Euphytica **116:** 95–103, 2000.

Genetic analysis of grain mould resistance in coloured sorghum genotypes

S. Audilakshmi[1*], J.W. Stenhouse & T.P. Reddy[2]

*Sorghum Breeding Unit, International Crops Research Institute for the Semi-Arid Tropics, Patancheru, Andhra Pradesh 502 324, India; [1]Present address: National Research Centre for Sorghum, Rajendranagar, Hyderabad 500 030, India; [2]Department of Genetics, Osmania University, Hyderabad 500 007, India; (*author for correspondence; e-mail: nrcshyd@ap.nic.in)*

Received 27 April 1999; accepted 4 February 2000

Key words: disease resistance, frequency distribution, gene action, generation mean analysis, segregation pattern, *Sorghum bicolor*

Summary

Grain moulds are a major constraint to sorghum production and to adoption of improved cultivars in many tropical areas. Information on the inheritance of grain mould reaction is required to facilitate breeding of resistant cultivars. The genetic control of grain mould reaction was studied in 7 crosses of 2 resistant sorghum genotypes. P_1, P_2, F_1, F_2, BC_1 and BC_2 families of each cross were evaluated under sprinkler irrigation for field grade and threshed grade scores and subjected to generation mean analysis. Frequency distributions for grain mould reaction were derived and F_2 and BC_1 segregation ratios were calculated. Grain mould reaction in crosses of coloured grain sorghum was generally controlled by two or three major genes. Resistance to grain moulds was dominant. Significant additive gene effects were also found in all cross/season combinations. Significant dominance effects of similar magnitude to additive effects were also observed in five out of ten cross/season combinations. Gene interactions varied according to the parents with both resistant and susceptible parents contributing major genes. Choice of parents with complementary resistance genes and mechanisms of resistance will be critical to the success of resistance breeding.

Introduction

Sorghum (*Sorghum bicolor* (L.) Moench) is grown worldwide for food, feed, fodder, fuel and industrial products. It is cultivated widely throughout tropical, subtropical and temperate regions, between latitudes 45°N and 45°S. Roughly 95% of the world's sorghum area lies in developing countries, mainly in Africa and Asia (ICRISAT and FAO, 1996). The crop is grown predominantly by subsistence farmers in areas subject to low rainfall and drought where it is used as food.

Most improved sorghum varieties and hybrids mature earlier than local varieties, often before the end of the rainy season. This results in increased exposure of the developing and maturing grain to conditions of high humidity and wetness. Grain moulds develop under these conditions and bring about decreased filling and size of the grain and chalky endosperm, which disintegrates during harvest and threshing. The moulded grain becomes unfit for human consumption because of mycotoxin formation and reduced grain quality, leading to non-remunerative prices in the market and losses to farmers. Grain moulds are estimated to cause losses worth US$ 130 million per annum globally (ICRISAT, 1992), in sorghum grain yields.

In studies conducted in the USA, Africa and India, it has been shown that *Fusarium monoliforme, F. semitectum* and *Curvularia lunata* are the major causal agents of grain moulds (Murty et al., 1980; Williams & Rao, 1981; Anonymous, 1976). In Texas, the predominant field fungi belong to the genera *Alternaria, Fusarium,* and *Curvularia* (Castor & Frederiksen, 1980). Only a few fungi are thought to infect sorghum spikelet tissues during the early stages of grain development. In approximate order of importance, these are *Fusarium monoliforme, Curvularia lunata, F. semitectum*, and *Phoma sorghina* (Forbes et al., 1992).

Table 1. Parents and crosses used in the study

Resistant / Susceptible	IS 14375 (R_1)	IS 14387 (R_2)
MS 422B (S_1)	**	**
SP 33316 (S_2)	*	*
GM 15018 (S_3)		*
AKMS 14B (S_5)	**	
AKR 150 (S_6)	*	

* Crosses made during 1995.
** Crosses made during 1995 and 1996.

Bandyopadhyay & Mughogho (1988), found that mould resistance screening without inoculation with mould causing fungi was feasible if sprinkler irrigation was used, and developed a field screening technique to determine grain mould reactions of sorghum genotypes. When ambient relative humidity was low, however, sprinkler irrigation could also be ineffective in maintaining suitable conditions for grain mould development. Using this technique, 156 germplasm accessions that were resistant to grain moulds were identified over several years of testing (Bandyopadhyay et al., 1988).

Study of the number, nature and diversity of genes controlling resistance should ideally precede exploitation of any genotype in a resistance-breeding programme. In an earlier report, the associations of grain mould resistance with various other characters and probable mechanisms of grain mould resistance were published (Audilakshmi et al., 1999). The present investigation was undertaken to determine the genetic basis of grain mould resistance in crosses between two resistant and five susceptible lines.

Materials and methods

Two grain mould resistant and five susceptible genotypes (listed in Table 1) were selected from the world germplasm collection for crossing on the basis of evaluations in 1994 (Audilakshmi, 1997; Audilakshmi et al., 1999). Seven crosses made during the 1994 rainy season, and three in the 1995 rainy season, are also given in Table 1. The F_1 hybrids and the parent lines were raised during the 1994 and 1995 post-rainy seasons and additional crosses were made to generate six families (P_1, P_2, F_1, BC_1, BC_2, and F_2) in each cross. The crosses and families were grown separately during the 1995 and 1996 rainy seasons at ICRISAT, Patancheru, Andhra Pradesh, India. The six families of each cross were grown in a randomised complete block design with three replications. Plot size varied for different families. Parental lines, F_1, BC_1 and BC_2, were grown in single-row plots and F_2s were grown in 4-row plots of 4 m grown on ridges with 0.75 m between ridges. The experimental materials were screened for grain mould resistance under sprinkler irrigation. In each replication, observations were recorded on ten random plants from P_1, P_2, and F_1, on 15 plants from BC_1 and BC_2, on 70 to 75 plants from F_2 in 1995 and on 65 plants from F_2 in 1996.

Field screening technique for grain moulds. The screening technique followed was that of Bandyopadhyay & Mughogho (1988). Sprinklers were arranged in a sequence grid pattern, the shortest distance between any two sprinklers being 12 m. The test plots were sprinkled for 1 h in the morning if it did not rain the previous night and same morning, and for an additional 1 h in the evening if it did not rain throughout the day. Overhead sprinkler irrigation was provided on this basis from flowering to grain maturity (black layer formation) and up to 2 weeks later when panicles were harvested.

Grain mould damage was evaluated after harvest as field grade (FGS) and threshed grade (TGS) scores. Panicles/threshed seeds from the plants of different families indicated above were scored visually for mould severity at 54 d after 50% flowering. FGS and TGS were recorded on a scale of 1 to 9, where 1 = free from mould, 2 = 5% of the panicle moulded, 3 = 10% of the panicle moulded, 4 = 15% of the panicle moulded, 5 = 30% of the panicle moulded, 6 = 40% of the panicle moulded, 7 = 50% of the panicle moulded, 8 = 60% of the panicle moulded, and 9 = $>$70% panicle moulded.

Generation mean analyses were carried out on the original and square root transformed data. Genetic effects of the generation means were estimated by a weighted least square regression (WLSR) analysis (Cavalli, 1952; Hayman, 1958) using the notation and definitions of Mather & Jinks (1977; pp. 36–67). Where m = mean, d = additive effects, h = dominance effects, i = additive $\times$ additive interactions, j = additive $\times$ dominance interactions, l = dominance $\times$ dominance interactions.

Since generation means of parents and progenies were estimated with equal percision, each generation was weighted by the variance of the mean for that generation. The equation fitted for Least Square Regression was

$$\underline{Y} = \underline{X}\underline{B} + E$$

$\underline{Y}$ = Vector of generation means = [P_1, P_2, F_1, F_2, BC_1, BC_2]′
$\underline{X}$ = Coefficient matrix
$\underline{\hat{B}}$ = Vector of parameter = [m d h i j l]′
E = Enov vector

The coefficient matrix is

Generations → ↓		Parameters [m]	[d]	[h]	[i]	[j]	[l]	← ↓
	P_1	1	1.0		1.00			
	P_2	1	–1.0		1.00			
	F_1	1		1.0			1.00	
	F_2	1		0.5			0.25	
	BC_1	1	–0.5	0.5	0.25	–0.25	0.25	
	BC_2	1	–0.5	0.5	0.25	–0.25	0.25	
→								←

Estimates of genetic parameters were derived from the equation

$$\underline{\hat{B}} = (X^T W^{-1} X)^{-1}(X^T W^{-1})Y$$

Where X^T = Transpose of X
$\underline{\hat{B}}$ = Vector of estimates of parameter
$\hat{W}$ = Diagonal matrix for weights
= diag[s^2_{P1}, S^2_{P2}, S^2_{F1}, S^2_{BC1},$S^2_{BC2}S^2_{F2}$]
W^{-1} = Inverse of weight matrix, W
Suitability of the six parameter model which included i, j, l, digenic interaction terms, in addition to m, d, h was judged by its R^2 value and by the model-associated F-statistic, which indicates whether a statistically significant relationship exists between the genetic effects and the genetic means. Significance of estimates of genetic parameters was tested by t-test. Step-wise regression was followed in an attempt to obtain the best possible regression for the given set of response and explanatory variables.

Similarly, estimates of genotype × environment interactions were calculated by weighted least square regression analysis, as described above, on the two years pooled data of three crosses.

Family means and frequency distributions for FGS and TGS were calculated using GENSTAT 5 (GENSAT 5, 1993). Segregation ratios for crosses and families were obtained from frequency distributions by classifying scores of 1–4 as resistant and above 4 as susceptible.

Results and discussion

The genotypes and crosses studied were evaluated for both field grade (FGS) and threshed grade (TGS) scores. However, the results obtained were essentially the same for these two measures of grain mould reaction. Accordingly, only results for FGS are presented and discussed.

The means of P_1, P_2, F_1, BC_1, BC_2, and F_2 families of different crosses made during 1995 and 1996 are shown in Table 2. The grain mould susceptible parent lines (P_1) had high FGS (7.37–9.03) and the resistant parents (P_2) had consistently low FGS (2.20–3.73), confirming their classifications by Audilakshmi et al. (1999). The two susceptible (S_1 and S_5) and two resistant (R_1 and R_2) lines showed similar FGS scores in 1995 and 1996. The F_1 and F_2 means of all the cross/season combinations tended towards the resistant parent, implying dominance of resistance to grain mould.

Generation mean analysis was conducted on a set of transformed square root data. Although this transformation produced a change in the scale of the observations, it did not seem to affect the interpretation of the data. Estimates of genetic effects on the original scale were comparable with those produced on the transformed scale and are presented in Table 2.

The χ^2 values in some crosses were high indicating that the model was not a good fit. Similar results were reported by Torres et al. (1993); they chose the regression analysis method as the most adequate test for generations derived from common parents; and discarded the χ^2 proposed by Mather & Jinks (1971) as the addition of F_2, F_3, and other generation mean values inflates the χ^2 value. We followed the same procedure to judge the goodness of fit. The R^2 values obtained in all crosses were very high (>97%), indicating adequacy of the models for FGS.

Significant additive effects were found in all cross/season combinations. Significant negative dominance effects of similar magnitude to additive effects were observed in only five of ten cross/season combinations, although the parental and F_1 means indicated dominance in all cases. Some genes may have similar but contrasting dominance effects which cancel each other (Mansur et al., 1993). Nelson (1984) observed the expression of additive gene effects by a number of dominant genes when put together. Esele et al. (1993) also reported that genes that individually conferred dominant grain mould resistance showed additive effects when present together.

Table 2. Means of the families and estimates of gene effects for field grade score in susceptible × resistant crosses of sorghum during 1995 and 1996

Means of the families

Cross		$S_2 \times R_1$	$S_6 \times R_1$	$S_2 \times R_2$	$S_3 \times R_2$	$S_1 \times R_1$		$S_1 \times R_2$		$S_5 \times R_1$	
Year		1995	1995	1995	1995	1995	1996	1995	1996	1995	1996
P_1	M	7.37	8.47	8.50	8.13	8.67	8.63	8.33	8.73	9.03	8.77
	SE	0.19	0.13	0.09	0.20	0.12	0.14	0.11	0.11	0.06	0.20
P_2	M	2.20	2.40	2.60	2.73	2.57	3.57	2.87	3.93	2.63	3.73
	SE	0.09	0.10	0.12	0.11	0.10	0.11	0.16	0.21	0.16	0.18
F_1	M	2.90	3.40	3.63	4.03	3.00	4.13	3.70	4.70	4.10	5.00
	SE	0.15	0.22	0.15	0.21	0.10	0.12	0.14	0.15	0.38	0.16
BC_1	M	5.22	6.71	5.18	6.64	6.16	4.53	6.80	5.09	6.04	5.59
	SE	0.30	0.27	0.31	0.27	0.29	0.13	0.31	0.15	0.30	0.14
BC_2	M	2.89	2.78	3.64	4.04	3.69	4.40	3.02	4.58	3.24	4.58
	SE	0.20	0.14	0.15	0.20	0.26	0.13	0.13	0.11	0.19	0.11
F_2	M	3.63	4.78	4.37	6.04	4.60	4.75	4.72	4.72	4.69	4.99
	SE	0.11	0.13	0.12	0.14	0.12	0.09	0.14	0.06	0.13	0.07

Estimates of gene effects

Cross	$S_2 \times R_1$	$S_6 \times R_1$	$S_2 \times R_2$	$S_3 \times R_2$	$S_1 \times R_1$		$S_1 \times R_2$		$S_5 \times R_1$	
Year	1995	1995	1995	1995	1995	1996	1995	1996	1995	1996
[m]	4.73*	5.44*	5.51*	6.56*	5.20*	6.10*	5.86*	4.66*	5.83*	4.93*
[d]	2.53*	3.03*	2.96*	2.69*	3.03*	2.53*	2.98*	2.46*	3.19*	2.51*
[h]	–1.88*	–0.64	–2.02*	–	–	4.01*	–2.14*	–	2.12*	–
[i]	–	–	–	–1.16*	0.42*	–	–	1.58*	–	1.24*
[j]	–	1.85*	–2.62*	–	–	4.79*	1.25*	–3.70*	–	–2.90*
[l]	–	–1.39*	–	–2.56*	2.20*	2.04*	–	–	–	
R^2	98.36	99.99	99.60	98.69	99.54	97.20	99.89	98.52	99.84	97.48

M = Mean, SE = Standard error, * Significant at $p = 0.05$, [m] = mean, [d] = additive gene action, [h] = dominance gene action, [d] = additive × additive gene interactions, [j] = additive × dominance gene interactions, [l] = dominance × dominance gene interactions.

All interaction gene effects (additive × additive, additive × dominance, and dominance × dominance) were inconsistent over crosses and seasons. However, significant additive × additive interactions were observed in those crosses where dominance effects were absent. Murty & House (1984) and Kataria et al. (1990) reported large dominance effects besides significant additive and additive × additive interaction effects for grain mould resistance. In other studies, additive gene action was predominant in the inheritance of resistance (Narayana & Prasad, 1983) and both additive and nonadditive components of variance determined the expression of mould reaction (Dabholkar & Baghel, 1983).

Estimates of G × E effects for the three crosses ($S_1 \times R_1$, $S_1 \times R_2$, $S_5 \times R_1$), tested during 1995 and 1996, are shown in Table 3. Both additive and dominance effects were significant and of approximately equal magnitude. Significant environmental effects were present in only one cross, but were small in

Table 3. Estimates of G × E effects for field grade score in sorghum during 1995 and 1996

Cross	$S_1 \times R_1$	$S_1 \times R_2$	$S_5 \times R_1$
[m]	5.75*	5.87*	5.83*
[d]	2.52*	2.68*	2.57*
[h]	–2.28*	–2.08*	–1.72*
[e]	–	–0.17*	–
[exd]	0.52*	0.53*	0.62*
[exh]	–0.33*	–	–0.40*
R^2	84.7	89.0	97.0

*Significant at $p = 0.05$, [e] = environment effect, [exd] = environment × additive gene interaction, [exh] = environment × dominance gene interaction.

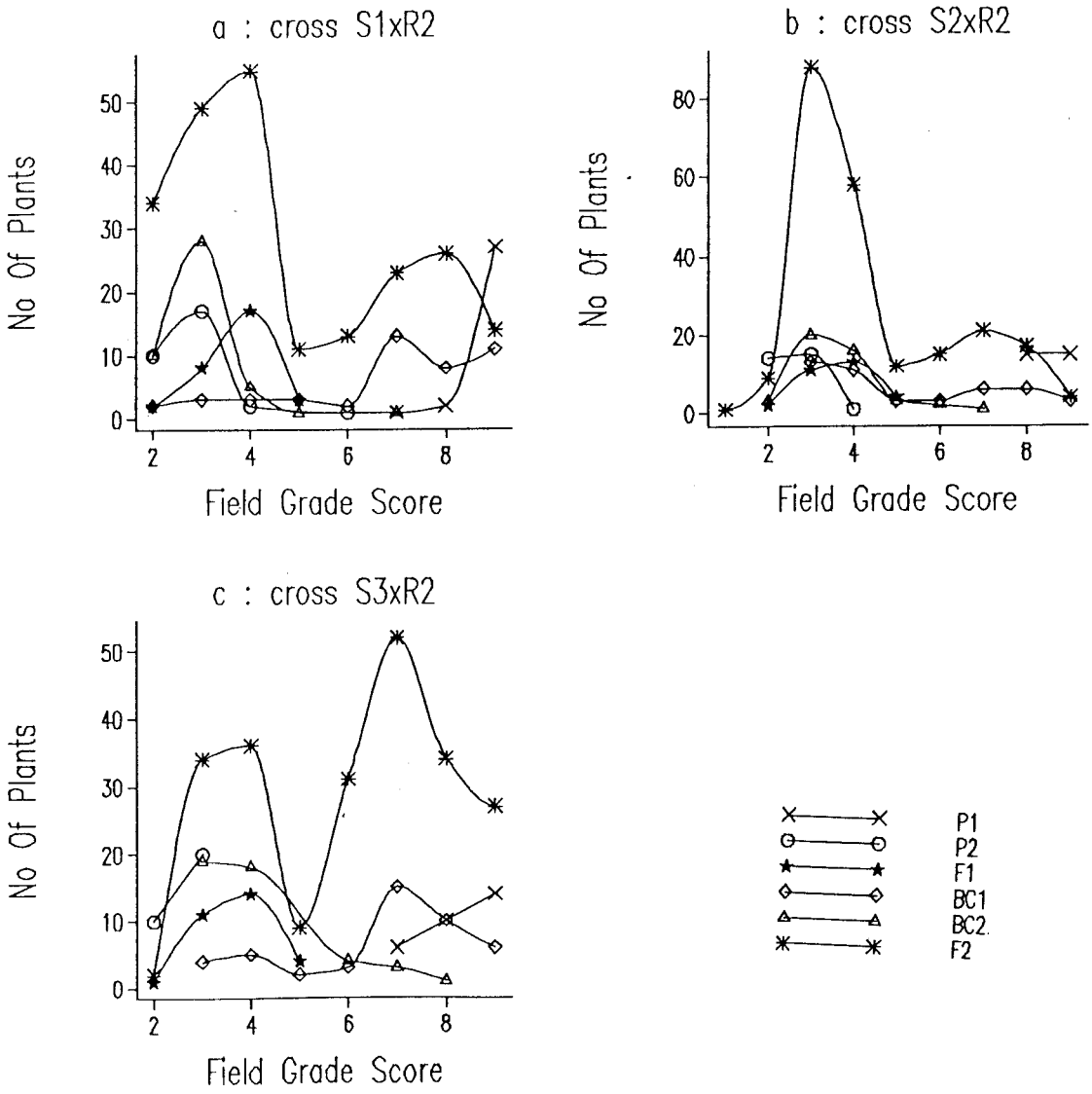

Figure 1. Frequency distributions of P_1, P_2, F_1, F_2, BC_1 and BC_2 families in different crosses of sorghum for FGS (1995).

magnitude. Significant environment × additive effects and environment × dominance effects were present in three and two crosses, respectively, but were also small in magnitude.

Frequency distributions for FGS of P_1, P_2, F_1, BC_1, BC_2, and F_2 families of the seven crosses, grown during 1995, are depicted in Figures 1 & 2. The frequency distributions of the parents and F_1s generally confirmed the patterns shown by family means, with the F_1 mode closer to the value of the resistant parent, confirming that resistance is generally dominant. The F_2 distributions were, in general, bimodal with modes in the 2–4 and 6–8 FGS ranges. The relative magnitude of the modes in the bimodal distributions varied in different crosses. The BC_1 and BC_2 distributions were skewed towards the recurrent parents but continued to overlap, implying epistatic interactions between genes. From these observations, we infer that grain mould reaction is controlled primarily by major genes which differed in different crosses.

Resistant parents R_1 and R_2 in crosses with different susceptible genotypes showed varied F_2 distributions (Figures 1 & 2), indicating that different

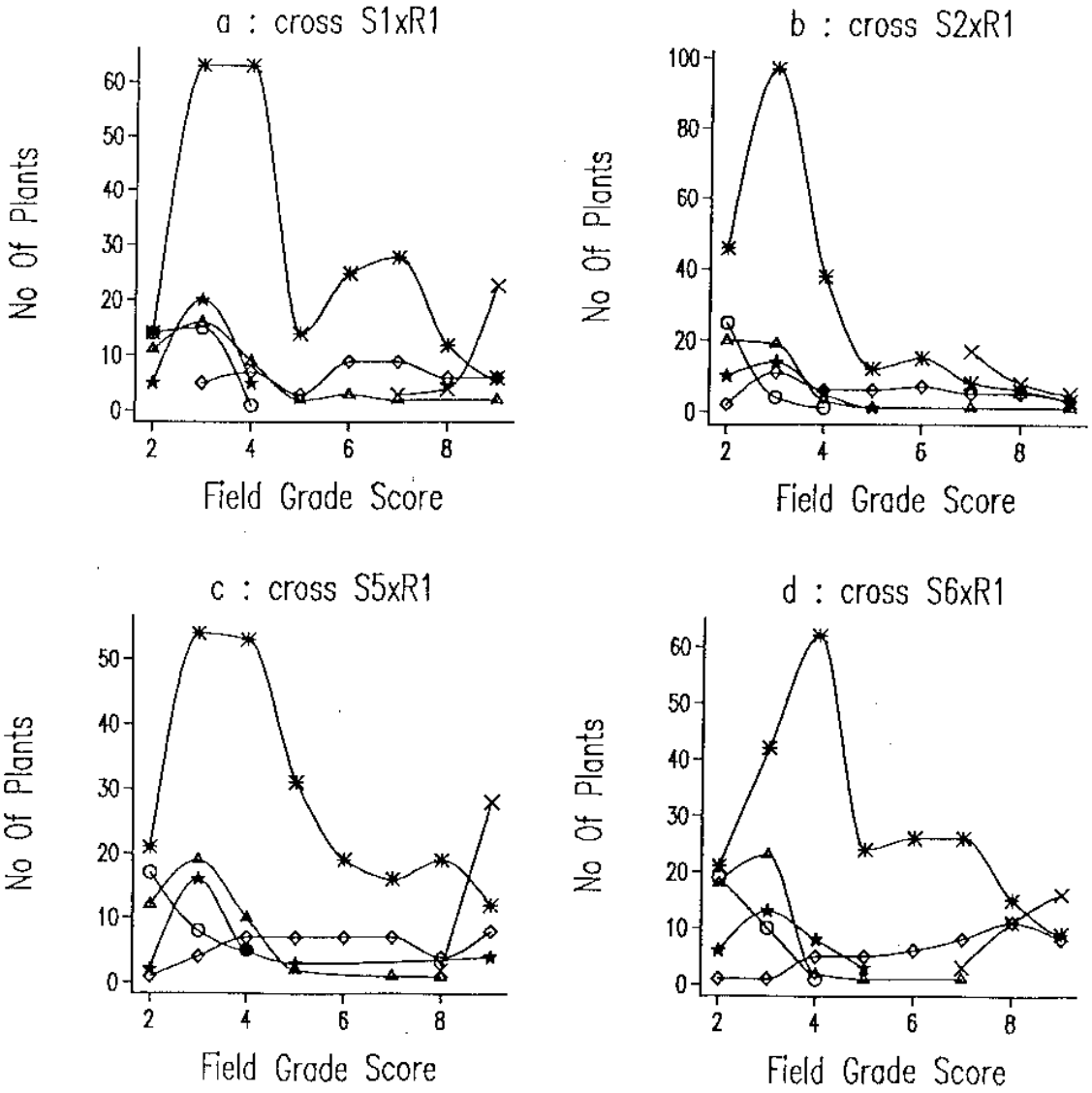

Figure 2. Frequency distributions of P_1, P_2, F_1, F_2, BC_1 and BC_2 families in different crosses of sorghum for FGS (1995). Data points symbols are as for Figure 1.

susceptible lines contributed different major genes as well as modifier genes.

Since most of the frequency distributions in different crosses showed strong peaks between FGS 2 and 4, plants showing FGS values of 1 to 4 (1 to 15% moulded seed) were classified as resistant and remaining plants with FGS values of 5 to 9 were classified as susceptible. Estimates of gene number and mode of inheritance were determined from the segregation patterns of F_2 and BC_1 populations in 1995, and the results are summarised in Table 4.

Of four crosses of the same red resistant line, IS 14375 (R_1), with different white susceptible lines, the observed F_2 segregation patterns in three crosses, $S_1 \times R_1$, $S_5 \times R_1$ and $S_6 \times R_1$, showed good fit to 9 resistant (R):7 susceptible (S) ratios. The BC_1 populations of these same crosses segregated into 1R:3S ratios. The F_2 (9R:7S) and BC_1 (1R:3S) segregation ratios indicate complementary epistasis; two independently assorting dominant genes are complementing with each other in determining grain mould resistance. Similar results were reported by Esele et al. (1993) in a differ-

Table 4. Segregation ratios for field grade score of F_2 and backcross populations derived from crosses of susceptible × resistant genotypes

Cross	Generation	Resistant genotype (R)	Susceptible genotype (S)	Expected ratio	χ^2	Probability
$S_1 \times R_1$	F_2	140	85	9:7	3.23	0.10–0.05
	BC_1	12	33	1:3	0.07	0.80–0.70
$S_5 \times R_1$	F_2	128	97	9:7	0.04	0.90–0.80
	BC_1	12	33	1:3	0.07	0.60–0.70
$S_6 \times R_1$	F_2	125	100	9:7	0.05	0.90–0.80
	BC_1	7	38	1:3	2.13	0.20–0.10
$S_2 \times R_1$	F_2	181	44	51:13	0.08	0.80–0.70
	BC_1	19	26	1:1	1.08	0.30–0.20
$S_1 \times R_2$	F_2	137	87	39:25	0.02	0.90–0.80
	BC_1	8	37	1:3	1.24	0.30–0.20
$S_2 \times R_2$	F_2	156	69	45:19	0.10	0.80–0.70
	BC_1	24	21	1:1	0.20	0.70–0.80
$S_3 \times R_2$	F_2	82	143	27:37	3.08	0.10–0.05
	BC_1	9	36	1:7	3.35	0.10–0.05

R = Resistant, S = Susceptible.

ent cross between red resistant and white susceptible parents; they suggested interaction between a pericarp gene and an intensifier gene for grain mould resistance. These results amply indicate that grain mould resistance in the red resistant (R_1) line is governed by two nonallelic genes.

In the fourth white susceptible (S_2) × red resistant (R_1) cross, the F_2 (51R:13S) and BC_1 (1R:1S) segregation ratios suggest interaction among three nonallelic dominant genes. Apparently, the resistant parent (R_1) has two major dominant genes that act in an additive fashion in determining grain mould resistance, while the susceptible parent (S_2) contributes a dominant epistatic gene that inhibits the expression of only one of the two dominant genes of the resistant parent.

In two crosses of brown resistant line IS 14387 (R_2) by different white grained susceptible lines, we observed modified trigenic ratios with varied gene interactions (Table 4). In $S_1 \times R_2$ (F_2 ratio 39R:25S), a basic dominant gene and two other nonallelic genes, one inhibitory and one anti-inhibitory in action, are implicated. In $S_2 \times R_2$ (F_2 ratio 45R:19S), the combined action of a basic dominant gene in conjunction with two duplicate complementary genes seems to determine grain mould reaction.

In a further cross involving the same brown resistant line ($S_3 \times R_2$), the observed F_2 segregation of 27R:37S implicates three nonallelic major genes acting in a complementary fashion. Esele et al. (1993) reported similar observations in a brown resistant × white susceptible cross.

From the segregation patterns observed in different crosses, it may be seen that two to three nonallelic dominant genes are governing grain mould resistance. One or two major dominant genes from the resistant parent and one dominant gene contributed by the susceptible parent interact to produce grain mould resistance. The dominant gene from the susceptible parent is different in different susceptibles or is modified to behave differently in different crosses. Similar results were reported by Shivanna et al. (1994) in a coloured resistant × white susceptible cross. They observed that grain mould resistance was governed by four genes, two genes with complementary interaction and the other two with additive effects. They further reported that resistance was due to four alleles, interacting additively, at two loci.

Means, estimates of gene effects, frequency distributions and segregation patterns reveal that grain mould resistance in coloured grain types is governed by dominant genes. Furthermore, major genes with epistatic interactions are involved. In the present study, IS 14375 (R_1), a red resistant genotype and IS 14387 (R_2), a brown resistant genotype, exhibited consistent resistance over two years. IS 14375 has been shown to have coloured glumes, high flavan-4-ols and high phenols in seed and glumes, extensive glume cover and hard seed, traits found to show strong as-

sociation with grain mould resistance (Audilakshmi, 1997). Similarly, IS 14387 had coloured glumes, high seed phenols, glume cover and hard seed. As such, these lines may be utilised as gene sources in breeding programmes aimed at stable and long lasting resistance to grain moulds, particularly where sorghum with coloured grain is acceptable.

Genes contributed by different susceptible parents might cause positive or negative effects on grain mould resistance in the segregating progenies. Accordingly, the choice of parents in breeding for grain mould resistance is critical. In this study, two white grained susceptible parents, MS 422B and SP 33316, showed positive contributions to grain mould resistance in their progenies.

Crossing between resistant or moderately resistant lines endowed with different resistance mechanisms is likely to be a successful line of attack in this case. Breeders of hybrids with grain mould resistance can take advantage of the dominance of resistance and dispersal of favorable genes among diverse parents to permit moderately resistant parent lines to produce more highly resistant hybrids. Although fixable additive gene effects are present in almost all the crosses, the presence of dominance and complementary epistasis would decrease the effectiveness of selection in early generations. Selection for grain mould resistance would be more effective if dominance and epistasis effects were reduced by a few generations of selfing but this would result in delay, possible loss of resistant genotypes, and reduce the efficiency of the breeding process.

Alternatively, mapping of the genes through biotechnological methods would be a highly effective approach for a genetically complex character such as grain mould resistance. Hopefully with mapping, the role of specific resistance loci can be identified, race specificity of partial resistance genes can be assessed, and interactions among resistance genes, plant development and environment can be analysed.

Acknowledgements

We gratefully acknowledge the support with the statistical analysis given by Dr Subash Chandra and Ms K Rukimini Devi of Statistics Unit, ICRISAT.

References

Anonymous, 1976. Development of improved high yielding sorghum cultivars with disease and insect resistance. In: Second annual progress report, pp. ii & 193. Texas A&M University, College Station, Texas, USA.

Audilakshmi, S., 1997. Genetics of grain-mould resistance and yield components in sorghum (*Sorghum bicolor*) varieties. PhD thesis, submitted to Osmania University, Hyderabad, India.

Audilakshmi, S., J.W. Stenhouse, T.P. Reddy & M.V.R. Prasad, 1999. Grain mould resistance and associated characters of sorghum genotypes. Euphytica 107: 91–103.

Bandyopadhyay, R. & L.K. Mughogho, 1988. Evaluation of field screening techniques for resistance to sorghum grain moulds. Plant Disease 72: 500–503.

Bandyopadhyay, R., L.K. Mughogho & K.E.P. Rao, 1988. Sources of resistance to sorghum grain moulds. Plant Disease 72: 504–508.

Castor, L.L. & R.A. Frederiksen, 1980. Fusarium and Curvularia grain molds in Texas. In: R.J. Williams, R.A. Frederiksen, L.K. Mughogho & G.D. Bengtson (Eds.), Sorghum Diseases, a World Review, pp. 93–102. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.

Cavalli, L.L., 1952. An analysis of linkage in quantitative inheritance. In: E.C.R. Reeves & C.H. Waddington (Eds.), Quantitative Genetics, pp. 135–144. HMSO, London.

Dabholkar, A.R. & S.S. Baghel, 1983. Diallel analysis of grain mould resistance in sorghum. Genetica Agraria 37: 327–334.

Esele, J.P., R.A. Frederiksen & F.R. Miller, 1993. The association of genes controlling caryopsis traits with grain mould reststance in sorghum. Phytopathology 83: 490–495.

Forbes, G.A., R. Bandyopadhyay & G. Gracia, 1992. A review of sorghum grain mould. In: W.A.J. de Milliano, R.A. Frederiksen & G.D. Bengtson (Eds.), Sorghum and Millets Diseases: A Second World Review, pp 253–264. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.

GENSTAT 5, 1993. GENSTAT 5 Committee of the Statistics Department, Rothamsted Experimental Station, AFRC Institute of Arable Crops Research, Harpenden, Hertfordshire AL5 2JQ, UK.

Hayman, B.I., 1958. The separation of epistatic from additive and dominance variation in generation means. Heredity 12: 371–390.

ICRISAT, 1992. ICRISAT medium term plan 1994–98. Volume 3. Research theme datasets. Patancheru, A.P. 502 324, India: International Crops Research Institute for the Semi-Arid Tropics, 229 pp.

ICRISAT & FAO, 1996. World Sorghum and millet economics: Facts, trends and outlook. Patancheru, A.P. 502324, India: International Crops Research Institute for the Semi-Arid Tropics and Viale delle Terme di Caracalla, 00100 Rome, Italy: Food and Agriculture Organisation of the United Nations. 68 pp.

Kataria, S.K., R. Singh & P.K. Shrotria, 1990. Inheritance of resistance to grain mould fungi in three sorghum, *Sorghum bicolor* crosses. Environment and Ecology 8: 1111–1113.

Mansur, L.M., A.L. Carriquiry & A.P. Rao-Arelli, 1993. Generation mean analysis of resistance to race 3 of soyabean cyst. Crop Science 33: 1249–1253.

Mather, K. & J.L. Jinks, 1971. Biometrical Genetics. Cornell University Press, Ithaca, NY.

Mather, K. & J.L. Jinks, 1977. Introduction to Biometrical Genetics. Cornell University Press, Cambridge, Great Britain.

Murty, D.S., K.N. Rao & L.R. House, 1980. Breeding for grain mold resistant sorghums at ICRISAT. In: R.J. Williams, R.A. Frederiksen, L.K. Mughogho & G.D. Bengtson (Eds.), Sorghum

Diseases, a World Review, pp. 154–163. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.

Murty, D.S. & L.R House, 1984. Components of generation means for resistance to grain mould-causing fungi *Curvularia* and *Fusarium* in sorghum. Cereal Research Cornmunications 12: 237–244.

Narayana, D. & M.N. Prasad, 1983. Inheritance of resistance to *Fusarium* grain mould in sorghum. In: Abstracts of contributed papers, Part 2, of the Fifteenth International Congress of Genetics, 12–21 Dec. 1983, p. 727. New Delhi, India Oxford and IBH Publishing Co.

Nelson, R.R., 1984. Strategy of breeding for disease resistance. In: P.B. Vose and S.G. Blixt (Eds.), Crop Breeding: A Contemporary Basis, pp. 32–50. Oxford, Pergamon.

Shivanna, H., R. Parameswarappa, S.S. Patil & K.H. Anahosur, 1994. Inheritance of grain mould resistance in sorghum. Journal of Maharashtra Agricutural University 19: 255.

Torres, A.M., M.T. Moreno & J.I. Cubero, 1993. Genetics of six components of autofertility in *Vicia faba*. Plant Breeding 110: 220–228.

Williams, R.J. & K.N. Rao, 1981. A review of sorghum grain moulds. Tropical Pest Management 27: 200–211.