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# Genetic basis of resistance to zonate leaf spot disease in forage sorghum

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**Summary.** Generation mean analysis was carried out for ten crosses between two resistant and two susceptible parents to find the genetic basic of resistance to zonate leaf spot disease in forage sorghum. In all crosses except one, at least one type of non-allelic interaction was present. Both additive and dominance gene effects were significant for most crosses. Duplicate type epistasis was present for the inheritance of this disease. Resistance to this disease revealed overdominance. Appropriate breeding plans were suggested to exploit the disease resistance.

**Key words:** Zonate leaf spot – *Gloeocercospora sorghi* – Foliar disease – Alternate breeding

## Introduction

Sorghum is the major fodder crop of northern India and is grown during summer and kharif seasons. Almost all forage sorghum varieties under cultivation in India have been found to be quite susceptible to various kinds of red leaf spot diseases. Zonate leaf spots caused by *Gloeocercospora sorghi* is among the most serious diseases of forage sorghum that cause considerable reduction in the yield as well as quality of this crop. No efforts have been made to improve resistance with regard to any kind of foliar diseases in forage sorghum. It is essential to understand the genetic basis of resistance to any kind of disease to formulate an effective breeding programme. Accordingly, the purpose of the present study was to estimate the gene effects responsible for governing resistance against zonate leaf spots in forage sorghum.

## Materials and methods

The experimental material consisted of two susceptible (PC-1 and JS263) and two resistant (S171 and *Sorghum roxburghii*) parents, ten crosses among these four parents and their $F_2$ generations, ten backcrosses with the first parent ($B_1$) and ten backcrosses with the second parent ($B_2$) of each cross. All material, namely 4 parents, 10 $F_1$'s, 10 $F_2$'s, and 20 backcrosses, was grown in the experimental research area of forage section during kharif, 1980, in a randomized block design comprising three replications. Backcrosses and $F_1$'s were grown in a single row plot, whereas $F_2$'s and parents were grown in 12 rows and 3 rows, respectively, of 4 meters each, at a distance of 30 cm apart. To create more chances for the disease to spread, after every row of experimental material there was one row of each susceptible parent, except in the $F_2$ generation in which these susceptible parental lines were grwon after 6 rows of each $F_2$. The artificial inoculum prepared from most infected lowest 3–4 leaves of growing forage sorghum was also sprayed after irrigating the field at the 25 day and 35 day stages of crop growth to supplement the natural infection.

The data were recorded for zonate leaf spots on each leaf of 120 plants in $F_2$'s and each leaf of 10 plants in the rest of the generations when the fungal infection was between 70 to 80 days of crop growth on the basis of symptoms given by Williams et al. (1978). Scoring of each leaf was done according to the modified disease rating scale of Scherff (1973).

The infection index was calculated according to Wheeler (1969) as Infection Index = sum of individual rating × 100/No. of leaves assessed × number of rating.

The data in percentage were subjected to angular transformation for the final statistical analysis. The scaling tests of Mather (1949) and Hayman and Mather (1955), the joint scaling test of Cavalli (1952) and generation mean analysis of Hayman (1958) and Jinks and Jones (1958) were applied for genetic analysis.

## Results and discussion

Highly significant variation for zonate leaf spots reaction among different generations of various crosses was observed in the present material (Grewal et al. 1986). Such differences among generation means are accounted in terms of estimation of additive and dominance gene effects and non-allelic interactions through generation

**Table 1.** Scaling tests (Mather 1949; Hayman and Mather 1955) for zonate leaf spot (*Gloeocercospora sorghi*) disease incidence for the ten crosses

| Sr. no. | Cross | A | B | C | D |
|---|---|---|---|---|---|
| 1 | *S. roxburghii* × S171 | −2.23* ±0.66 | −2.09** ±1.06 | 0.36 ±1.44 | 2.34* ±0.48 |
| 2 | JS263 × PC-1 | −0.43 ±1.48 | 1.57 ±1.77 | −8.43* ±1.88 | −4.78* ±1.09 |
| 3 | *S. roxburghii* × JS263 | −3.36** ±1.60 | −3.67** ±1.79 | −9.63* ±1.86 | −1.29 ±1.18 |
| 4 | *S. roxburghii* × PC-1 | −1.63 ±1.59 | −0.70 ±1.70 | −7.04* ±2.05 | 4.69* ±1.11 |
| 5 | S171 × JS263 | −2.65 ±2.01 | −3.28 ±1.80 | −11.26* ±2.24 | −2.66** +1.28 |
| 6 | S171 × PC-1 | 0.73 ±1.89 | 6.22* ±1.56 | 5.23** ±2.05 | −0.86 ±1.19 |
| 7 | JS263 × *S. roxburghii* | −5.85* ±1.29 | −3.85 ±2.18 | −17.77* ±1.64 | −4.03* ±1.31 |
| 8 | JS263 × S171 | −5.62* ±1.41 | −6.47* ±1.81 | −11.90* ±1.85 | 0.09 ±1.22 |
| 9 | PC-1 × *S. roxburghii* | 5.51* ±1.46 | 1.13 ±1.92 | −2.70 ±1.72 | −4.67* ±1.24 |
| 10 | PC-1 × S171 | 2.87 ±1.67 | −0.08 ±1.87 | −5.65* ±1.83 | −4.22* ±1.23 |

* Significant at the 1% level
** Significant at the 5% level

mean analysis. Scaling tests of Mather (1949) and Hayman and Mather (1955) indicated the presence of non-allelic interaction in all the crosses, (Table 1) as one or the other scale was significant. The significance of Chi-square values of Cavalli's (1952) three parameter model confirmed the presence of such non-allelic interactions (Table 2) and indicated that this model was inadequate to estimate gene effects. The estimates of additive and dominance gene effects are always biased in the presence of epistasis. Accordingly, to know the nature of epistasis and to estimate gene effects without bias data were analysed through six parameter models as suggested by Hayman (1958) as well as Jinks and Jones (1958).

It was interesting to note that in the cross *S. roxburghii* × JS263, which revealed the presence of non-allelic interactions through scaling and joint scaling tests, epistasis was absent when data were analysed through six-parameter models (Tables 3 and 4). Such a situation may arise due to the presence of high genotype × environment interactions because the estimates of gene effects were not biased by linkage since inter-allelic interactions were not involved. Thus, for this cross where epistasis was absent, Cavalli's model (1952), which revealed the significance of additive as well as dominance gene effects, would be considered fit.

The results of six parameter models revealed that for the cross between resistant × resistant parents, i.e. *S. roxburghii* × S171, only dominance gene effect was significant; whereas for the cross between susceptible × susceptible parents, i.e. JS263 × PC-I, only additive gene effect was significant. However, in both the cases additive × additive and dominance × dominance types of epistasis were present. The two models differed in the case of a cross between susceptible × susceptible parents, as the Jinks and Jones (1958) model revealed that both additive as well as dominance gene effects were important for this cross. Two models also differed for the cross JS263 × *S. roxburghii*, but in the reverse way, in revealing the result of gene effects. Such discrepancies may be attributed, in part, to differences in the expectations of these parameters in the two models and to the heterogeneity of the variances of different generations used in this study. The results obtained using the Jinks and Jones (1958) model contradicted the Hayman (1958) model with regard to mean (m) values of the two crosses PC-1 × *S. roxburghii* and PC-1 × S171, with negative mean values in the former method that should not be theoretically so. However, unexpected results may be due to sampling error. Moreover, these negative values would not affect the overall estimation of results as they were non-significant.

All the crosses between resistant × susceptible and susceptible × resistant parents, except S171 × JS263, S171 × PC-1 and JS263 × S171 in which only additive gene effects with either 'i' or 'j' or 'l' type of epistasis was

**Table 2.** Estimates of joint scaling test (Cavalli 1952) for the ten crosses for zonate leaf spot (*Gloeocercospora sorghi*) disease incidence

| Sr. no. | Cross | m | d | h | $\chi^2$ |
|---|---|---|---|---|---|
| 1 | *S. roxburghii* × S171 | 1.08 ±0.29 | −0.25 ±0.30 | 0.42 ±0.59 | 8.45* |
| 2 | JS263 × PC-1 | 16.86 ±0.37 | 5.61* ±0.38 | −7.10* ±0.73 | 31.45* |
| 3 | *S. roxburghii* × JS263 | 11.22 ±0.32 | −10.99* ±0.33 | −5.92* ±0.65 | 26.92* |
| 4 | *S. roxburghii* × PC-1 | 6.62 ±0.33 | −5.81* ±0.33 | 1.35 ±0.70 | 23.53* |
| 5 | S171 × JS263 | 11.76 ±0.37 | −10.65* ±0.38 | −2.48* ±0.79 | 25.90* |
| 6 | S171 × PC-1 | 7.41 ±0.37 | −5.57* ±0.38 | 2.38* ±0.75 | 16.95* |
| 7 | JS263 × *S. roxburghii* | 10.22 ±0.30 | 10.51* ±0.32 | 1.11 ±0.58 | 117.34* |
| 8 | JS263 × S171 | 11.08 ±0.35 | 10.58* ±0.37 | 0.03 ±0.63 | 49.63* |
| 9 | PC-1 × *S. roxburghii* | 6.42 ±0.31 | 5.99* ±0.33 | 2.67* ±0.60 | 24.84* |
| 10 | PC-1 × S171 | 6.42 ±0.38 | 5.28* ±0.38 | 2.27* ±0.70 | 19.78* |

* Significant at the 1% level

**Table 3.** Estimates of gene effects for zonate leaf spot (*Gloeocercospora sorghi*) disease incidence in the ten crosses using the six parameter model of Hayman (1958)

| Sr. no. | Cross | m | d | h | i | j | l |
|---|---|---|---|---|---|---|---|
| 1 | *S. roxburghii* × S171 | 1.48 ±0.16 | −0.62 ±0.36 | −4.34* ±1.16 | −4.69* ±0.96 | −0.07 ±0.49 | 9.03* ±2.03 |
| 2 | JS263 × PC-1 | 12.41 ±0.25 | 4.64* ±0.96 | 3.50 ±2.32 | 9.57* ±1.18 | −1.00 ±1.05 | −10.71** ±4.29 |
| 3 | *S. roxburghii* × JS263 | 7.43 ±0.27 | −11.11* ±1.05 | −1.57 ±2.49 | 2.59 ±2.37 | 0.15 ±1.10 | 4.44 ±4.60 |
| 4 | *S. roxburghii* × PC-1 | 8.16 ±0.27 | −6.08* ±0.96 | −9.15* ±2.39 | −9.38* ±2.22 | −0.46 ±1.03 | 11.72* ±4.37 |
| 5 | S171 × JS263 | 9.64 ±0.29 | −10.39* ±1.13 | 5.27 ±2.73 | 5.32** ±2.56 | 0.31 ±1.20 | 0.60 ±5.07 |
| 6 | S171 × PC-1 | 8.82 ±0.29 | −7.81* ±1.03 | 3.07 ±2.53 | 1.73 ±2.39 | −2.74** ±1.11 | −8.69 ±4.62 |
| 7 | JS263 × *S. roxburghii* | 8.60 ±0.29 | 10.26* ±1.17 | 10.30* ±2.66 | 8.07* ±2.62 | −0.99 ±1.22 | 1.63 ±4.97 |
| 8 | JS263 × S171 | 9.81 ±0.33 | 11.13* ±1.01 | 0.42 ±2.52 | −0.19 ±1.09 | 0.42 ±1.09 | 12.29* ±4.47 |
| 9 | PC-1 × *S. roxburghii* | 7.05 ±0.29 | 7.81* ±1.69 | 12.22* ±2.55 | 9.34* ±2.41 | 2.18 ±1.15 | −15.99* ±4.70 |
| 10 | PC-1 × S171 | 6.28 ±0.27 | 6.55* ±1.10 | 11.23* ±2.56 | 8.44* ±2.46 | 1.48 ±1.18 | −11.22** ±4.78 |

* Significant at the 1% level
** Significant at the 5% level

**Table 4.** Estimates of gene effects for zonata leaf spot (*Gloeocercospora sorghi*) disease incidence in the ten crosses using the six parameter model of Jinks and Jones (1958)

| Sr. no. | Cross | m | d | h | i | j | l |
|---|---|---|---|---|---|---|---|
| 1 | *S. roxburghii* × S171 | 5.91 ±2.24 | −0.55 ±0.34 | −13.37** ±6.61 | −4.69** ±2.22 | −0.14 ±2.23 | 9.03** ±4.48 |
| 2 | JS263 × PC-1 | 7.98 ±2.22 | 5.64* ±0.42 | 14.22** ±6.30 | 9.57* ±2.18 | −2.00 ±2.10 | −10.71** ±4.29 |
| 3 | *S. roxburghii* × JS263 | 9.33 ±2.40 | −11.26* ±0.35 | −6.02 ±6.80 | 2.59 ±2.37 | 0.30 ±2.21 | 4.44 ±4.60 |
| 4 | *S. roxburghii* × PC-1 | 15.67 ±2.25 | −5.62* ±0.36 | −20.87* ±6.34 | −9.38* ±2.22 | −0.92 ±2.06 | −11.72* ±4.37 |
| 5 | S171 × JS263 | 7.15 ±2.60 | −10.71* ±0.40 | 4.66 ±7.38 | 5.32** ±2.56 | 0.63 ±2.41 | 0.60 ±5.07 |
| 6 | S171 × PC-1 | 5.11 ±2.43 | −5.07* ±0.41 | 11.76 ±6.81 | 1.73 ±2.39 | −5.49** ±2.23 | −8.69 ±4.62 |
| 7 | JS263 × *S. roxburghii* | 3.85 ±1.84 | 11.26* ±0.35 | 8.67 ±7.51 | 8.07* ±2.62 | −1.99 ±2.45 | 1.63 ±4.97 |
| 8 | JS263 × S171 | 12.67 ±2.47 | 10.71* ±0.40 | −11.86 ±6.81 | −0.19 ±2.44 | 0.84 ±2.19 | 12.29* ±4.47 |
| 9 | PC-1 × *S. roxburghii* | −3.05 ±2.50 | 5.62* ±0.36 | 28.21* ±7.06 | 9.34* ±2.48 | 4.37 ±2.30 | −15.99* ±4.70 |
| 10 | PC-1 × S171 | −1.59 ±2.49 | 5.07* ±0.41 | 22.46* ±7.11 | 8.44* ±2.46 | 2.96 ±2.36 | −11.22* ±4.78 |

* Significant at the 1% level
** Significant at the 5% level

significant, revealed the significance of both additive × additive and dominance × dominance interactions. Magnitude of dominance gene effects was also higher in such crosses. Variation in the significance of genetical parameters in reciprocal crosses may be ascribed to sampling error.

Comparisons of signs (negative or positive) of the dominance gene effects (h) and dominance × dominance interaction (l) parameters in crosses where both these parameters were significant revealed duplicate types of gene interactions (as the signs of these two parameters were opposite), confirming the importance of dominance gene effects along with additive gene effects in the inheritance of zonate leaf spot disease resistance. Rana et al. (1982), investigating the inheritance of sorghum downy mildew resistance, also reported the presence of a duplicate type of interaction. The potence ratio [h]/[d], where both 'h' and 'd' parameters were significant, revealed the degree of dominance to be over-dominance, indicating more importance of dominance gene effects with regard to this disease resistance.

In such situations the most suitable breeding plan would be one that mops up the additive gene effects and at the same time maintains appropriate heterozygosity for harnessing the interaction effects. An alternate breeding approach and a system of recurrent selection gives maximum opportunity for rearrangement of genes and can raise the genetic ceiling of the concerned population by accumulating favourable additive genes through intercrossing the selects, and hence could prove to be the most appropriate. Reciprocal recurrent selection seems to be more effective in utilizing both additive and dominant gene effects and theoretical considerations indicate that the presence of non-allelic interactions would favour reciprocal recurrent selection as compared with recurrent selection for general combining ability. For the crosses with duplicate types of epistasis associated with significant additive gene effects, pedigree and backcross breeding would be helpful to accumulate the required resistance.

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