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GENETIC CONTROL AND HERITABILITY OF RESISTANCE TO GRAIN MOLD IN $F_{2:5}$ SORGHUM FAMILIES WITH NON-PIGMENTED TESTA

ABSTRACT.

Grain mold limits the productivity of food-type *Sorghum bicolor* by reducing grain yield and quality. The disease is caused by a complex of fungi including *Curvularia lunata*, *Fusarium sp.*, *Alternaria sp. Phoma sp*., and *Helminthosporium sp*. The presence of many fungal species and environmental factors make breeding and selecting for tolerance difficult. It is the goal of many sorghum breeding programs to improve this trait. However, further information on the heritability of the trait is needed. The objectives of this study were: (1) to determine the genetic control and heritability of resistance to grain mold in 131 $F_{2:5}$ derived sorghum families with non-pigmented testa and (2) to estimate the expected genetic advance under various environments of evaluation. Seed from 131 $F_{2:5}$ derived families and their parents (RTx430 and Sureño) were evaluated in six environments in Texas over three years. In all environments, grain mold was rated at 40-45 days after flowering. Significant variation was detected in environments, genotypes and the genotype * environment interaction. Across all environments, broad sense heritability was estimated as 0.86 and narrow sense heritability was estimated as 0.59. Genetic advance estimates for grain mold resistance ranged from 5-16% with a selection intensity of 10%. A minimum of four genes segregating for grain mold resistance was estimated, depending upon methodology. The results indicate that additive variance is important for grain mold resistance and that improvement in this trait may be realized through breeding.

Key words: *Alternaria, Curvularia Fusarium, Helminthosporium,* recombinant inbred lines

INTRODUCTION

Grain mold is a world wide disease of sorghum (*Sorghum bicolor* (L.) Moench). That has serious implications for the utilization of the caryopsis for food or feed. Molds discolor the grain, break down the endosperm and significantly deteriorate processing qualities. Mold damaged or weathered grain cannot be decorticated; the flour or grits are badly discolored and cannot be used for food (Prom, *et al.* 2005). Thus, resistance to grain mold is considered to be

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the most important factor in food quality of sorghum grain (Rosenow *et al.* 1995).

About 40 genera of fungi have been detected in moldy sorghum grain, and some of these are capable of producing mycotoxins. The most common fungal species associated with sorghum grain mold are *Fusarium thapsinum* (Montes *et al.* 2003)*, Curvularia sp., Cladosporium sp., Alternaria sp*. (Stack and Pedersen 2003), and *Fusarium verticillioides* (Funnell and Pedersen 2004). The severity of grain mold is strongly influenced by environment with the greatest increases in disease occurring in warm and humid (or wet) weather, especially after physiological maturity of the grain (Montes *et al.* 2003; Williams and Rao 1981).

While sorghum lines and hybrids with improved tolerance to grain mold and weathering have been developed, further improvements are continually needed (Bejosano *et al.* 2001). However, selection for grain mold resistance is difficult because the inheritance of resistance is complex due to the presence of different mechanisms of resistance, numerous different causal pathogens, and strong environmental effect (Audilakshmi *et al.* 2005).

Sorghum geneticists have long suspected that both qualitative and quantitative loci influence grain mold resistance. Esele *et al.* (1993) showed that several qualitatively inherited pericarp traits such as color and pigmented testa influence the level of grain mold resistance.While several qualitative loci affect grain mold resistance, they do not account for all the variations observed for grain mold resistance in sorghum. Therefore, resistance to grain mold in sorghum is considered a quantitatively inherited trait. Using a diallel design, Dabholkar and Baghel (1980) found that general and specific combining ability components of variation for grain mold resistance were highly significant. Rodriguez *et al.* (2000) indicated that the genetic effect for grain mold resistance across environments may be more complicated than a simple additive-dominance model. All of these factors cause significant genotype-environment interactions (G × E) that reduce the accuracy for estimating disease resistance and selecting appropriate germplasm (Rodriguez-Herrera *et al.* 1997).

Although extensive research has been done trying to identify the chemical and physical properties of sorghum associated with grain mold resistance (Williams and Rao 1981). However, high seed tannin content adversely affects digestibility and utilization of sorghum grain as food (Harris and Burns 1973). In the semi arid tropics where sorghum is grown, more than 50% of sorghum grain production is used as human food. Thus, research on grain mold resistance should focus on factors that contribute to resistance and are consistent with the acceptability of grain as human food (Williams and Rao 1981). The objectives of the present study were: (1) to determine the genetic control and heritability of resistance to grain mold in 131 $F_{2:5}$ derived sorghum families with non-pigmented testa, and (2) to estimate the expected genetic advance under various environments of evaluation.

MATERIALS AND METHODS

Germplasm development

The experimental material consisted of 131 F_2-derived in F_5 recombinant inbred lines (RIL). These RIL were developed by single-seed descent from the cross between Sureño and RT × 430. Sureño is a dual-purpose grain and forage variety, with resistance to grain molding (Meckenstock *et al.* 1993). Sureño does not have a pigmented testa and is known for excellent grain quality for both food and feed purposes. RT × 430 is a widely adapted inbred line with excellent combining ability, and is a common restorer line in many U.S. grain sorghum hybrids. It is highly susceptible to grain mold (Miller 1984). RT × 430 has a yellow endosperm and it does not have a pigmented testa. In the summer of 1993, at Lubbock, TX, hand-emasculated panicles of Sureño were pollinated with pollen from RT × 430 plants. In the winter of 1993, parents and F_1 seed were planted in Puerto Rico to produce an F_2 population through self-pollination. From this F_2 population, 150 panicles were randomly self-pollinated and then these were advanced at various locations associated with the Texas A&M sorghum program. In the final generation $F_{2:5}$ seed were bulked for replicated evaluation sites

In 1996, the test was planted at College Station, TX under two moisture levels (with and without sprinkler irrigation). Sprinkler irrigation was applied 4 hours, once a week during grain development to enhance the grain mold severity. These environments are subsequently denoted CD96 and CW96, respectively. In 1997, the test was planted at Beeville, TX, Corpus Christi, TX, and College Station, TX (under two moisture levels). These environments are subsequently denoted BE97, CC97, CD97 and CW97, respectively.

Agricultural practices standard for each region were used in all of these locations. At all environments, significant levels of grain mold occurred naturally; therefore inoculation was not necessary, although fungal species predominance varied across locations (Rodriguez-Herrera *et al.*, 2006). In all experiments, grain mold was rated at 40-45 days after flowering on all RIL. Grain mold was recorded on a 1 to 5 scale (Frederiksen *et al.* 1991), where: 1 = seed bright, free from mold damage. 2 = moderately resistant to mold, seed slightly discolored, 3 = moderately susceptible, considerable discoloration, 4 = susceptible, extensive discoloration and deterioration of seed, and 5 = very susceptible, seed essentially all dead, embryos dead and endosperm deteriorated. To stabilize the variances before analysis, the data were transformed with the log of data + 1 (Lentner and Bishop 1993).

Experimental design

At each location, the experimental design was a randomized complete block design with two replications. The $F_{2:5}$ lines and the parents were treatments. The effects of $F_{2:5}$ lines were considered random, and the parents were considered fixed. The experimental unit was one row 6.3 m in length, with a row to row spacing of 0.76 m. Separate analyses of variance were conducted for each environment. Homogenity of variance of each individual analysis indicated that combined anal-

ysis was appropriate, so a combined analysis was performed using data from all six environments. Procedure univariate (SAS 1990) was used to estimate variance, means of parents and RIL, and to produce the normal probability plot. Differences between the mean of the ten highest scores and RT × 430, and between the mean of the 10 lowest scores and Sureño were compared by means of Least Significant Differences ($P< 0.05$). These comparisons were made in each environment.

Heritability estimates

The combined analysis of variance was used to estimate broad-sense heritability from the variance component. Broad sense heritability on an entry-mean basis (Nyquist 1991) was calculated as follows:

$$H^2 = \frac{\sigma_g^2}{\left[\frac{\sigma_{error}^2}{r \times E} + \frac{\sigma_{g \times e}^2}{E} + \sigma_g^2\right]}$$

where:
σ_g^2 — genetic variance,
σ_{error}^2 — experimental error,
$\sigma_{g \times e}^2$ — genotype × environment interaction variance,
r — number of replications,
E — number of environments,
H^2 — broad-sense heritability

Values were derived from variance components estimated using expected mean squares (Dudley and Moll 1969). The 90% confidence intervals for heritability were calculated on a progeny mean basis (Knapp *et al.* 1985).

Narrow-sense heritability of grain mold resistance was estimated for each environment to identify the effect of the specific environment on the expected gains for selection. Narrow-sense heritability estimates were based on variance components estimates on a progeny mean basis at each environment using the following formula:

$$h^2 = \frac{\sigma_a^2}{\sigma_p^2}$$

where:

σ_a^2 — additive genetic variance for $F_{2:5}$ lines; $\sigma_a^2 = \frac{\sigma_{among\ F_{2:5}\ lines}^2}{\left(15/16\right)}$,

σ_p^2 — phenotypic variance; $\sigma_p^2 = \sigma_a^2 + \frac{\sigma_{error}^2}{r}$,

h^2 — narrow-sense heritability,
σ^2_{error} — error mean square,
r — number of replications, (Montoya *et al.* 1997).

The 90% confidence intervals for heritability were calculated on a progeny mean basis (Knapp *et al.* 1985).

Minimum number of genes controlling grain mold resistance

Quantitative estimates of the number of genes segregating for grain mold resistance were made using the formula:

$$N = \frac{(GR)^2}{4.27 \times \left[\sigma^2_{F_{2:5}} - \frac{\left(\sigma^2_{P_R} - \sigma^2_{P_S} \right)}{2} \right]}$$

Minimum number of genes controlling grain mold resistance was estimated using two different values of genotypic range (GR). *GR* was estimated as the difference between the two parents $(P_R - P_S)$ and as the phenotype range of the $F_{2:5}$ lines.

P_R — the mean of the resistant parent,
P_S — the mean of the susceptible parent
$\sigma^2_{P_R}$ — the variance of the resistant parent
$\sigma^2_{P_S}$ — the variance of the susceptible parent
$\sigma^2_{F_{2:5}}$ — the variance of the $F_{2:5}$ generation,
N — the estimated number of segregating genes (Bjarko and Line 1988).

Genetic advance

The genetic advance (Ga) expected from a 10% selection intensity within each environment was calculated for grain mold resistance and reported as percentage according to the standard formula (Fehr 1991):

$$Ga = h^2 \times k \times \sigma_p$$

where:
h^2 — heritability,
k — standardized selection differential,
σ_p — phenotypic standard deviation ($F_{2:5}$)

RESULTS

Grain mold severity was high in all six test environments, with environmental means for grain mold score ranging from 3.12 at CD97 to 4.82 at CW96

(Table 1). The cross between Sureño and RT × 430 produced $F_{2:5}$ lines that exhibited a wide range of variation in grain mold severity at the four environments in 1997. In 1996, at College Station, TX, unusual heavy and consistent rainfall occurred for approximately two weeks at grain maturity, resulted in grain mold scores of 4.39 for Sureño and 4.99 for RT × 430. This resulted in skewed grain mold scoring.

Table 1

Environmental means, means of parental lines and $F_{2:5}$ families, and range for the $F_{2:5}$ families for the grain molding score in six test environments in Texas

Environment	Means				Range $F_{2:5}$ families
	Overall	RT × 430	Sureño	F2:5 families	
CD96	4.79	4.99	4.41	4.55	3.75 ÷ 5.00
CW96	4.82	4.99	4.36	4.54	3.25 ÷ 5.00
CD97	3.12	4.23	1.81	3.35	2.15 ÷ 4.50
CW97	3.37	4.46	2.11	3.43	2.25 ÷ 4.65
BE97	3.19	4.75	1.63	3.47	2.00 ÷ 4.75
CC97	3.24	4.23	2.02	3.32	2.25 ÷ 4.51

Analysis of variance showed significant differences for grain mold severity among RIL and between parents at each environment (Table 2).

Table 2

Calculated means squares for the individual analysis of variance on grain mold severity for 131 $F_{2:5}$ sorghum families Sureño and RT × 430 grown at 6 environments.

Source of variation	df	Environments					
		CD96	CW96	CD97	CW97	BE97	CC97
Replication	1	0.002ns	0.005ns	0.113***	0.043***	0.793***	0.001ns
Genotypes	131	0.005***	0.007***	0.053**	0.051***	0.079***	0.053***
Parents (P)	1	0.091***	0.104***	3.119***	2.648***	4.987***	2.476***
$F_{2:5}$ families	129	0.004***	0.006***	0.029***	0.031***	0.039***	0.034***
P vs $F_{2:5}$ families	1	0.017ns	0.028ns	0.059ns	0.058ns	0.117ns	0.061ns
Error	159	0.002	0.002	0.011	0.008	0.016	0.012
Total	291						
C.V		2.4	2.3	7.5	6.1	8.7	7.6

, * Significant at the 0.01 and 0.001 probability levels, respectively, ns, nonsignificant

The combined analysis of variance for the $F_{2:5}$ families indicated that there were significant differences among environments, and genotypes ($F_{2:5}$ families) (Table 3). In addition, the G × E interaction was significant for grain mold severity, which indicates that there are shifts in performance of genotypes across environments. In all environments, transgressive segregation was observed for lines more susceptible than RT × 430, but lines more resistant than Sureño were observed in only two of the six environments (Table 1).

Table 3

Expected and calculated means squares for the combined analysis of variance on grain mold severity for 131 $F_{2:5}$ derived lines grown at six environments

Source of variation	Expected mean squares	df	Type III mean square
Environment (E)	$\sigma^2_{error} + 2\times\sigma^2_{g\times e} + 129\times\sigma^2_{r(e)} + 258\sigma^2_e$	5	7.042**
Replication (E)	$\sigma^2_{error} + 129\times\sigma^2_{r(e)}$	6	0.163**
$F_{2:5}$ families	$\sigma^2_{error} + 2\times\sigma^2_{g\times e} + 12\times\sigma^2_g$	129	0.091**
$F_{2:5}$ families × E	$\sigma^2_{error} + 2\times\sigma^2_{g\times e}$	645	0.012**
Error	σ^2_{error}	775	0.009
Total		1560	

**, Significant at 0.01 probability level

The combined broad-sense heritability for grain mold was estimated at 0.86 with a 90% confidence interval of 0.89-0.83 (Table 4). The narrow sense heritability estimates averaged 0.65 with a range of 0.56 to 0.73. Narrow sense heritability estimate combining data of six environments was 0.59 with a 90% confidence interval of 0.69-0.51. These relatively high heritabilities are similar in magnitude to the estimates obtained in a random mating population of sorghum for grain mold resistance (Ibrahim *et al.* 1985).

Table 4

Narrow (h^2) and broad (H^2) sense heritability estimates for grain mold resistance in the cross of Sureńo * RTx430 and expected gains from selection at 6 environments

Environment	Narrow sense heritability (h^2)	90 % Confidence interval		Gain from selection [%]
		upper	lower	
CD96	0.64	0.69	0.53	5
CW96	0.73	0.76	0.64	7
CD97	0.62	0.67	0.51	13
CW97	0.72	0.76	0.63	16
BE97	0.56	0.62	0.42	14
CC97	0.66	0.71	0.55	15
Combined heritability				
Broad (H^2)	0.86	0.89	0.83	
Narrow (h^2)	0.59	0.69	0.51	11

The expected gains, from selection of the top 10% of progenies in each environment for grain mold resistance, ranged from 5-16% (Table 4). The low expected gains from selection of CD96 and CW96 were due to extreme grain mold pressure in these environments which narrowed the phenotypic variation. In 1997, the gains from selection were similar, ranging from 13-15%.

In these environments and for this population, the number of genes controlling grain mold resistance was 2.7 while the method utilizing differences between parents resulted in an estimate of 3.68. Both methods for estimating

number of genes assume that no linkage exists between the loci involved, the effect of all loci involved are equal, no dominance, and no epistasis. The main difference is that the method using the difference of the parental means to estimate the genotypic range assumes that all genes for resistance are in a single parent of the cross and the method using the phenotypic range of the segregating population as an estimate of the genotypic range does not (Bjarko and Line 1988).

DISCUSSION

Analysis of variance of individual environments showed significant differences for grain mold severity among genotypes (Table 2). This suggests that environmental conditions and fungal inoculum at each location were appropriate to differentiate among genotypes based on grain mold resistance. Also, analysis of variance revealed significant differences between parents at each environment. No statistical differences were observed between the mean of parents and the mean of $F_{2:5}$ families suggesting that additive genes may be involved in grain mold resistance.

The combined analysis of variance for the $F_{2:5}$ families indicated that the effect due to environment was significant (Table 3). The environmental conditions that have the most influence on microorganism-related seed deterioration are moisture, temperature, and oxygen, but in most systems, moisture is the factor of overriding importance (Mc Gree 1986). In the present study, genotype ($F_{2:5}$ families) and GxE interactions were significant for grain mold severity. Indira *et al.* (1991) evaluated 91 inbred lines at four locations and found that the genotypes showed significant GxE interactions. Thus, multiple location trials would be more effective for distinguishing genotypic differences and selecting progeny with desirable grain mold resistance.

The $F_{2:5}$ families' distribution plot after elimination of the 1996 environments showed that $F_{2:5}$ families approximated a normal curve (Fig 1), suggesting that expression of grain mold resistance is polygenic.

The mean of the 10 most resistant $F_{2:5}$ lines was not significantly different from the mean of the resistant parent (Sureńo) in 5 of 6 environments.

Broad sense heritability estimates after combining data from all six environments was 0.86 with a 90% confidence interval of 0.89-0.83 (Table 4). These high estimates of broad sense heritability suggest that improvements in grain mold resistance can be realized through breeding.

The narrow sense heritability estimates averaged 0.65 with a range of 0.56 to 0.73 (Table 4). Dabholkar and Baghel (1983) reported high estimates of narrow-sense heritability for grain mold resistance (0.81 and 0.52) at two different environments using data from a diallel analysis. One possible explanation of these relatively high heritability estimates obtained by Dabholkar and Baghel (1983) is that heritabilities were estimated for a single environment, and genotype x environment interaction is not accounted for in the total phenotypic vari-

ance. In our study, narrow sense heritability estimate combining data of six environments was 0.59 with a 90% confidence interval of 0.69-0.51. This suggests that early generation selection for grain mold resistance in sorghum may be effective when Sureńo is used as a source of resistance. Dabholkar and Baghel (1980) indicated that additive variance, dominance and non allelic interactions are important in the sorghum expression to *Curvularia spp*. infection.

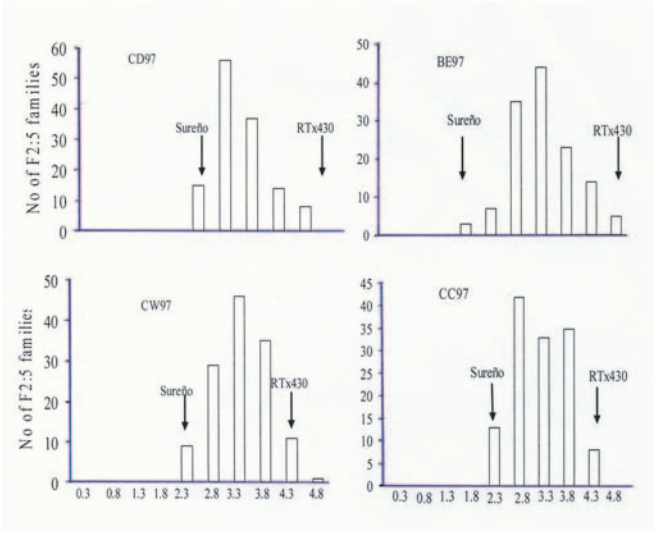

Fig. 1. Distribution of grain mold rating of 131 $F_{2:5}$ families at four environments in 1997

The expected gains, from selection of the top 10% of progenies in each environment for grain mold resistance, ranged from 5-16% (Table 4). This rate of gain was based on a moderate intensity of selection (10%), which allows high gains from selection, but could reduce the genetic variability for grain mold resistance in the short term. Results indicated that over both years, experiments with sprinkler irrigation had a higher gain for selection.

Quantitative estimates of the number of genes segregating for grain mold resistance were lower when the phenotype range in $F_{2:5}$ families was used to estimate genotypic rate (2.76), than when the difference between parents was used as a measure of genotypic range (3.68). In both methods, the presence of linkage, dominance, or unequal effects at different loci would result in an underestimation of the actual number of segregating genes present, while the presence of epistasis may cause either an overestimation or an underestimation of the actual number of segregating genes (Falconer and Mackay, 1996). The method of estimating number of genes using the difference between parents seems more appropiate in the present study because of the high susceptibility of RT × 430 to grain mold. Thus, four genes may be the minimum number of genes governing grain mold resistance. Shivanna *et al.* (1994) reported that inheritance of

grain mold resistance in sorghum may be governed by four independently segregating genes, two with complementary intergenic interactions (between them) and the other two having additive interaction. The method using the difference between parents provides, in most cases, a conservative estimate of the number of genes involved (Falconer and Mackay, 1996), Additionally, because several different traits have been associated with grain mold resistance (panicle shape, grain hardness, antifungal proteins, plant height, prolamins, thickness of the mesocarp, etc) (Harris and Burns, 1973; Esele *et al.*, 1993; Bejosano *et al.*, 2001; Prom *et al.*, 2005). In this case, it seems that linkage among these traits may contribute to underestimating the real number of genes conferring grain mold resistance in sorghum for food.

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