apparent increased susceptibility in the field was associated with the e_1 allele for earliness. When disease progress curves were corrected for anthesis date, inbreds with the e_1 allele were more resistant than inbreds without the allele. Disease ratings made early in the season in the 1991 field experiment did not correlate well with disease ratings made later in the season. Leaf blight increased on early cultivars after anthesis. Therefore, leaf blight in the field must be assessed at similar growth stages. When inoculated with *P. grisea*, seedlings of some inbreds with the e_1 allele had smaller lesion dimensions than inbreds without the allele, and no lesions developed on Tift 23DA_1E. Differences between reactions of the inbreds in the field and the greenhouse could be due in part to differences in susceptibility to other pathogens with an undetermined contribution to the leaf blight complex.

Additional keywords: alloplasmic, near-isogenic, *Pennisetum glaucum*, plant maturity.

Pearl millet (*Pennisetum glaucum* (L.) R. Br.) is used as an annual summer forage crop in the southeastern United States and could potentially become an important grain crop as well. Because of its present use, maintaining healthy foliage is important to provide high-quality forage. Considering that rust (caused by *Puccinia substriata* Ell. & Barth. var. *indica* Ramachar & Cumm.) adversely affects the yield and digestibility of pearl millet forage (15), it is likely that other foliar diseases also reduce forage yield or quality.

Several pathogens cause leaf blight of pearl millet (8). Pyricularia leaf spot, caused by *Pyricularia grisea* (Cke.) Sacc., was first identified in the United States in 1968 (13). This pathogen has become an important component of the complex of organisms that cause leaf blight of pearl millet in Georgia. Infections are usually visible throughout the season, and it has been the only leaf-blighting pathogen targeted in efforts for breeding for resistance (7).

One approach used in pearl millet breeding at the USDA-ARS Forage and Turf Research Unit at Tifton, GA, includes backcrossing various genes or cytoplasms into inbreds with good combining ability. Alleles used for improvement of forage or grain millets include the d_2 allele for dwarf stature (2), the e_1 allele for earliness (6), and the *tr* allele conferring a trichomeless character (3). Some of these alleles may affect the response of plants to diseases. The *tr* allele has been associated with increased rust susceptibility in the field (3), yet also confers a degree of resistance to smut, caused by *Moesziomyces penicillariae* (Bref.) Vanky (14). Cytoplasms used include the fertile B cytoplasm (1) and the male-sterile A_1 (1) and A_4 (5) cytoplasms.

Many of the elite pearl millet inbreds developed at Tifton possess one or more of the above-mentioned alleles, and nearly all are in one of the three cytoplasms. Therefore, an evaluation of the effects of these substitutions on disease resistance or susceptibility was considered useful in interpreting disease ratings of germ plasm in the field and greenhouse. Several near-isogenic and alloplasmic derivatives of the inbred Tift 23B are available for experimental purposes. A mutation-breeding program was conducted at Tifton during the 1960s and 1970s. From the original tall, late-maturing, trichomed inbred Tift 23B, plants with the e_1 and *tr* alleles were selected. Tift 23B has been the recurrent parent in seven or more backcrosses to selections with the early, trichomeless, and dwarf genes, and to the sources of the A_1 and A_4 cytoplasms.

The objectives of this study were to evaluate the effects of gene and cytoplasm substitutions on leaf blight development in the field and lesion development on seedlings following inoculation with *P. grisea*.

MATERIALS AND METHODS

1988 field experiment. On 22 June 1988, 18 derived lines of inbred Tift 23 with allelic or cytoplasmic substitutions were planted at the rate of 0.31 kg/ha in the field at the Coastal Plain Experiment Station in a randomized complete block design with six replications. The inbreds (normally designated with the prefix "Tift," but omitted here for brevity) were 23B, 23A_1, 23A_4, 23DB, 23DA_1, 23DA_4, 23BE, 23A_1E, 23A_4E, 23BS, 23A_1S, 23A_4S, 23DBE, 23DA_1E, 23DA_4E, 23DBS, 23DA_1S, and 23DA_4S. The symbols D, E, and S indicate homozygous substitutions for the d_2, e_1, or *tr* allele, respectively. Symbols B, A_1, and A_4 indicate substitutions of genotypes into these cytoplasms. Plots were 4 m long and spaced 1 m apart. Fertilizer (5-10-15 N-P-K) was applied in the row at planting at the rate of 280 kg/ha.

Visual ratings for percent foliage in the plots with leaf blight (chlorosis and necrosis) were taken 9 August, 18 August, 1 September, and 13 September 1988. Ratings of leaf blight rather than pyricularia severities were taken since it it difficult to separate the effects of different leaf-blighting pathogens, particularly after leaves turn necrotic. Area under the disease progress curves

(AUDPCs) were calculated by

$$\text{AUDPC} = \Sigma[Y_{(i+1)} - Y_i]/2 \times [X_{(i+1)} - X_i]$$

where Y_i = percent severity at time X_i. AUDPCs were analyzed by least squares analysis of variance, and sums of squares were partitioned into replication and cultivar effects (9). Differences in AUDPCs due to cytoplasm or gene substitution were examined by nonorthogonal, single-degree-of-freedom linear contrasts (11).

1991 field experiment. The inbreds 23B, $23A_1$, $23A_4$, 23DB, $23DA_1$, $23DA_4$, 23BE, $23A_1E$, $23A_4E$, 23DBE, $23DA_1E$, and $23DA_4E$ were planted 16 May 1991 in a randomized complete block with 10 replications in the field at the Coastal Plain Experiment Station. Plot dimensions, establishment, and maintenance in 1991 were identical to those evaluated in 1988.

Plots were visually rated for percent foliage with leaf blight on 10 June, 19 June, 2 July, 12 July, 23 July, 31 July, and 12 August. Ratings ceased when rust started to increase throughout the test to levels that would confound leaf blight ratings. Date of 50% anthesis was recorded for each plot. After flowering, plant heights were measured.

AUDPCs were calculated as described for the 1988 experiment. To estimate leaf blight severities at similar growth stages, disease severities were transformed to $\ln\{0.01 + [Y/(1 - Y)]\}$, where Y = proportion of disease. A factor of 0.01 was included in the logit transformation to include observed ratings of zero. Transformed severities were regressed against time (days after planting) by quadratic regression. Transformed blight severities were predicted from the regression equations for the observed heading date. Predicted values were back-transformed to percent severities.

Days to anthesis, heights, AUDPCs, observed final severities, and predicted severities at anthesis were analyzed by least squares analysis of variance (9). Sums of squares were partitioned into replication and inbred effects. AUDPCs were compared by single-degree-of-freedom linear contrasts. Means for observed final severities and predicted severities at anthesis were differentiated by Fischer's least significant difference. Pearson's correlation coefficients between severities at each evaluation date and AUDPCs with days to anthesis and plant heights were calculated.

To identify fungi involved in the leaf blight complex, leaf samples were taken from plants in the fourth and tenth replication on 12 July and 2 August 1991, respectively. Five leaf pieces with relatively isolated lesions were selected from different plants throughout the plots of each inbred. Samples were randomly taken, and a variety of lesion types were collected. Leaf samples (about 1.5 × 2.5 cm^2) were surface disinfected for 1 min in a 0.5% NaOCl solution and plated on 20% V8 juice and 1.5% NaOH (V8) agar. Leaf pieces were incubated at 24 C under continuous fluorescent lighting. Fungi growing from leaf pieces were identified microscopically 3–5 days after plating and were subcultured to facilitate identification when necessary.

Greenhouse experiments. Three experiments were conducted during 1990 and 1991 to evaluate differences in susceptibility of seedlings to *P. grisea*. In the first experiment, 12 pearl millet inbreds; 23B, $23A_1$, $23A_4$, 23DB, $23DA_1$, $23DA_4$, 23BE, $23A_1E$, $23A_4E$, 23DBE, $23DA_1E$, and $23DA_4E$ were planted in 10-cm-diameter pots containing equal volumes of coarse building sand, peat moss, and perlite, amended with 8.8 g/L of agricultural gypsum and 5.2 g/L of Osmocote fertilizer (3- to 4-mo release, N-P-K 14-6.1-11.6, Sierra Chemical Co., Milpitas, CA). Plants were grown under natural lighting, and greenhouse temperatures ranged from about 33 C during the day to 23 C during the night. Stands were thinned to five plants per pot.

Plants were inoculated with a bulk inoculum used for screening breeding lines for resistance. The inoculum consists of a mixture of 10 or more isolates of *P. grisea*. Lesions are periodically selected from field- and greenhouse-grown plants, and *P. grisea* is isolated. After determining that isolates are free of contaminants, four 5-mm-diameter plugs from colonies are placed on V8 agar in petri plates and fungi are allowed to colonize the surface under conditions as described above. After about 7 days, plates are placed in a draft-free room and lids are removed for 4 days until the agar has dried. Conidia and mycelia are scraped off the dried agar, and approximately equal quantities of each isolate are bulked and stored in plastic self-sealing bags at −72 C. Isolates are bulked because no differences in pathogenicity to pearl millet among *P. grisea* isolates have been detected to date, and the mixture ensures against loss of pathogenicity of the inoculum from subculturing.

Before being used, packets of conidia were submerged in a 40 C water bath for 5 min. Conidia were suspended in deionized water with one drop of Triton B 1956 per 100 ml. Final inoculum concentration was 4×10^4 conidia per milliliter.

Plants were inoculated when three leaves were fully expanded. Pots were placed in a randomized block design in an inoculation chamber where they were misted to dripping with inoculum. Plants were kept in the chamber overnight and automatically misted with deionized water for 1 min every 30 min. After 18 h, plants were returned to the greenhouse bench in a randomized block design. As a result of damping-off within some of the pots, not all inbreds were represented in each block.

Seven days after inoculation, lengths (L) and widths (W) of lesions on the third leaves of the seedlings were measured from each pot. In the first experiment, inbreds were planted in each of six pots, and up to 10 lesions were measured per pot. In the second and third experiments, inbreds were planted in each of 10 pots, and up to six lesions were measured within each pot. All lesions measured were bounded by healthy leaf tissue. Those that developed on leaf margins were ignored. Over the three experiments, an average of 154 lesions were measured on each inbred. Approximate lesion areas were calculated by $\text{AREA} = (L \times W \times \pi)/4$. Lesion lengths, widths, and areas were analyzed by the general linear model procedure of SAS (9), and means were differentiated by Fisher's least significant difference.

RESULTS

Field experiments. In both 1988 and 1991, inbred was a significant source of variation ($P < 0.01$) for AUDPC, and differences could be discerned among inbreds (Table 1). Single-degree-of-freedom contrasts indicated that substitution with the e_1 allele resulted in greater AUDPCs (Table 2). In both years, no other allele or the cytoplasms had any effect on leaf blight.

In 1991, differences existed among inbreds ($P < 0.01$) not only for AUDPC, but also for heading date, height, final disease ratings, and predicted disease severities at anthesis. Mean separation of 1991 final disease severities revealed lower severities on inbreds without the e_1 allele (Table 3). However, comparison of predicted severities at anthesis resulted in an opposite ranking. Inbreds with the e_1 allele had lower severities predicted at anthesis.

Leaf blight ratings made early in the 1991 season were not correlated with those made later in the season. Days to anthesis

TABLE 1. Mean values for area under the disease progress curves for near-isogenic, alloplasmic pearl millets evaluated in 1988 and 1991

Gene substitution	Cytoplasm		
	B	A_1	A_4
1988			
23	309.8	328.0	361.3
23D[x]	401.2	419.9	350.8
23E[y]	766.5	907.2	897.5
23S[z]	397.6	329.9	305.9
23DE	911.4	873.8	856.0
23DS	355.3	314.9	320.3
1991			
23	843.5	904.0	986.8
23D	637.8	759.8	813.5
23E	1,727.0	1,702.5	1,770.4
23DE	1,338.0	1,124.7	1,543.4

[x] D indicates homozygous substitution with the d_2 allele for dwarfness.
[y] E indicates homozygous substitution with the e_1 allele for earliness.
[z] S indicates homozygous substitution with the *tr* allele for trichomelessness.

was positively correlated with leaf blight ratings of younger plants, but correlations became negative by 57 days after planting (Table 4). Inbreds with the e_1 allele reached anthesis about 51 days after planting (Table 3), so a rapid increase in leaf blight must have occurred on the early inbreds after anthesis. Height was not consistently correlated with leaf blight ratings across dates.

Isolations from leaves in 1991 indicated several fungi were involved in the leaf blight complex. Not enough isolations were made to discern differences in infection by the different fungi among inbreds, therefore data were pooled across inbreds. A total of 256 fungi were examined from the 12 inbreds at the two sampling dates.

Fungi isolated and known to be pathogenic based on previous inoculations and their frequency of isolation were *P. grisea* (28.1%), *Phyllosticta penicillariae* (7.0%), *Gleocercospora sorghi* (4.7%), *Bipolaris setariae* (2.7%), *Drechslera dematioidea* (2.0%), and *Exserohilum rostrata* (0.4%).

Potential pathogens isolated included *Curvularia* spp. (28.1%), *Fusarium* spp. (14.8%), and miscellaneous "*Helminthosporium*" spp. (3.9%). Several species of each genus were present, as determined by conidium morphology. None of these isolates were evaluated for pathogenicity. The remaining fungi isolated were considered saprophytes, such as *Alternaria* spp. (4.7%) and *Cladosporium* spp. (0.4%), or were unidentified (3.1%).

Isolation frequencies of the fungi were generally similar across sampling dates, with the exception of *Fusarium* spp., which comprised 1.2% of the isolations from the first sampling date, and 21.3% from the second.

Greenhouse experiments. No lesions developed on $23DA_1E$, and this inbred was excluded from the analyses. Inbred differences were significant ($P < 0.01$) for lesion length and area, but not for width (Table 5).

Some inbreds without the e_1 allele were more susceptible than corresponding inbreds with it, as indicated by larger lesion dimensions. Inbreds 23B, $23A_1$, and $23DA_1$ had longer lesions than 23BE, $23A_1E$, and $23DA_1E$, respectively (Table 6). Inbreds 23B and $23DA_1$ had wider lesions than 23BE and $23DA_1E$, respectively. Inbreds 23B, $23A_1$, $23A_4$, and $23DA_1$ had greater lesion areas than did 23BE, $23A_1E$, $23A_4E$, and $23DA_1E$, respectively.

TABLE 2. Single-degree-of-freedom linear contrasts of area under the disease progress curves for leaf blight of near-isogenic, alloplasmic pearl millets evaluated during 1988 and 1991

	Mean squares	
Contrast	1988	1991
B vs. A_1 cytoplasm	14.1	38.2
B vs. A_4 cytoplasm	34.7	4,030.0
A_1 vs. A_4 cytoplasm	93.2	4,853.2
Trichomed vs. trichomeless	303.8	...[y]
Late vs. early	128,476.8**[z]	151,272.6*
Tall vs. dwarf	370.0	24,567.0
Error	9,543.0 (df = 84)	37,537.6 (df = 99)

[y] Trichomeless inbreds were not evaluated in 1991.
[z] * and ** indicate significance at $P = 0.05$ and 0.01, respectively.

TABLE 3. Observed and predicted leaf blight severities and days to anthesis of near-isogenic, alloplasmic pearl millets evaluated in 1991

	Leaf blight severity (%)		
Inbred	Observed final	Predicted at anthesis	Days to anthesis
23BE	96.2 a[z]	4.2 a	51.5 a
$23A_4E$	96.1 a	3.2 a	49.8 a
$23A_1E$	95.8 a	2.7 a	49.5 a
$23DA_4E$	89.7 a	3.2 a	50.2 a
23DBE	79.2 b	3.8 a	51.6 a
$23DA_1E$	73.8 b	1.7 a	50.9 a
$23A_4$	58.5 c	20.1 c	70.1 bc
$23A_1$	55.0 cd	17.2 c	69.6 b
$23DA_4$	52.0 cd	16.2 bc	70.4 bc
23B	50.7 cd	20.0 c	72.3 cd
$23DA_1$	49.1 de	13.0 b	69.4 b
23DB	41.3 e	16.8 bc	74.3 cd
LSD ($P = 0.05$)	8.2	4.0	2.2

[z] Means within a column followed by the same letter do not differ ($P = 0.05$).

DISCUSSION

Substitutions for the *tr* and d_2 alleles and the A_1, A_4, and B cytoplasms did not affect leaf blight development in the field. During both 1988 and 1991, the e_1 allele was associated with an apparent increased susceptibility to leaf blight when only observed AUDPCs were examined.

Shaner et al (10) observed a relationship between later heading date in wheat (*Triticum aestivum* L.) and reduced severity of septoria leaf blotch, caused by *Septoria tritici* Rob ex. Desm. The greater resistance was hypothesized to be the result of either escape from disease, a differing physiology of cultivars with delayed senescence, or linkage of loci with alleles that confer resistance and maturity. The resistances of several cultivars were ranked similarly, with only minor modifications, after disease progress curves were adjusted for heading date.

Data from 1991 allowed us to calculate predicted disease severities at a common developmental stage, which indicated that inbreds homozygous for the e_1 allele were more resistant at anthesis than inbreds without the allele. These results indicate that evaluation of leaf blight at a common growth stage is essential for comparison among cultivars.

Previous observations and isolations made in 1991 suggest that *P. grisea* is a major component of the leaf blight complex. The finding of reduced lesion dimensions on seedlings with the e_1 allele when inoculated with *P. grisea* supports the results obtained by correcting the 1991 disease progress curves for anthesis date. Lower leaf blight severities at anthesis on the early inbreds may be due to the increased resistance associated with the e_1 allele.

Isolations from the 1991 field experiment indicated that, although *P. grisea* is an important leaf-blighting pathogen, several other fungi are involved in the disease complex. Differences in disease reactions among inbreds between the field and greenhouse studies may be due in part to differences in susceptibility to some of these other leaf-blighting pathogens, particularly as the plants mature. The susceptibility of Tift 23B to *B. setariae* increases as the plants mature (12). Effects of host development on susceptibility to the other pathogens is not known.

There appeared to be an interaction of the e_1 and d_2 alleles in the A_1 cytoplasm that prevented the development of lesions on seedlings of $23DA_1E$ in greenhouse inoculations with *P. grisea*. This resistance probably loses some of its effectiveness as plants mature. In the 1991 field experiment, 27 fungi were isolated from

TABLE 4. Pearson's correlation coefficients (r) between days to anthesis and height with leaf blight ratings of near-isogenic, alloplasmic pearl millets evaluated in 1991

	Leaf blight severity (%) at days after planting							
	25	34	47	57	68	76	88	AUDPC
Days to anthesis	0.22*[z]	0.21*	0.49**	−0.29**	−0.71**	−0.77**	−0.84**	−0.78**
Height	−0.13	−0.04	0.42**	0.17+	−0.10	−0.11	−0.18+	−0.10

[z] +, *, and ** indicate significant correlations at $P = 0.10$, 0.05, and 0.01, respectively.

TABLE 5. Analyses of variance of lesion dimensions caused by *Pyricularia grisea* on seedlings of near-isogenic, alloplasmic pearl millets in the greenhouse

Source		df	Mean squares		
			Length	Width	Area
Experiment		2	46.61**[z]	60.37*	772.54**
Replication(exp)	Error a	23	1.96	16.52*	24.08
Inbred		10	6.69**	17.07+	79.12**
Exp × inbred		20	4.55**	11.08	47.07**
Rep × inbred(exp)	Error b	210	2.01**	9.93**	18.42**
Error c		1,428	1.30	5.49	11.86

[z] +, *, and ** indicate significance at $P = 0.10$, 0.05, and 0.01, respectively.

TABLE 6. Lengths, widths, and areas of lesions caused by *Pyricularia grisea* on the third leaf of seedlings of near-isogenic, alloplasmic pearl millets inoculated in the greenhouse

Inbred	Lesion dimensions		
	Length (mm)	Width (mm)	Area (mm^2)
23B	3.45 a[y]	2.43 a	5.88 a
$23A_1$	3.39 ab	1.89 ab	5.55 a
$23A_4$	3.15 bc	1.73 b	4.73 b
23BE	3.00 cd	1.70 b	4.62 bc
$23DA_4$	2.93 cd	1.65 b	4.45 bc
$23DA_1$	2.84 d	1.67 b	4.23 bc
$23A_1E$	3.05 cd	1.59 b	4.12 bc
23DBE	2.92 cd	1.55 b	3.96 bc
$23A_4E$	2.99 cd	1.52 b	3.94 c
23DB	3.13 bc	1.46 b	3.87 c
$23DA_4E$	2.83 d	1.55 b	3.84 c
$23DA_1E$	0.00*[z]	0.00*	0.00*
LSD ($P = 0.05$)	0.26	0.54	0.80

[y] Mean values within a column followed by the same letter are not significantly different ($P = 0.05$).

[z] No lesions formed, and this inbred was not included in the analyses of variance.

the 10 lesion samples of $23DA_1E$. Four of these isolations were *P. grisea*.

The reduced leaf blight susceptibility at anthesis and increased resistance to *P. grisea* associated with the e_1 allele may be an additional advantage for its use in breeding grain varieties of pearl millet. It is being incorporated into promising grain types because reducing the time from planting to harvest helps to avoid drought stress and allows greater flexibility in double-cropping and rotation systems. Earliness is not a desired character in forage production (4), so the allele will have limited usefulness in forage pearl millet improvement.

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