Indian J. Genet., 70(1): 11-16 (2010)

Genetic analysis of resistance to spot blotch (*Bipolaris sorokiniana*) in wheat

Hanif Khan[1], S. M. S. Tomar and S. Chowdhury

Division of Genetics, Indian Agricultural Research Institute, New Delhi 110 012

(Received: January 2009; Revised: November 2009; Accepted: December 2009)

Abstract

Diallel analysis of spot blotch resistance was laid out involving six resistant and two susceptible genotypes of spring wheat of diverse origin in order to evaluate their general combining ability (GCA) and specific combining ability (SCA). The parents chosen showed wide variation for area under disease progress curve (AUDPC) of spot blotch. GCA and SCA effects were statistically significant for AUDPC score suggesting that additive as well as non-additive genetic mechanisms were involved in the expression of resistance in these parents. Wheat genotypes Chirya-3, Shanghai-4, Suzhoe 128-OY, Suzhoe 1-58, Longmai and Cuanmai#18 had significantly negative GCA effects for AUDPC in F_1 generations, suggesting their prime suitability for use in wheat breeding programs to improve resistance to spot blotch. The estimate of narrow-sense heritability was 0.69 whereas broad-sense heritability was 0.92 in F_1s. The results indicated predominance of additive gene action in the inheritance of spot blotch resistance in spring wheat.

Key words: Combining ability, diallel, spot blotch, resistance, wheat, AUDPC

Introduction

Spot blotch [*Bipolaris sorokiniana* (Sacc.) Shoem] in India and South-East Asia was not considered to be of major importance until after Green Revolution [1]. During and after Green Revolution (1970's) the change in agronomic practices such as increased use of fertilizers, irrigation, and continuous cultivation of Mexican wheat on large areas made wheat populations more vulnerable to spot blotch [2], which became an increasingly important problem in wheat growing areas. Crop intensification with rice-wheat cropping system and expansion of wheat cultivation to non-traditional areas has been also cited as the reason for the appearance of disease in epidemic proportions in Indian subcontinent [3, 4]. Yield losses due to spot blotch are variable but can be significant in warmer wheat growing areas. Although spot blotch has assumed status of number one disease in the eastern parts of India, more recently it has also expanded into the cooler, traditional irrigated rice-wheat production areas [5, 6]. It was believed that source of inoculum of *Bipolaris sorokiniana* on wheat in rice-wheat cropping system may be some of the grasses or rice stubbles. However study conducted on source of inoculum and reappearance of spot blotch in rice-wheat cropping system in eastern India by Pandey *et al.* [7] revealed that the infected wheat seeds are the most important source of inoculum as it carried 26% to 86% infection. In tropical regions like India, Bangladesh, Philippines, Bolivia, Brazil, Paraguay and Zambia the disease needs to be given a special attention that include identification of resistant sources, information on inheritance and gene action of resistance to the disease. Resistance to spot blotch in high yielding genotypes is low to moderate [8]. This limitation makes programme difficult and need new strategies for greater spot blotch resistance in wheat genotypes.

The best strategy to address the spot blotch problem is to develop resistant cultivars, although this may take considerable time [4]. However to do so, the genetics of the resistance in improved genotypes must be further understood. Previous studies have reported the resistance to be qualitatively [9, 10], as well as quantitatively inherited [11-13]. The resistance source identified so far fall in the three categories *viz.*, Latin American, Chinese and wild relatives of wheat or alien species. The most Chinese sources are from the Yangtze river basin e.g. Shanghai-4, Suzhoe-8 and Yangmai-6 [14].

---

[1] Present address: Scientist (Plant Breeding), Central Institute for Arid Horticulture, Beechwal, Bikaner 334 006

Information on heritability and response to selection to the disease is needed to make progress in breeding for resistance to this disease. Therefore, the present investigation was undertaken to further enhance the knowledge of mode of inheritance and gene action of spot blotch resistance in wheat.

Material and methods

Six resistant genotypes *viz*., Chirya-3, Shanghai-4, Suzhoe 128-OY, Suzhoe 1-58, Longmai, and Chuanmai #18 and two susceptible genotypes *viz*., Sonalika, HD 2329 were selected for combining ability study based on screening study under natural and artificial epiphytotic condition during 2001-02 and 2002-03 respectively. These eight genotypes were grown and crossed during *Rabi* 2002-03, in a diallel mating fashion excluding reciprocals to produce 28 F_1 populations. The 28 F_1 hybrids along with the eight parents were planted in randomised block design with three replications in the field at Pusa Bihar during 2003-04 in the rows of 2m length, with 25cm row spacing and 10cm distance between seed to seed. Total sixty seeds per cross were sown in three replication where each replication had one row of twenty plants. To provide maximum chance of disease spread during flowering time [15] late sowing was done. The standard agronomic practices were followed while epiphytotic condition was created in the field. While Pusa Bihar is a 'hot spot' for spot blotch of wheat and experimental material were also provided additional inoculum artificially. Spot blotch was induced by artificially inoculating spreader rows of HUW-234 at required intervals in parental lines and F_1 populations. A pure culture of Pusa isolate (locally most aggressive) of *B. sorokiniana* was multiplied on sorghum grains and spores were harvested in water. A spore suspension of 10^4/ml was uniformly sprayed at three stages *viz*., tillering, flag leaf emergence and anthesis during evening hours, following the method of Chaurasia *et al.* [8]. Plots were irrigated immediately after inoculation to maintain a high relative humidity. The disease severity was scored on each plant using double-digit scale (00-99) developed as a modification of Saari and Prescott's severity scale to assess foliar diseases [16, 17] by visually scoring the percent diseased area on the flag (F) and penultimate (F-1) leaves. Three disease readings were recorded at 85, 95 and 105 days after sowing. The area under disease progress curve (AUDPC) was calculated using the percent severity estimates corresponding to the three ratings as outlined by Das *et al.* [18].

$$\text{AUDPC} = \sum^{-} \qquad + \qquad -$$

Where x_i is the disease severity on the i^{th} date, t_i is the i^{th} day; n is the number of scoring dates. The AUDPC measures the amount of the disease as well as the rate of progress, and has no units.

Analysis of variance was conducted for parents and F_1s. Combining ability analyses were conducted according Griffing's [19] Method 2, Model 1. The diallel analyses were done using MSTAT-C computer program. The hypothesis that general combining ability (GCA) estimates of parents and specific combining ability (SCA) estimates of the F_1s equalled zero was tested by a two tailed *t*-test.

Results and discussion

High spot blotch severity occurred in the experimental plots at Pusa Bihar as shown by >90% diseased leaf area on the flag leaves of the susceptible parent HD 2329 and Sonalika. The symptoms of the spot blotch were uniformly visible on all plants in a plot. Isolates of representative samples showed a high incidence of spot blotch pathogen *B. sorokiniana.* The analysis of variance with respect to area under disease progress curve (AUDPC) of spot blotch showed significant differences among the parents and cross combinations and parent vs. F_1s (Table 1). Furthermore, a significant parent vs crosses effects indicates average heterosis for resistance. GCA and SCA effects were significant in F_1 generation, indicating that both additive and non additive genetic mechanism contributed significantly in the inheritance of resistance to the disease.

Table 1. Analysis of variance for 8 parents and 28 F_1s for AUDPC for spot blotch

Source of variation	DF	Mean sum of square
Replication	2	389.10
Parents	7	297615**
F_1	27	71506**
P vs. F_1s	1	168487**
Error	70	1374

** : Significant at 1 per cent

Combining ability analysis

Predominance of additive-genetic effect was reflected

Table 2. Analysis of variance for GCA and SCA effects for AUDPC

Source of variation	DF	Mean sum of square
GCA	7	152962**
SCA	28	11505**
Error	70	1374
GCA/SCA		13.24
SE (g_i)		2.00
SE (S_{ij})		6.14

**: Significant at 1 per cent

Table 3. Variance components of AUDPC for spot blotch

Var. component	Value
σ^2gca	14141
σ^2sca	11505
σ^2e	1374
V_A	28282
V_D	11505
σ^2gca/σ^2sca	1.23
h^2_n	0.69
h^2_b	0.92

Table 4. Parental means and estimates GCA effects of AUDPC of spot blotch from eight parent diallel analysis in wheat

Parent	Mean double-digit score at last observation (105 DAS)	Mean AUDPC	GCA estimates
Sonalika	7,9	801	207**
HD2329	7,9	727	185**
Chirya-3	1,3	47	–121**
Shanghai-4	2,3	87	–65**
Suzhoe 128-OY	2,3	86	–69**
Suzhoe 1-58	2,3	91	.43**
Longmai	2,4	103	.41**
Chuanmai#18	2,4	102	–52**
Mean		255.5	
SE(Gi)			2.00
CD at 5%			3.98
CD at 1%			5.28
SE(Gi-Gj)			3.03

**: Significant at 1 per cent

by the large variance components due to GCA (Table 2) and larger value of V_A than V_D (Table 3). The mean square for GCA was 13 times greater than mean square for SCA. The magnitudes of GCA and SCA mean squares are indicative of relative importance of additive and non-additive gene action in the inheritance of a trait [20]. These findings suggest that additive genetic mechanisms were more important than non-additive ones in controlling resistance to spot blotch. This is in agreement with the results of Sharma *et al.* [21]. Additive variance was found to be larger than non-additive variance and accounted for 69% of total variance in F_1 generation of diallel crosses (Table 3). Non genetic additive variance was estimated to be 28% of the total phenotypic variance in F_1s. The estimate of narrow-sense heritability was 0.69 whereas broad-sense heritability was 0.92 in F_1s. These results again suggest that additive gene actions were primarily responsible for inheritance of resistance to spot blotch in these crosses. This is in agreement with the earlier report by Sharma *et al.* [4]. Therefore, the degree of resistance in progenies appears to be largely predictable from the parent's performance and highly effective in pre-selection of lines for use in a crossing program or in population improvement.

Mean values and the GCA effects of the eight parental genotypes for AUDPC are listed in Table 4. The eight parents varied widely for AUDPC indicating different levels of resistance to spot blotch. The most resistant homozygous line was Chirya-3 followed by Suzhoel28-OY, Shanghai 4, Suzhoel-58, Chuanmai#18 and Longmai. As expected, the two susceptible cultivars, Sonalika and HD 2329 had the highest AUDPC. Similarly, the GCA estimates for the eight parents differed significantly (Table 4). Among the eight parents, six had significantly negative and two had significantly positive GCA estimates. GCA estimates of the parents were mainly associated with mean AUDPC. Six resistant parents had significantly negative GCA estimates from F_1 generations. Their ranking also remained almost same for mean AUDPC and GCA estimates. In general, the GCA estimates indicated that Chirya-3, Suzhoel28-OY, Shanghai 4 and Chuanmai#18 would be most desirable parents to use in hybridization to develop progenies with spot blotch resistance.

Mean AUDPC values for the 28 crosses ranged from 45 to 802 (Table 5) in F_1. Cross involving Chirya-3 as one of the parent always had lower AUDPC than most of the other crosses. Mean AUDPC for eight

Table 5. Cross mean and estimates of SCA effects for AUDPC of spot blotch in eight parent diallel analysis in wheat

No.	Cross name	Mean double-digit score of F_1 (105 DAS)	AUDPC in F_1			
			Mean	SCA	MPH	BPH
1	Sonalika/HD 2329	7,9	802	228.4**	4.9	10.3
2	Sonalika/Chirya-3	1,3	48	-218.4**	-88.6	3.6
3	Sonalika/Shanghai-4	4,5	201	-122.4**	-54.8	129.8
4	Sonalika/Suzhoe 128-OY	3,5	176	-142.5**	-60.2	105.0
5	Sonalika/Suzhoe 1-58	4,6	288	-57.0**	-35.4	216.5
6	Sonalika/Longmai	5,6	308	-38.5**	-31.7	200.3
7	Sonalika/Chuanmai #18	5,6	275	-61.4**	-39.1	169.6
8	HD 2329/Chirya-3	1,3	46	-199.9**	-88.2	-2.1
9	HD 2329/Shanghai-4	4,5	199	-103.8**	-51.4	126.7
10	HD2329/Suzhoe 128-OY	3,5	185	-112.7**	-54.5	115.1
11	HD2329/Suzhoe 1-58	5,6	317	-6.8	-22.5	248.3
12	HD 2329/Longmai	4,6	218	-48.0**	-33.1	170.3
13	HD 2929/Chuannai # 1 8	4,6	208	-107.2**	-49.8	103.9
14	Chirya-3/Shanghai-4	1,3	46	50.9**	-31.3	-1.4
15	Chirya-3/Suzhoel28-OY	1,3	47	57.4**	-27.1	3.6
16	Chirya-3/Suzhoe 1-58	1,3	48	30.6**	-30.7	2.1
17	Chirya-3/Longmai	1,3	45	26.5**	-39.3	-2.9
18	Chirya-3/Chuanmai#18	1,3	45	36.9**	-39.0	-2.8
19	Shanghai-4/Suzhoe128-OY	2,3	85	37.5**	-2.3	-1.5
20	Shanghai-4/Suzhoe1-58	2,3	88	15.0*	-0.9	1.2
21	Shanghai-4/Longmai	2,4	97	20.8**	1.1	9.9
22	Shanghai-4/Chuanmai #18	2,4	95	30.1**	0.2	8.4
23	Suzhoe 128-OY/Suzhoe 1-58	2,3	89	19.5**	1.2	3.1
24	Suzhoe 128-OY/Longmai	2,4	94	23.0**	-0.3	9.3
25	Suzhoe 128OY/Chuanmai # 18	2,4	92	31.8**	-1.8	7.3
26	Suzhoe 1-58/Longmai	2,4	94	2.8	-2.6	3.7
27	Suzhoe 1-58/Chuanmai #18	2,4	97	10.3	-0.5	6.6
28	Longmai/Chuanmai #18	2,4	100	11.52	-2.3	-2.0
	Mean		158.2			
	SE			6.14	25.24	30.13
	CD at 5%			12.22		
	CD at 1%			16.21		
	SE(Sij-Sik)			9.08		
	SE(Sij-Skl)			8.56		

*,**: Significant at 5 per cent 1 per cent

parents was 255.5 (Table 4), which was higher than the mean (158.2) of the 28 F_1s (Table 5). This suggests that dominance is involved in the inheritance of resistance to spot blotch. A similar result was reported by Sharma *et al.* [4], where degree of dominance determined the genetic control of spot blotch resistance in 36 crosses.

Table 5 displays SCA effects and heterosis compared to mid parent and better parent (low AUDPC score). SCA effects were generally highly significant. Out of 28 crosses 11 crosses showed a significantly negative SCA effect, whereas 13 crosses had a significantly positive SCA effect. The hybrids with lowest spot blotch infection and significant SCA effects were Sonalika/Chirya-3, HD 2329/Chirya-3, Chirya-3/Shanghai-4, Chirya-3/Suzhoel28-OY, Chirya-3/Suzhoel-58, Chirya-3/Longmai and Chirya-3/Chuanmai #18. In all these crosses Chirya-3 was common parent which contributed towards higher resistance to spot blotch. Among the crosses between resistant and susceptible parents mid parent heterosis (MPH) for the resistance was a general phenomenon as shown by the high negative values in Table No. 5. A range of F_1s were more resistant than their respective parental means and sometime surpassed even their better parent (Tables 4 & 5). The similar results were reported by Buerstmayr *et al.* [22] in combining ability studies of resistance to head blight in wheat.

Consequences for resistance breeding

The results obtained herein are of relevance for breeding F_1 hybrid and common homozygous cultivars. As the mid parent heterosis for spot blotch resistance seems to be common, an F_1 hybrid involving one resistant parent may express adequate resistance. The best parental combiners could furthermore be crossed to obtain superior homozygous genotypes in their progenies [23]. For line breeding the significant SCA effects also mean that the resistance level of the progenies can not be predicted by the mid-parent mean and hence the chance of selecting improved line depend upon the specific combination of the parents. As such, crosses using parent with higher GCA estimates can be expected to produce superior progenies [4]. In general, the predominance of GCA effects for AUDPC indicates that resistance to spot blotch can be improved through selection. Parents with high level of resistance and significant negative GCA estimates were identified in the present study. On the basis of AUDPC values and GCA estimates Chirya-3, Shanghai-4 and Suzhoel 28-OY are superior sources for spot blotch for use in wheat breeding programs aimed to improve this trait. Hence, genetic improvement of resistance to spot blotch in wheat using simple pedigree procedure could be successful due to high heritability and predominance of additive gene effects.

Acknowledgement

This research in part was conducted with financial support from Council of Scientific and Industrial Research (India) through fellowship to the first author.

References

1. **Saari E. E.** 1998. Leaf blight disease and associated soil-borne pathogens of wheat in South and South East Asia. *In*: Duveiller E., Dubin H. J., Reeves J. and McNab A. (eds.). *Helminthosporium* blights of wheat: spot blotch and tan spot. 37-51 CIMMYT Mexico D.F.
2. **Karwasra S. S., Beniwal M. S. and Rajender Singh.** 1998. Occurrence, cultivar reaction and yield losses due to leaf blight of wheat. Indian Phytopath., **51**: 363-364.
3. **Duveiller E. and Gilchrist L. I.** 1994. Production constraints due to *Bipolaris sorokiniana* in wheat for the warmer areas. *In*: Saunders D. A. and Hettel G. P. (eds.). Wheat in Heat stressed Environments: Irrigated dry areas and rice wheat farming systems. 96-112. CIMMYT Mexico. D.F.
4. **Sharma R. C., Sah S. N. and Duveller E.** 2004. Combining ability analysis of resistance to *Helminthosporium* leaf blight in spring wheat. Euphytica, **136**: 341-348.
5. **Duveiller E.** 2003. Controlling foliar blight of wheat in rice-wheat system of Asia. Pl. Dis., **88**: 52-56.
6. **Singh V. and Singh R. N.** 2007. Management of spot blotch of wheat (*Triticum aestivum*) caused by *Bipolaris sorokiniana.* Indian-Journal-of-Agricultural-Sciences, **77**: 323-326.
7. **Pandey S. P., Kumar S., Kumar U., Chand R. and Joshi A. K.** 2005. Sources of inoculum and reappearance of spot blotch of wheat in rice-wheat cropping systems in eastern India. European J. Pl. Pathol., **111**: 47-55.
8. **Chaurasia S., Joshi A. K., Dhari R. and Chand R.** 1999. Resistance to foliar blight of wheat: A search. Genet. Res. and Crop Evol., **46**: 469-475.
9. **Singh B. N., Singh R. N., Singh A. K. and Singh S. P.** 2000. Inheritance of resistance in wheat to *Cochliobolus sativus* causing spot blotch. Indian Phytopath., **53**: 486-487.
10. **Ragiba M., Prabhu K. V. and Singh R. B.** 2004. Recessive genes controlled resistance to *Helminthsporium* leaf blight in synthetic hexaploid wheat. Plant Breeding, **123**: 389-391.
11. **Kutchner H. R., Bailey K. L., Rossnagel B. G. and Legge W. G.** 1994. Heritability of common root rot and spot blotch resistance in barley. Can. J. Plant. Pathol., **16**: 287-294.

12. **Sharma R. C., Dubin H. J., Devkota R. N. and Bhatta M. R.** 1997. Heritability estimates of field resistance to spot blotch in four spring wheat crosses. Plant Breeding, **116**: 64-68.
13. **Joshi A. K., Kumar S., Chand R. and Orize-Ferrara G.** 2004. Inheritance of resistance to spot blotch caused by *Bipolaris sorokiniana* in spring wheat. Plant Breeding, **123**: 213-219.
14. **Van-Ginkel M. and Rajaram S.** 1998. Breeding for resistance to spot blotch in wheat. Global Perspective. *In*: Duveiller E., Dubin H. J., Reeves J. and Me Nab A. (eds.). *Helminthosporium* Blights of wheat: Spot blotch and Tan spot. 162-170 CIMMYT Mexico, D.F.
15. **Sharma R. C. and Duveiller E.** 2003. Effect of stress on Helminthosporium leaf blight in wheat. *In*: Rasmussen J. B., Friesen T. L. and Ali S. (eds.). Proc. 4th Int. Wheat Tan Spot and Spot Blotch Workshop. 140-144 North Dakota State University, Fargo.
16. **Saari E. E. and Prescott J. M.** 1975. A scale for appraising the foliar intensity of wheat diseases. Plant Dis. Rep., **59**: 377-380.
17. **Anonymous.** 1996. Annual report, Directorate of Wheat Research (DWR), Karnal. Appendices IV & V.
18. **Das M. K., Rajaram S., Mundt C. C. and Kronstad W. E.** 1992. Inheritance of slow rusting resistance to leaf rust in wheat. Crop Sci., **32**: 1452-1456.
19. **Griffing B.** 1956. Concepts of general and specific combining ability in relation to diallel crossing systems. Aust. J. Biol. Sci., **9**: 463-493.
20. **Haussmann B. I. G., Hess D. E., Sissoko I., Kayentao M., Reddy B. V. S., Welz H. G. and Geiger H. H.** 2001. Diallel analysis of sooty stripe resistance in sorghum. Euphytica, **122**: 99-104.
21. **Sharma R. C., Sah S. N., Sanjaya-Gyawali, Duveiller E.** 2003. Genetic control of resistance to *Helminthosporium* leaf blight in wheat. *In*: Rasmussen J. B., Friesen T. L. and Ali S. (eds.). Proc. 4th Int. Wheat Tan Spot and Spot Blotch Workshop. 68-73. North Dakota State University, Fargo.
22. **Buerstmayr H., Lemmens M., Berlakovich S. and Ruckenbauer P.** 1999. Combining ability of resistance to head blight caused by *Fusarium culmorum* (W.G. Smith) in the F_1 of a seven parent diallel of winter wheat (*Triticum aestivum* L.). Euphytica, **110**: 199-206.
23. **Christie B. R. and Shattuck V. I.** 1992. The diallel cross: design, analysis and use for plant breeders. Plant Breeding Reviews, **9**: 9-37.