Theor Appl Genet (1991) 82:399–404

THEORETICAL AND APPLIED GENETICS

Genetics of leaf blight resistance in wheat

B. Sinha*, R.M. Singh and U.P. Singh**

Department of Genetics and Plant Breeding, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi-221 005, India

Received September 8, 1990; Accepted March 7, 1991
Communicated by A.R. Hallauer

Summary. Studies on the genetics of leaf blight caused by *Alternaria triticina* using generation mean analysis revealed that additive components played a major role, but that dominance components also contributed significantly in controlling the variability for leaf blight resistance in wheat crosses. Furthermore, the additive × additive type of epistasis was predominant in the first three crosses, whereas in the fourth cross additive × dominance (*j*) and dominance × dominance (*l*) components of epistasis were most significant. Because of this it may be desirable to follow a simple recurrent selection scheme for higher tolerance, to isolate resistant plants from the segregating populations derived from crosses of parents of diverse origin following the pedigree method of breeding. CPAN-1887 was very tolerant to leaf blight in the present study and should be utilized in hybridization programs to develop leaf-blight-resistant varieties.

Key words: Leaf blight – Joint scaling test – Generation mean analysis – Epistasis – Additive gene action

Introduction

Leaf blight, caused by *Alternaria triticina* Prasada and Prabhu, has assumed alarming proportions, especially in the northeastern and eastern regions of India. Frisullo (1982) reported *A. triticina* on durum wheats in southern Italy as well. It has been observed that rust-resistant Mexican dwarf varieties are highly susceptible to leaf blight.

According to Sokhi and Joshi (1974a), maximum yield reduction (35%) was observed when the top two or three leaves were badly damaged, whereas infection on lower leaves was of little consequence. Garg et al. (1972) subsequently recorded the existence of six races; however, they did not notice any major differences in morphological and cultural behavior, which observation was also supported by Jain and Prabhu (1974) using monosporial cultures. Furthermore, Prabhu and Swaminathan (1968) showed that if the content of nonreducing sugars was low, the disease incidence was high and vice versa.

The disease appears as small, oval, discolored lesions, irregularly scattered on the leaves. Subsequently the lesions enlarge, become irregular in shape, and change from brown to grey. Later, several lesions coalesce to cause blight, resulting in the death of the entire leaf. The first sign of infection always appears on the lower leaves, gradually spreading to the upper leaves. Leaf sheath, ear, glumes, and awns are also affected in severe cases of infection. Usually the disease appears when the plants are about 7–8 weeks old, and severely affected plants have small spikes with only a few unfilled grains. The pathogen survives on plant debris in the field (Prabhu and Prasada 1966) and the spores that are produced infect the lower leaves first. Kumar and Arya (1973) suggested that the principal mode of perpetuation of *A. triticina* is through conidia present on the seed surface or dormant mycelium inside the seed coat. The contamination of seed with fungus occurs in the field. Thus, observations suggest that the pathogen perpetuates via seed as well as through plant debris in the soil (Raut et al. 1983; Sharma et al. 1983).

The genetics of resistance to this disease has been studied in intervarietal crosses, viz., NP835 × NP830, NP852 × NP830, C306 × NP830, where NP830 was the susceptible parent while the others were resistant (Narula

* Present address: Directorate of Extension Education, R.A.U. Pusa, Samastipur – 848125, Bihar, India
** To whom offprints should be addressed

and Srivastava 1971). All the F_1s were as susceptible as NP830, the susceptible parent. F_2 results suggested that the resistance was controlled by two pairs of recessive genes. Kulshrestha and Rao (1976) studied the inheritance of *A. triticina* resistance in a diallel study involving ten wheat cultivars. They reported that susceptibility of NP830 was controlled by a dominant gene and that of NP852, C281, E5477, E5550, E5878, and UP303 by the recessive alleles of the genes present in NP830 and NP891.

In order to avoid recurring losses in wheat yield due to incidence of diseases like *Alternaria* leaf blight, it is necessary to develop resistant varieties. An understanding of the type and complexity of gene action controlling this reaction is a pre requisite of planning effective breeding program, whose aim is to breed varieties with durable resistance.

In the present study, a series of diverse lines having leaf blight resistance was used to produce F_1, F_2, and backcross generations, which were then analyzed for gene effects controlling the leaf blight reactions.

Materials and methods

One moderately resistant line of spring wheat to leaf blight complex, CPAN-1887, one moderately susceptible line, HUWL-39, and two susceptible lines, CPAN-1922 and Sonalika, were selected from the National Genetic Stock Nursery of the All India Coordinated Wheat Improvement Project. The parentages of these lines are given in Table 1.

The following lines were crossed in 1985–86 to study the inheritance of adult plant leaf blight resistance: CPAN-1887 × CPAN-1922, CPAN-1887 × HUWL-39, CPAN-1887 × Sonalika, and HUWL-39 × Sonalika. The F_1s and parents were raised in the 1986–87 winter season to produce new F_2s and backcrosses with both the parents.

Field assessment of the four crosses and their different generations was carried out in "blight nurseries" at the Agricultural Research Farm, Banaras Hindu University. The material was planted on November 27, 1987. Each cross to be investigated, consisting of the two parental lines, populations of F_1, F_2, BC_1 and BC_2, was sown in plots of 1 × 2.50 m in a randomized complete block design with three replications. Each parental line, its F_1 as well as BC_1 and BC_2, had a single-row plot with 20 plants spaced at 5 cm within and 25 cm between rows. Each F_2 had six rows of 20 plants each.

Spreader rows of a highly susceptible variety, NP 830, were planted every 11 rows and also around the entire experimental field on three separate dates to create a local epidemic.

The inoculum obtained from a single isolate of a race, Kanpur, was multiplied on wheat straw following the procedure of Sokhi and Joshi (1972). A thick conidial suspension prepared in water from the inoculum collected from the wheat straw medium was used for field inoculation. The concentration of spores was 1,000 spores/ml water. When the plants in the test plot were at the boot leaf stage, they were sprayed with a spore suspension by a Spraymate plastic atomizer, in the evening, after which the field was heavily irrigated.

Plants were scored three times on the basis of disease intensity; however, the last scoring, done later in the crop growth, was taken for data analysis when the disease intensity had reached its maximum expression. Every plant in each plot was scored on the basis of a scale developed in India for appraising foliar intensity of wheat diseases. This 0–9 scale (Joshi et al. (1982) ranks the infection types on the basis of disease intensity of the entire plant. The range of the scale was reduced to a minimum of 6–9, as none of the plants from the three replications in each of the four crosses could be given a score of 5 or less. There were no clear-cut susceptible and resistant classes and the observations reflected a continuous variation in reaction type; thus, data were analyzed by averaging the ratings of each plant per plot to get a mean plot rating, using the simple scaling test proposed by Mather (1949) and the joint scaling tests of Cavalli (1952) and Hayman and Mather (1955). The Bartlett's test was applied to test the homogeneity of error variances. Since the calculated Chi-square (5 *df*) was found to be highly significant, the hypothesis of homogeneous variances was rejected, thus suggesting a weighted analysis of the data.

Table 1. Pedigree and source of the parents

Parents	Pedigree	Source
CPAN-1887	Timgalin/Revm/2/Skemer W58 44-6-M	CIMMYT, Mexico
CPAN-1922	Ore F1 158/Fd1/Mexifen-S/ 2* Tib 63/3/Coc 75	CIMMYT, Mexico
HUWL-39	Kavkaz-K.4500. LAU, Coc 75 SWO 176	Varanasi, India
Sonalika	(II-54-388-An) × (Yt54XNIOB) L.Rojo.	Pantnagar, India

The standard procedure consisted of estimating the parameters – *m* (mean), (*d*) additive gene effect, (*h*) dominance gene effect, (*i*) additive × additive type of gene interaction, (*j*) additive × dominance type of gene interaction, and (*l*) dominance × dominance type of gene interaction – from means of the available types of generations, followed by a comparison of the observed generation means with expected values derived from the estimates of the six parameters. However, as only six generations were available in each of the four crosses, there was perfect agreement between the observed family means and those expected; therefore, the goodness of fit test of the six parameter model could not be conducted. Nevertheless, the estimates of the six parameters were obtained by using weighted least squares, taking weights as the reciprocals of the squared standard errors of each mean (Mather and Jinks 1982).

After examining the estimates of the six parameters, those parameter(s) that were not found to be significant were deleted, using the sequential model fitting scheme as described by Mather and Jinks (1982).

Results

Bartlett's test for homogeneity of error variances of different generations revealed highly significant χ^2 values, suggesting that error variances are heterogeneous for reaction to leaf blight pathogen (Table 2). Thus, the weighted least square technique was used for the analysis of gene action.

Following the results of the homogeneity test, the simple scaling test of Mather (1949) was applied to determine the presence of epistasis in different wheat crosses

Table 2. Bartlett's test for homogeneity of error variances of different generations of four wheat crosses for their reactions to *Alternaria* leaf blight of wheat

Crosses	Chi-square	Error variance	Analysis suited
CPAN-1887 × CPAN-1992	265.5	Heterogenous	Weighted
CPAN-1887 × HUWL-39	161.91	Heterogenous	Weighted
CPAN-1887 × Sonalika	309.28	Heterogenous	Weighted
HUWL-39 × Sonalika	70.46	Heterogenous	Weighted

Table 3. Scaling test for four wheat crosses for their reactions to *Alternaria* leaf blight of wheat

Crosses	Scale A	Scale B	Scale C	Scale D
CPAN-1887 × CPN-1992	0.37 +0.27 (1.37)	−0.22 +0.18 (1.20)	−0.83** +0.28 (2.97)	−0.49* +0.19 (2.49)
CPAN-1887 × HUWL-39	−0.23 +0.25 (0.93)	−1.39** +0.21 (0.68)	1.78** +0.38 (4.68)	1.71** +0.17 (9.82)
CPAN-1887 × Sonalika	−0.63 +0.49 (1.28)	0.68** +0.25 (2.67)	−0.96** +0.31 (3.15)	−0.51 +0.30 (0.31)
HUWL-39	−0.28 +0.32 (0.72)	1.44** +0.27 (5.34)	1.27** +0.28 (4.48)	0.04 +0.21 (0.18)

Values after '+' sign are SE and may be positive or negative
Values in parentheses are *t*-statistic
* Denotes significant and ** highly significant

Table 4. Joint scaling test for gene effects under three parameters [*m*, (*d*), (*h*)]. Model of four wheat crosses for their reactions to leaf blight

Crosses	Mean *m*	Estimates of additive effect (*d*)	Estimates dominance effect (*h*)	Chi-square *df*=3
CPAN-1887 × CPAN-1992	7.94** +0.03 (254.51)	1.02** +0.03 (32.37)	−0.47** +0.06 (7.56)	12.78
CPAN-1887 × HUWL-39	6.83** +0.05 (129.29)	0.22** +0.05 (4.39)	−0.49** +0.12 (4.23)	145.65
CPAN-1887 × Sonalika	7.70** +0.03 (257.39)	1.25** +0.03 (41.17)	−0.09 +0.05 (1.59)	19.71
HUWL-39 × Sonalika	7.37** +0.05 (154.23)	0.88** +0.05 (17.54)	0.38** +0.01 (3.94)	42.28

Values after '+' sign are SE and may positive or negative
Values in parentheses are *t*-statistic
** Denotes highly significant

Table 5. Estimates of gene effects for reactions to *Alternaria* leaf blight of wheat for the cross CPAN-1887 × CPAN-1922 using six- and three-parameter models

Parameter	Estimates	
	Six-parameter model	Three-parameter model
Mean *m*	**6.98+0.39 (17.70)	**7.56+0.04 (197.96)
Additive effect (*d*)	**1.02+0.03 (31.19)	**1.01+0.03 (31.49)
Dominance effect (*h*)	1.67+1.05 (1.60)	– –
Adtv. adtv. inter. (*i*)	*0.97+0.39 (2.47)	**0.41+0.05 (8.08)
Adtv. dom. inter. (*j*)	−0.59+0.31 (1.87)	– –
Dom. dom. inter. (*l*)	−1.12+0.67 (1.66)	– –
Chi-square	–	4.71
Prob	–	0.2–0.1

Values after '+' sign are SE and may positive or negative
Values in parentheses are *t*-statistic
* Denotes significant and ** highly significant

(Table 3). Perusal of data in Table 3 indicates that out of four scales (A, B, C, and D), at least two were significant in each of the four crosses studied, suggesting the involvement of either one or two of the three [(*i*), (*j*), and (*l*)] kinds of epistasis studied.

The joint scaling test to verify the adequacy of the three parameter models *m*, (*d*), and (*h*) revealed a lack of good fit in each of the four crosses analyzed (Table 4), strongly suggesting the presence of epistasis.

Based on the findings of the test, for epistasis, the six-parameter model was fitted to the observed means of families in each of the four crosses. The results are presented in Tables 5–8 and are described below for each cross.

CROSS I: CPAN-1887 × CPAN-1922

In this cross, analysis under the six-parameter model suggested that, of the six parameters, three components – *m*, (*d*), and (*i*) – were significant (Table 5). Removal of nonsignificant components like (*h*), (*j*), and (*l*) from the six-parameter model revealed that the standard error of all three components was considerably reduced and χ^2 (3) gave a good fit to the observed means of families (Table 5).

CROSS II: CPAN-1887 × HUWL-39

All six parameters were found to be significant in this cross (Table 6). The relative magnitude of the (*h*) compo-

Table 6. Estimates of gene effects for the cross CPAN-1887 × HUWL-39 using six-parameter model for reactions to *Alternaria* leaf blight of wheat

Parameters	Estimates
Mean *m*	**10.31 + 0.35 (29.25)
Additive effect (*d*)	**−0.41 + 0.06 (7.07)
Dominance effect (*h*)	**−8.94 + 0.91 (9.86)
Adtv. adtv. inter. (*i*)	**−3.41 + 0.35 (9.79)
Adtv. dom. inter. (*j*)	**−1.16 + 0.27 (4.34)
Dom. dom. inter. (*l*)	** 5.03 + 0.62 (8.18)
Chi-square	–
Prob.	–

Test of adequacy is not possible as no degrees of freedom are left
Values after '+' sign are SE and may be positive or negative
Values in parentheses are *t*-statistic
* Denotes significant and ** highly significant

Table 7. Estimates of gene effects for reactions to *Alternaria* leaf blight of wheat for the cross CPAN-1887 × Sonalika using six- and five-parameter model

Parameter	Estimates	
	Six-parameter model	Five-parameter model
Mean *m*	**6.71 + 0.62 (10.90)	**7.27 + 0.15 (49.61)
Additive effect (*d*)	**1.24 + 0.03 (40.23)	**1.23 + 0.03 (40.23)
Dominance effect (*h*)	2.00 + 1.73 (1.15)	*0.38 + 0.17 (2.26)
Adtv. adtv. inter. (*i*)	1.01 + 0.61 (1.65)	**0.45 + 0.15 (3.02)
Adtv. dom. inter. (*j*)	*1.30 + 0.55 (2.38)	**1.59 + 0.45 (3.53)
Dom. dom. inter (*l*)	−1.06 + 1.13 (0.94)	– –
Chi-square	–	0.87
Prob.	–	0.50 − 0.03

Chi-square values suggest that the model may be adequate
Values after '+' sign are SE and may be positive or negative
Values in parentheses are *t*-statistic
* Denotes significant and ** highly significant

Table 8. Estimates of gene effects for reactions to *Alternaria* leaf blight of wheat for the cross HUWL-39 × Sonalika using six- and five-parameter models

Parameter	Estimates	
	Six-parameter model	Five-parameter model
Mean *m*	**7.34 + 0.44 (16.82)	**7.28 + 0.05 (140.69)
Additive effect (*d*)	**0.82 + 0.05 (15.91)	**0.82 + 0.05 (15.91)
Dominance effect (*h*)	1.26 + 1.24 (1.02)	*1.44 + 0.25 (5.74)
Adtv. adtv. inter. (*i*)	−0.06 + 0.43 (0.15)	– –
Adtv. dom. inter. (*j*)	**1.67 + 0.40 (4.18)	**1.6 + 0.39 (4.21)
Dom. dom. inter (*l*)	−1.15 + 0.83 (1.39)	** −1.26 + 0.27 (4.66)
Chi-square	–	0.02
Prob.	–	0.90 − 0.80

Chi-square values suggest that the model may be adequate
Values after '+' sign are SE and may be positive or negative
Values in parentheses are *t*-statistic
** Denotes highly significant

nent was highest, followed by the (*l*) component, whereas other components were relatively less important for this cross.

CROSS III: CPAN-1887 × Sonalika

Analysis of genetic components of means in this cross suggested that only three components – *m*, (*d*), and (*j*) – were important in controlling the genetic variability of the character in question. The least important component, (*l*), was omitted from the analysis and the remaining five parameters were assessed. Results on the relative magnitude of components with their standard errors revealed that the precision of estimates increased in most cases, i.e., the value of the SE was reduced with a concomitant increase in the magnitude of some components like *m* and (*j*). All five components appeared to play a significant role in the degree of tolerance to *Alternaria* leaf blight in wheat. The $\chi^2(3)$ gave high goodness of fit to the means of the generations (Table 7).

CROSS IV: HUWL-39 × Sonalika

In this cross, three components, – *m*, (*d*), and (*j*) – played predominant role in wheat for the expression of variability of the degree of tolerance to *A. triticina*. Here again, the least important component, (*i*), was omitted from the analysis to obtain the remaining five components and test their significance. The data in Table 8 clearly indicate

that precision improved and the χ^2 test also provided a complete good fit to the means. The relative magnitude of the (*j*) component was highest, followed by (*h*) and (*d*), respectively.

Discussion

In order to analyze the nature of classical epistasis, the signs of the (*h*) and (*l*) components were screened in those crosses where both components were significant, after omission of certain less important parameters. It was noted that in cross II, CPAN-1887 × HUWL-39, and cross IV, HUWL-39 × Sonalika, the (*h*) and (*l*) components possessed opposite signs, thereby suggesting the predominance of a duplicate type of epistasis.

Gene dispersion was also verified by comparing the magnitude of (*h*) and (*d*) and the higher estimates of the (*h*) component over (*d*) in two crosses, i.e., CPAN-1887 × HUWL-39 and HUWL-39 × Sonalika.

If parents used in a cross are in the association phase (genes with increasing effect in one parent and genes with decreasing effect in the other parent), the (*h*) component is always smaller as compared to (*d*); however, if they are in the dispersion phase (genes with increasing and decreasing effects are randomly distributed among parents), the estimates of the (*h*) component are always higher than (*d*) due to the accumulation of dominant parental genes in the hybrid.

From the foregoing presentation, it may be concluded that the additive component plays an important role in controlling the variability for leaf blight resistance in all four crosses of wheat.

Although there was an inconsistency in the estimates of the (*h*) parameter from cross to cross, the significant estimates of dominance effect (*h*) noted in a majority of crosses cannot be overlooked; hence, this component also appears to contribute considerably to the expression of variability for leaf blight resistance in wheat. In addition to these components, the additive × additive type of epistasis (*i*) appears to play a considerable role in controlling the variability of this character, at least in the first three crosses of wheat. However, in the cross IV (HUWL-39 × Sonalika), the additive × dominance (*j*) and dominance × dominance (*l*) components of epistasis emerged as more important.

Simple inheritance of resistance to leaf blight of wheat was studied in intervarietal crosses (Narula and Srivastava 1971; Sokhi and Joshi 1974b; Kulshrestha and Rao 1976; Srivastava et al. 1981). Results indicated the involvement of two pairs of recessive genes for resistance. Present findings, using biometrical procedures, clearly illustrate the significance of additive and additive × additive gene effects in controlling the expression of variability for leaf blight resistance in wheat. However, the importance of the dominance component in a majority of the crosses has also been noted.

Based on the results of gene action analysis in these crosses of wheat, it is suggested that simple recurrent selection or pedigree schemes for higher tolerance may be effectively employed to isolate resistant plants from the population.

It is evident from the analysis of gene action in all four crosses for reaction to this wheat pathogen that this character is predominantly under the control of additive gene action; hence, tolerant lines can be isolated from the segregating populations derived from the crosses in which CPAN-1887 is involved as a donor parent. The idea behind the inclusion of CPAN-1887 in crosses is also supported by the fact that this line expresses less disease than others.

Acknowledgements. The authors express appreciation to K.D. Rai of the Computer Center. Banaras Hindu University, for developing computer programs of the models used in this study. Appreciation is also expressed to Dr. C. M. Pandey for statistical advice.

References

Cavalli LL (1952) An analysis of linkage in quantitative inheritance. In: Reeve ECR, Waddington CH (eds) Quantitative inheritance. HMSO, London, pp 135–144

Frisullo S (1982) Fungal parasites of plants in Southern Italy. 1. *Alternaria triticina* Prabhu and Prasada on durum wheat. Phytopathol Mediterr. 21:113–115

Garg VR, Arya HC, Kant U (1972) Varietal reaction and physiologic specialization in *Alternaria triticina* causing leaf blight of wheat in Rajasthan. Indian J Mycol Plant Pathol 2:24–31

Hayman BI, Mather K (1955) The description of gene interaction in continuous variation. Biometrics 11:69–82

Jain KL, Prabhu AS (1974) Comparative studies on variation in some monoconidial isolates of *Alternaria triticina*. Indian J Mycol Plant Pathol 4:67–73

Joshi LM, Singh DV, Srivastava KD (1982) Current techniques in wheat pathology. In: Division of mycology and plant pathology. Multiplication of inoculum. IARI, New Delhi, pp 111–115

Kulshrestha VP, Rao MV (1976) Genetics of resistance to an isolate of *Alternaria triticina* causing leaf blight of wheat. Euphytica 25:769–775

Kumar VR, Arya HC (1973) Certain aspects of perpetuation and recurrence of leaf blight of wheat in Rajasthan. Indian J Mycol Plant Pathol 3:93–94

Mather K (1949) Biometrical genetics, 1st edn. Methuen, London, pp 71–72

Mather K, Jinks JL (1982) Biometrical genetics, 3rd edn. Chapman and Hall, London, pp 92–93

Narula PN, Srivastava OP (1971) Genetics of *Alternaria* resistance in wheat. Indian J Genet 31:105–107

Prabhu AS, Prasada R (1966) Pathological and epidemiological studies on leaf blight of wheat caused by *Alternaria triticina*. Indian Phytopathol 19:95–112

Prabhu AS, Swaminathan MS (1968) Inverse relationship between resistance to rusts and leaf blight in wheat. Curr Sci 13:379–380

Raut JG, Guldhe SM, Wangikar PD (1983) Seed-borne infection of *Alternaria triticina* in wheat and its control. Indian Phytopathol 36(2): 274–277

Sharma SC, Randhawa HS, Sharma HL (1983) Seed infection in relation to the susceptibility of wheat to *Alternaria triticina* and *Cochliobolus sativus*. Indian Phytopathol 36(2): 372–374

Sokhi SS, Joshi LM (1972) Multiplication of inoculum of *Alternaria triticina* for field inoculations. Indian J Microbiol 12: 108–109

Sokhi SS, Joshi LM (1974a) Estimation of losses in yield due to leaf blight disease of wheat caused by *Alternaira triticina*. Indian J Mycol Plant Pahtol 4: 29–33

Sokhi SS, Joshi LM (1974b) Inheritance of *Alternaria* resistance in wheat. Indian J Genet 34: 457–459

Srivastava OP, Singh RA, Rao MV (1981) Note on genetics of seedling resistance to *Alternaria* leaf blight of wheat. Indian J Agric Sci 51: 810–811