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Full Length Research Paper

Inheritance of resistance to *septoria tritici* blotch (STB) in some Iranian genotypes of wheat (*Triticum aestivum* L.)

Shahrbanou Vakili Bastam[1], S. Sanaz Ramezanpour[1]*, Hassan Soltanloo[1], Shaban Kia[2], Mehdi Kalate[2], Mohammad Hadi Pahlevani[1]

[1]Plant Breeding and Biotechnology Department, Gorgan University of Agricultural Science and Natural Resources, Gorgan, Iran.
[2]Faculty staff of Gorgan Agricultural research centre, Gorgan, Iran.

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***Septoria tritici* blotch (STB), caused by *Mycosphaerella graminicola* (anamorph *S. tritici*) is currently the most serious foliar disease of wheat worldwide. Understanding of mechanisms for resistance inheritance in genotypes would potentially lead to more efficient deployment of host plant resistance. As part of our effort to improve STB resistance, inheritance of seedling STB resistance was investigated by an eight-parent half diallel set of crosses in Iranian wheat genotypes. Parents and F1 crosses were planted in plastic pot at greenhouse in a randomized complete block design (RCBD) with three replicates. Plants at the second-leaf stage were inoculated with an isolate of *S. tritici*. Infection response and Picnidia density ratings of the first and second leaves and their AUDPC used for diallel analysis. Significant GCA and SCA were observed in the analysis of variance. The ratio of GCA sum of squares relative to SCA sum of squares suggested that GCA was more important than SCA. Additive alleles effects played the major role in host response to STB in studied varieties. Significant values of both D and H components suggested that all traits were under the control of both additive and dominance gene effects. For all traits high narrow and broad sense heritabilities observed. Recessive genes in infection response, iAUDPC, pAUDPC and dominant alleles in Picnidia density led to decreasing level of traits and increasing resistance to STB. Genotypes Line#10 and N-81-18 had high negative GCA effects and should be promising parents in breeding programs for enhancement of STB resistance.**

Key words: *Septoria tritici* blotch, wheat, diallel, GCA, SCA.

INTRODUCTION

Septoria tritici blotch (STB), caused by the ascomycete fungus *Mycosphaerella graminicola* (anamorph *S. tritici*) is currently the most serious foliar disease of wheat in Europe and several other temperate and subtropical regions of the world (Eyal et al., 1987; Polley and Thomas, 1991). It is a major problem in characterized by temperate and wet environment during the growing season (Eyal et al., 1987). In highly susceptible cultivars, this disease may reduce grain yield by 50% (Eyal and Ziv, 1974). STB got epidemic in Golestan province of Iran in 2002 - 2003 and the estimated yield damage reported by Kia et al. (2005) was 7.49 to 24.61%.

Resistance to STB may be isolate-specific or quantitative. Isolate-specific resistance is near-complete, oligogenic (Somasco et al., 1996; Arraiano, 2001; McCartney et al., 2002) and follows a gene-for-gene relationship (Brading et al., 2002), whereas quantitative or partial resistance is incomplete, polygenic (Jlibene et

*Corresponding author. E-mail: ramezanpours@gau.ac.ir. Tel: +989122910164. Fax: +981714420981.

Abbreviation: GCA, General combining ability; **SCA,** Specific combining ability; **AUDPC,** Area under disease progress curve; **iAUDPC,** Infection response area under disease progress curve; **pAUDPC,** Picnidia density area under disease progress curve.

al., 1994; Simon and Cordo, 1998; Zhang et al., 2001) and isolate nonspecific (Chartrain et al., 2004b). Specific interactions between wheat cultivars and *M. graminicola* isolates occur in both seedling tests and under field conditions (Arraiano et al., 2001a, b; Brown et al., 2001; Kema et al., 1996a, b, 1997). This raises the possibility that the specific interactions may operate through a gene-for-gene mechanism (Eyal et al., 1973; Kema et al., 1996a, 2000) in which, for every gene conferring resistance in the host, there is a corresponding gene for avirulence in the pathogen (Flor, 1971).

Resistance to STB controlled by one major gene was identified in some plant materials (Rillo and Caldwell, 1966; Rosielle and Brown, 1979; Wilson, 1979; Lee and Gough, 1984). Resistance to STB based on several genes also was identified (Rosielle and Brown, 1979). In recent years, 12 major genes for resistance to *M. graminicola*, Stb1 to Stb12, have been identified and mapped (Somasco et al., 1996; Arraiano et al., 2001c; Brading et al., 2002; Adhikari et al., 2003, 2004 a,b,c; Chartrain et al., 2004, 2005). Jlibene and Bouami (1995) indicated that several components of the partial resistance to STB also may be combined into the same genetic background by crossing. Several quantitative studies have indicated the presence of general and specific combining ability of resistant to STB (Van Ginkel and Scharen, 1987; Danon and Eyal, 1990; Jlibene et al., 1994; Simon and Cordo, 1997, 1998).

The use of fungicides to control STB is expensive and not entirely reliable. Isolates of *M. graminicola* resistant to strobilurin (Qo inhibitor, QoI) fungicides have become common in Europe and there has been widespread failure of QoI fungicides to control STB (Anonymous, 2003). Resistant cultivars provide an effective and economical way to control the disease. Therefore, Knowledge about genetics of a disease is essential for effective resistance breeding (Arraiano et al., 2007).

A better understanding of the relative importance of general and specific combining abilities (GCA/SCA) and genetic components of resistance to STB would potentially lead to more efficient development of resistant cultivars and deployment of germplasm resources. Therefore, the objective of the present research was to estimate the effects of combining abilities for STB resistance, determining the number of genes involved in resistance to *M. graminicola*, finding the action of resistant genes, evaluating heredity of resistance and other genetic components in several Iranian wheat genotypes exhibiting various levels of STB resistance.

MATERIALS AND METHODS

Eight spring wheat genotypes were selected based on preliminary field and greenhouse observations of their reaction to *S. tritici*. Three out of eight genotypes were line while the rest were cultivar. Line pedigrees and STB infection responses of all genotypes are presented in Table 1. F_1 crosses were obtained by hand emasculation and pollination in the field of Agricultural research center of Gorgan, Golestan in 2008. About 50 hybrid seeds were obtained from each cross combination.

Thirty-six genotypes including parents and F_1 were included in the test. Tajan was used as a susceptible check. Five seeds of each genotype were planted in plastic pots under randomized complete block design with three replications in the greenhouse of Gorgan agricultural research center.

Pathogen production and disease evaluation

One isolate of *S. tritici* originating from field collections of Gorgan was used. For extracting the pathogen, direct method of Eyal, (1999) was followed. At first, pieces of diseased leaves containing Picnidia were sticked on glassy microscope slide with tape. Slides placed on the sterile filter paper in the petri plates and wetted with distilled water. Petri plates moved to incubator for 24 h at 24°C. Conidia of isolate were streaked on PDA media (39 g dextrose agar, 1 L water and 500 mg Coloramephnicle antibiotic) in petri plates with a sterile wire loop. The plates were placed in incubator at 20 ± 2°C. After a week, small pink colonies moved to PDA media without antibiotic and kept in incubator at 20 ± 2°C. When the edge of the pink colony began to darken, the conidia were ready to harvest. Segments of fungi colonies with 1 - 2 cm diagonal, placed in Erlene meyers containing YMS liquid medium and put on shaker with 130 rpm speed and 20°C temperature. After a week, conidial suspension filtered through two layers of cheesecloth and adjusted to approximately 10^6 -10^7 mL^{-1} of conidia as determined by hemacytometer counts.

Plants were inoculated when the second leaf was fully expanded. After inoculation, plants were kept moist by spraying water with atomizer several times a day for 3 days. To avoid losing wet, a plastic cover put on the pots and kept in a dark chamber for 48 h and 20°C temperature and over 80% wet. Then pots moved to greenhouse at the 20°C temperature and over 80% wet condition.

Disease ratings of the first and second leaves were recorded after fourteen days post-inoculation based on 1 to 9 scales (Zhang et al., 2001) for 4 times with 4 interval day. Two traits including infection response (amount of infection response and chlorosis) and density of picnidia (amount of picnidia coverage) were recorded. The results of observations were used to compute area under disease progress curve (AUDPC) for each trait (iAUDPC and pAUDPC). AUDPC was calculated based on Moldovan et al. (2005) according to the following function:

$$\text{AUDPC} = \sum_{i}^{n-1} [(y_i + y_{i+1})/2](t_{i+1} - t_i)$$

in which n is the number of assessment times (minimum 2), y is the disease measurement and t is the time (days) from inoculation.

Infection response and picnidia density of fourth assessment at 30 days after inoculation, while genotype as susceptible check was severely diseased (90% or more of lesions bearing picnidia) and AUDPC of each traits used for analysis.

Data analysis

Average of disease score of first and second leaves were used as data for each trait and also data for each replication was the average of disease score of 5 plants in each pot.

All data were tested for normality using the Kolmogorov-smirnov's test in SPSS software. All data except those belongs to pAUDPC were normal, so they were transformed using arcsine

Table 1. Eight winter wheat parents and their *S. tritici* infection response.

Genotypes	Pedigree	Infection response
Line#10	BOBWHITE#1/FENGKANG	R
N-81-18	MILAN/ SHA7	MR
N-80-19	SW89.3064/STAR	MR
Chamran		MS
Moghan3		MS
Tajan		S
Zagros		S
Koohdasht		S

S = susceptible, MS = moderately susceptible, R = resistant and MR = moderately resistant.

Table 2. Analysis of variance of *S. tritici* blotch scores for eight parents and 28 F_1 crosses. Following Steel and Torrie (1984) and Walters and Morton (1978).

S.O.V	Df	Mean square			
		Infection response	iAUDPC	Picnidia density	pAUDPC
Block	2	0.244	6.022	0.029	0.752
Genotype	35	4.635**	51.671**	2.354**	37.482**
a	7	13.867**	110.782**	5.840**	64.633**
b	28	1.990**	36.898**	1.483**	30.695**
b_1	1	3.474**	4.285	0.122	18.778**
b_2	7	3.347**	7.807**	1.716**	12.582**
b_3	20	1.441**	48.711**	1.470**	37.630**
Error	70	0.258	2.616	0.04	0.395
C.V.		7.783	12.3	10.888	6.59

** = Significant at the 0.01 level of probability.
a = Additive gene effect, b = Dominance gene effect, b_1 = Directional dominance deviation, b_2 = Gene distribution among the parents and b_3 = Effect of specific genes.

Table 3. Mean squares of general/specific combining abilities and their ratio.

S.O.V	Mean square			
	Infection response	iAUDPC	Picnidia density	pAUDPC
GCA	5.553**	79.091**	2.661**	47.627**
SCA	0.431**	1.757*	0.316**	3.711**
Error	0.086	0.87	0.013	0.132
$\frac{2GCA}{2GCA+SCA}$	0.963	0.989	0.944	0.962

* = Significant at the 0.05 level of probability, ** = Significant at the 0.01 level of probability.
GCA = General combining ability, SCA = Specific combining ability.

square root to adjust them to a normal distribution. Data analysis was performed using SAS v9.1, D2 and Diallwin98 genetic software. Graphical trends created by excel software.

RESULTS AND DISCUSSION

Different genotypes showed significant differences for all traits (Table 2). For estimating combining ability effects, method 2, model 1 Griffing (Griffing, 1956) that contains parents and F_1 crosses was used. The analysis of variance for combining ability (Table 3) showed the significant variation for all characters, indicating a wide range of variability among the genotypes. Highly significant variation due to general combining ability (GCA) as

Table 4. General combining ability effects (diagonal values) and specific combining ability effects (above diagonal) for studied characters.

	Chamran	Tajan	Zagros	Moghan3	N-81-18	Koohdasht	N-80-19	Line#10
Infection response								
Chamran	0.285**	-0.77**	-0.078	-0.004	-0.604*	-0.632*	-0.072	0.695**
Tajan		0.579**	-0.635	0.074*	-0.315	-0.051	-0.20	0.609*
Zagros			0.679**	-0.440	0.043	0.015	-1.217**	0.426
Moghan3				0.189*	0.103	-0.119	-0.726**	-0.125
N-81-18					-0.544**	0.141	0.395	0.316
Koohdasht						0.733**	-0.604*	0.663*
N-80-19							-0.576**	0.431
Line#10								-1.344**
iAUDPC								
Chamran	0.518	-1.094	1.661*	0.996	-1.631	-1.153	-0.816	0.746
Tajan		2.027**	-1.800*	0.864	0.699	0.397	1.109	-0.447
Zagros			2.152**	-0.437	1.182	0.754	-1.399	2.521**
Moghan3				0.545**	0.300	-1.032	-2.095*	-0.863
N-81-18					-2.801**	1.087	0.346	0.749
Koohdasht						3.924**	0.503	1.343
N-80-19							-2.055**	0.489
Line#10								-4.312**
Picnidia density								
Chamran	-0.297**	-0.183	0.367**	-0.034	-0.04	0.002	0.327**	0.392**
Tajan		0.914**	-0.066	1.021**	-0.682**	-0.168	-0.968**	-0.277**
Zagros			0.239**	-0.220*	0.16	1.007**	-0.001	0.231**
Moghan3				-0.252**	0.498**	-0.002	-0.385**	-0.529**
N-81-18					-0.618**	0.35**	-0.325**	0.254**
Koohdasht						0.368**	-1.005**	0.393**
N-80-19							0.168**	0.385**
Line#10								-0.522**
pAUDPC								
Chamran	-0.520**	0.358	1.953**	0.982**	-0.651**	1.221**	0.994**	0.340
Tajan		3.268**	0.082	4.498**	-0.433	0.154	-2.846**	-2.539**
Zagros			1.062**	-0.193	0.864**	3.285**	-0.806**	1.713**
Moghan3				-0.859**	-0.322	0.279	-1.531**	-1.313**
N-81-18					-2.802**	2.72**	0.268	1.006**
Koohdasht						2.483**	-2.546**	1.427**
N-80-19							-0.082	1.445**
Line#10								-2.549**

* = Significant at the 0.05 level of probability, ** = Significant at the 0.01 level of probability.

well as specific combining ability (SCA) indi-cated the importance of additive as well as non-additive types of gene action in inheritance of these characters. High value of Baker ratio (Baker, 1978) for all traits, showed the more importance of additive effects than non-additive effects of genes. Same results recorded by Zhang et al. (2001), Van Ginkel and Scharen (1987), Danon and Eyal (1990) and Jlibene et al. (1994).

To select most resistant genotypes, those with less value of all the studied traits are desirable. Thus, negative values of GCA, SCA and heterosis are useful. Between genotypes, Line#10 had the most negative GCA value (Table 4) to reduce infection response and iAUDPC (increasing resistance) and also GCA of N-81-18 genotype was negative and highly significant for two of these traits. For picnidia density and pAUDPC, N-81-18 and Line#10 genotypes had the most negative GCA values, respectively, too. Negative GCA value in Line#10 and N-81-18 indicating that resistance to STB was consistently inherited in crosses with these parents.

The best SCA combination to reduce symptoms of disease and increasing resistance belongs to hybrids between N-80-19 and Zagros, Moghan3, Koohdasht and Tajan for infection response, iAUDPC, picnidia density

Table 5. Estimates of mid parent and better parent heterosis for studied characters.

Crosses	Infection response		iAUDPC		Picnidia density		pAUDPC	
	%MP	%BP	%MP	%BP	%MP	%BP	%MP	%BP
Chamran*Tajan	-14.58**	-17.08**	-14.85	-27.39*	-3.06	-20.56**	8.66	-37.93**
Chamran*Zagros	-9.16	-14.17**	19.58	12.68	9.44**	5.69**	39.35**	29.56**
Chamran*Moghan3	-6.8	-8.33*	1.06	-1.64	2.61	0.94	22.71**	20.21**
Chamran*N-81-18	-9.51	-21.67**	-12.71	-55.96**	2.22	0.55	4.69	-15.55**
Chamran*Koohdasht	-11.46*	-13.75**	-10.01	-36.09**	3.54	-3.75*	4.93	-21.74**
Chamran*N-80-19	-9.37	-16.67**	-16.05	-40.35**	0.42	-11.25**	4.28	-19.55**
Chamran*Line#10	10.83*	-16.67**	15.58	-47.29**	8.12**	5.83**	15.49*	-3.11
Tajan*Zagros	-14.29**	-16.79**	-12.48	-18.11	-0.28	-14.03**	11.93*	-24.88**
Tajan*Moghan3	-5.58	-9.62*	2.3	-7.54	7.77**	-8.06**	49.17**	0.08
Tajan*N-81-18	-6.18	-20.83**	13.14	-42.65**	-9.58**	-28.75**	-1.84	-68.66**
Tajan*Koohdasht	-5.21	-5.42	8.04	-5.5	-3.54	-13.75**	9.97	-9.94
Tajan*N-80-19	-10.21*	-20**	5.75	-31.09*	-17.92**	-23.75**	-42.83**	-65.59**
Tajan*Line#10	10.42*	-19.58**	6.19	-69.22**	-3.96*	-23.75**	-22.01**	-87.19**
Zagros*Moghan3	-12.22*	-18.75**	-3.83	-8.04	2.36	0.28	16.99**	4.71
Zagros*N-81-18	-4.1	-21.25**	24.85	-25.30	5.83**	0.42	8.6	-21.42**
Zagros*Koohdasht	-6.04	-8.75*	18.5	-0.68	15.21**	11.67**	56.02**	39.14**
Zagros*N-80-19	-21.87**	-34.17**	-12.44	-43.65**	-1.25	-9.17**	-7.69	-21.73**
Zagros*Line#10	7.08	-25.42**	42.76**	-27.02*	8.12**	2.08	35.25**	6.87
Moghan3*N-81-18	-1.87	-12.5**	4.17	-41.78**	6.39**	3.06	7.09	-10.64*
Moghan3*Koohdasht	-5.76	-9.58*	-11.24	-34.62**	2.29	-3.33*	19.04**	-10.14*
Moghan3*N-80-19	-15.35**	-21.11**	-31.27*	-58.27**	-7.92**	-17.92**	-21.86**	-48.19**
Moghan3*Line#10	3.19	-22.78**	-2.95	-68.53**	-2.29	-6.25**	-1.93	-18.03**
N-81-18*Koohdasht	0.13	-14.31**	22.45	-46.88**	5.48**	-3.47*	41.76**	-5.15
N-81-18*N-80-19	-0.83	-5.69	5.63	-13.31	-7.64**	-20.97**	-5.56	-49.62**
N-81-18*Line#10	10.90*	-4.44	25.67	6.04	5.21**	4.58	19.56**	17.92**
Koohdasht*N-80-19	-12.5*	-22.08**	5.13	-45.26**	-13.54**	-17.92**	-27.77**	-30.62**
Koohdasht*Line#10	12.71*	-17.08**	29.53*	-59.43**	7.5**	-2.08	29.71**	-15.56**
N-80-19*Line#10	6.87	-13.33**	11.58	-26.99*	1.04	-12.92**	7.09	-35.33**

* = Significant at the 0.05 level of probability, ** = Significant at the 0.01 level of probability.

and pAUDPC traits, respectively.

Twenty out of twenty-eight crosses showed negative mid parent heterosis (Table 5) for infection response but only eleven were significant. All of the crosses showed negative better parent heterosis and twenty-five were significant. Maximum decrease over the mid parent and also highest negative better parent heterosis was recorded in hybrid between Zagros and N-80-19 (-21.87% and -34.16%, respectively).

Heterotic studies for iAUDPC revealed that ten crosses showed negative mid parent heterosis and twenty-six crosses showed negative better parent heterosis. Only Moghan3*N-80-19 cross showed significant negative mid parent heterosis (-31.27%) for iAUDPC, whereas eighteen crosses were significant for negative better parent heterosis. Tajan*Line#10 (-69.22%) followed by Moghan3*Line#10 (-68.53%) and Moghan3*N-80-19 (-58.27%) recorded highest negative better parent heterosis for iAUDPC.

Heterosis results for picnidia density are given in Table 5. Eleven crosses showed negative mid parent heterosis and eighteen crosses had negative better parent heterosis. Six of eleven and all of eighteen were significant. Maximum negative mid parent heterosis recorded by Tajan*N-80-19 (-17.91%), whereas maximum better parent heterosis recorded by Tajan*N-81-18 (-28.75%) followed by Tajan*N-80-19 and Tajan*Line#10 (both -23.75%). Table 5 shows eight crosses which revealed negative mid parent heterosis for pAUDPC where four are significant. Twenty-one of the crosses showed negative better parent heterosis with eighteen significant. Cross Tajan*N-80-19 recorded highest negative mid parent heterosis (-42.83%). Maximum negative better parent heterosis recorded by Tajan*Line#10 (-87.19%) followed by Tajan*N-81-18 (-68.66%) and Tajan*N-80-19 (-65.59%).

Complete analysis of variance (Table 2) following Walters and Morton (1978) exhibited that item a, which measures additive gene effects were highly significant for all traits and accounted for a high proportion of the total

Table 6. Estimates of genetic components for variation of four studied characters.

Genetic Component	Infection response	iAUDPC	Picnidia density	pAUDPC
B-1	$1.085 \pm 0.107^{n.s}$	$0.872 \pm 0.103^{n.s}$	$0.810 \pm 0.204^{n.s}$	$0.819 \pm 0.126^{n.s}$
$D \pm S.E_{(D)}$	$4.578 \pm 0.1756^{**}$	$36.287 \pm 0.6652^{**}$	$1.9217 \pm 0.0677^{**}$	$21.1981 \pm 0.6211^{**}$
$H_1 \pm S.E_{(H1)}$	$2.3981 \pm 0.4037^{**}$	$9.341 \pm 1.5291^{**}$	$1.6026 \pm 0.1556^{**}$	$17.014 \pm 1.4278^{**}$
$H_2 \pm S.E_{(H2)}$	$1.3535 \pm 0.3512^{**}$	$7.346 \pm 1.3303^{**}$	$1.0755 \pm 0.1354^{**}$	$13.2139 \pm 1.2422^{**}$
$F \pm S.E_{(F)}$	$3.8035 \pm 0.4149^{**}$	$7.623 \pm 1.5717^{**}$	$1.4997 \pm 0.16^{**}$	$5.7999 \pm 1.4675^{**}$
h^2	$0.5218 \pm 0.2355^{*}$	$0.3078 \pm 0.8922^{n.s}$	$0.0064 \pm 0.0908^{n.s}$	$2.9173 \pm 0.833^{**}$
$\sqrt{H_1/D}$	0.7238	0.507	0.9132	0.8959
$\frac{H_2}{4H_1}$	0.1411	0.197	0.1678	0.1942
K_D/K_R	3.6943	1.5222	2.4922	1.3604
h^2/H_2	0.4	0	0	0.2
$h^2_{N.S}$	0.60	0.77	0.61	0.72
$h^2_{B.S}$	0.83	0.87	0.95	0.97

* = Significant at the 0.05 level of probability, ** = Significant at the 0.01 level of probability.
D = additive effect, H_1 and H_2 = dominance effect, F = determines frequencies of dominant to recessive alleles in parents, H^2 = determines the overall dominance effect due to heterozygous loci, $h^2_{N.S}$ = narrow sense heritability, $h^2_{B.S}$ = broad sense heritability, K_D/K_R = ratio of dominant to recessive genes in parents.

Table 7. Analysis of variance for Wr + Vr in studied characters.

S.O.V	Df	Infection response	iAUDPC	Picnidia density	pAUDPC
Block	2	1.521	20.503	0.004	0.323
$W_r + V_r$	7	4.664^{*}	101.271^{*}	0.605^{*}	128.424^{*}
Error	14	0.561	32.646	0.016	2.283

* = Significant at the 0.05 level of probability.

Table 8. Analysis of variance for Wr – Vr in studied characters.

S.O.V	Df	Infection response	iAUDPC	Picnidia density	pAUDPC
Block	2	0.037	1.489	0.020	1.466
$W_r - V_r$	7	$0.063^{n.s}$	$5.732^{n.s}$	0.107^{*}	10.716^{*}
Error	14	0.123	2.598	0.009	0.829

* = Significant at the 0.05 level of probability, n.s = Not significant at the 0.05 level of probability.

variation. The over all dominance component b, was also highly significant, indicating the important role of dominance effect. Zhang et al. (2001) reported that non-additive effects are important at the seedling stage resistance. The significant value of b_1 for infection response and pAUDPC indicated the presence of directional dominance of the genes for these characters and non-significant value of b_1 for picnidia density and iAUDPC showed the absence of directional dominance effect of the genes. Asymmetry of gene distribution among the parents for all traits was represented by significant b_2 component, while significant b_3 for all traits except iAUDPC indicated the presence of specific gene effects.

For the validity of additive-dominance model, two scaling tests were employed following Mather and Jinks (1982). For all traits, the regression coefficient test indicated that b differed significantly from zero but not from unity (Table 6) and according to second test, Wr + Vr (Table 7) was significant, indicating the presence of dominance, whereas for infection response and iAUDPC, Wr –Vr (Table 8) being non-significant, indicated the absence of non allelic interaction. Thus, both tests suggested adequacy of the additive-dominance model for these characters, but for picnidia density and pAUDPC, Wr –Vr being significant indicating the presence of non allelic or epistatic interaction. Failure of both tests completely invalidates the additive- dominance model.

However, if one of them fulfils the assumption, the additive-dominance model was considered partially adequate. Johnson and Askel (1964), Wilson et al. (1978) and Azhar and Mcneilly (1988) have also estimated the components of variance for such type of partially adequate models.

The estimates of genetic components of variations (Table 6) revealed significant values of both D and H components suggesting that all traits were under the control of both additive and dominance gene effects. Unequal values of H_1 and H_2 for all of them indicated the presence of positive and negative alleles in unequal frequencies. This was supported by $H_2/4H_1$ ratio which indicated the presence of positive and negative alleles in unequal frequencies. It was suggested that where the genes are equally distributed among parents, this value is equal to 0.25 (Singh and Chaudhry, 1985). For all traits, F component was positive and significant, indicating the present of dominance genes and also confirmed by the ratio of dominant to recessive genes (K_D/K_R) which is more than 1 for all traits. Significant value of h^2 for infection response and pAUDPC indicated the presence of overall dominance effect due to heterozygous loci affecting the expression of these traits. For picnidia density and iAUDPC, value of h^2 was not significant displaying the absence of dominance effect due to heterogeneity at loci. The average degree of dominance $(H_1/D)^{1/2}$ for all traits was less than 1 indicating partial dominance with additive gene effect.

The positive intercept of Wr/Vr regression line (Figures 1a, 2a, 3a and 4a) for all traits also indicated additive gene action with partial dominance. The number of gene group differentiating the parents (h^2/H_2) was less than unity for all the characters suggesting the control of one gene group. Which, as discussed in introduction, is in agreement with Rillo and Caldwell (1966), Rosielle and Brown (1979), Wilson (1979) and Lee and Gough (1984).

High narrow and broad sense heritabilities were recorded (Table 6) for all traits. Heritability in broad sense estimates the genetic proportion (additive + dominant + interaction) of the total phenotypic variation, while heritability in narrow sense estimates only the additive portion. Their relative magnitude explicates the proportion of additive variation within genetic variation. Thus, here greater portion of heritable variation was of additive nature.

Placement of array points displayed (Figure 1a) that Koohdasht had the maximum dominant genes for infection response being nearest to the origin, whereas, Line#10 had the least dominant genes being farthest from the origin. For iAUDPC, N-81-18 and Tajan possessed the maximum dominant and recessive genes, respectively (Figure 2a). For picnidia density, Zagross had the maximum dominant genes and Line#10 had the most recessive genes (Figure 3a). N-80-19 genotype possessed maximum dominant genes for pAUDPC, whereas moghan3 had the most recessive genes for this trait (Figure 4a).

To find out the correlated response of dominant genes

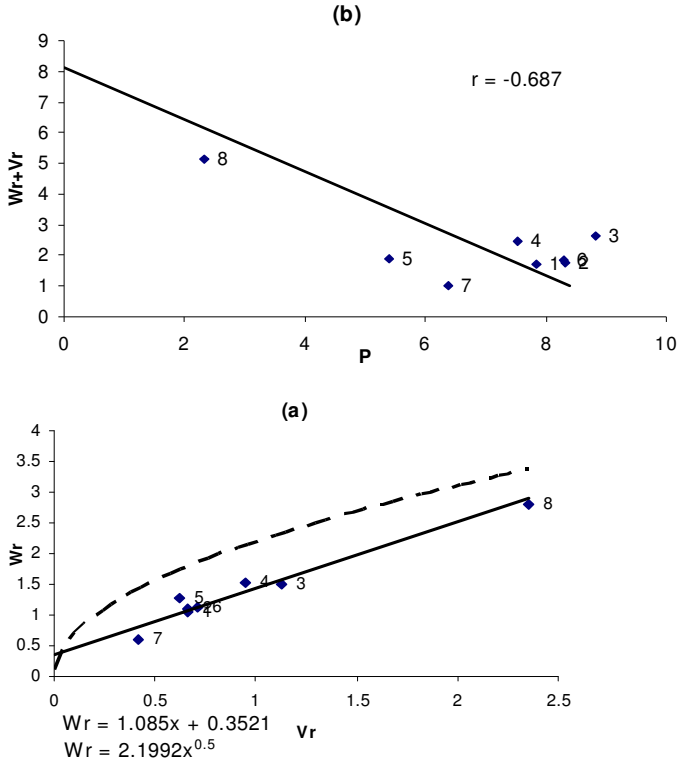

Figure 1. Wr/Vr graph (a) and Wr +Vr/P graph (b) for infection response.
(1. Chamran, 2. Tajan, 3. Zagros, 4. Moghan3, 5. N-81-18, 6. Koohdasht, 7. N-80-19, 8. Line#10).

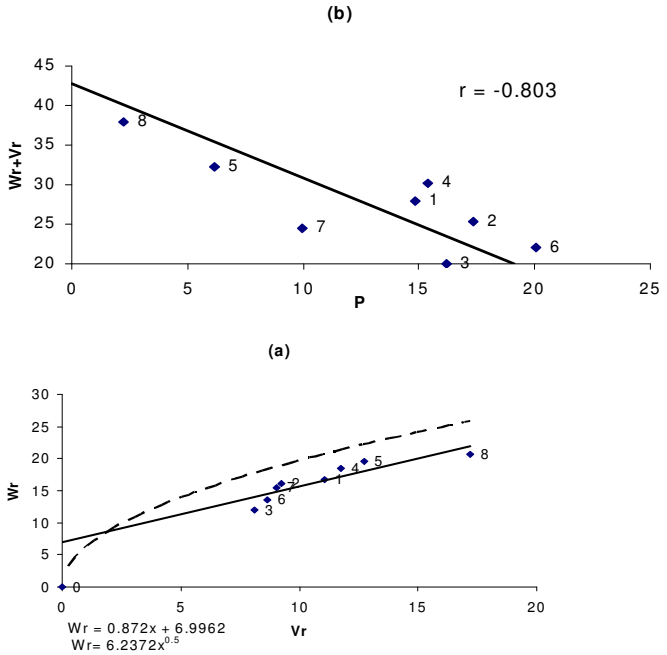

Figure 2. Wr/Vr graph (a) and Wr +Vr/P graph (b) for iAUDPC.
(1. Chamran, 2. Tajan, 3. Zagros, 4. Moghan3, 5. N-81-18, 6. Koohdasht, 7. N-80-19 and 8. Line#10).

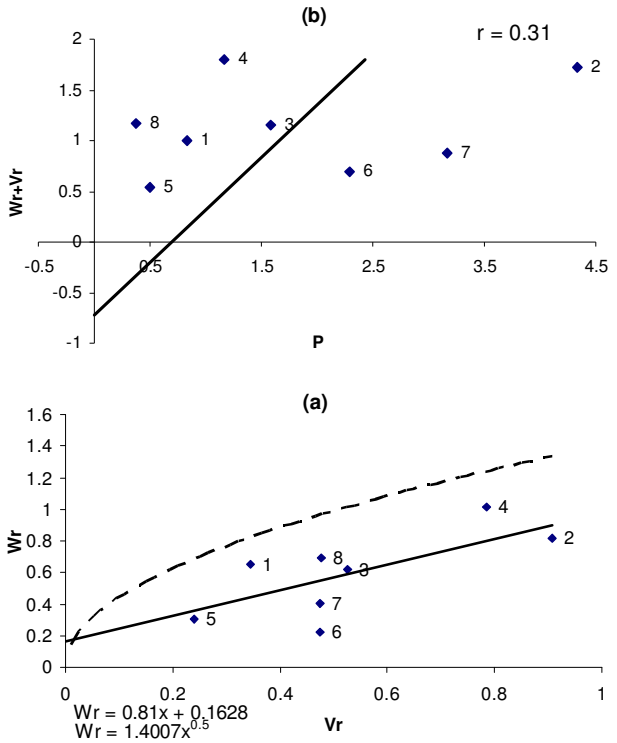

Figure 3. Wr/Vr graph (a) and Wr +Vr/P graph (b) for Picnidia density.
(1. Chamran, 2. Tajan, 3. Zagros, 4. Moghan3, 5. N-81-18, 6. Koohdasht, 7. N-80-19, 8. Line#10).

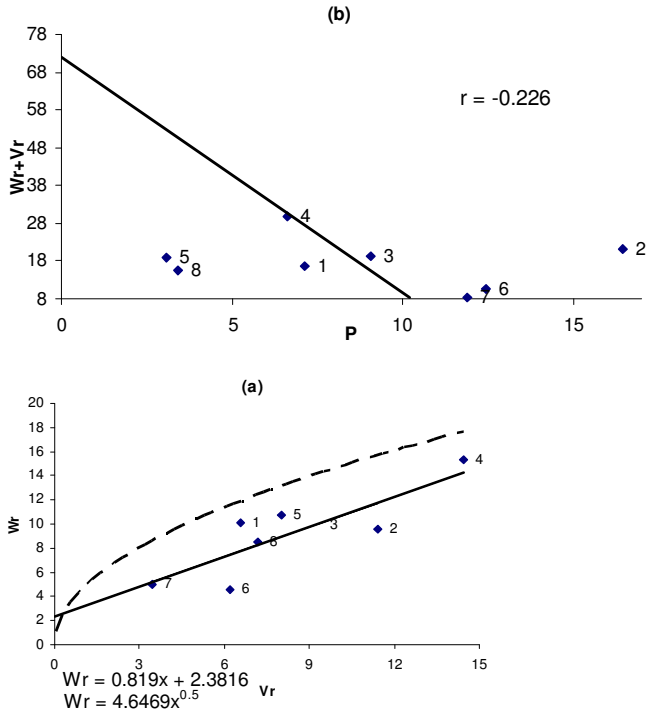

Figure 4. Wr/Vr graph (a) and Wr +Vr/P graph (b) for pAUDPC.
(1. Chamran, 2. Tajan, 3. Zagros, 4. Moghan3, 5. N-81-18, 6. Koohdasht, 7. N-80-19, 8. Line#10).

with phenotype of the common parent, Wr+Vr values of the arrays were plotted against the parental values (Figures1b, 2b, 3b and 4b). For all traits, except picnidia density, the graph presented that parents with least symptoms level had greater Wr+Vr values and parents with most symptoms level had smaller Wr+Vr values. Thus, it was clear that greater level of infection response, iAUDPC and pAUDPC resulted due to more dominant genes. Dominant genes increased the symptoms and recessive genes decreased them. Whereas for picnidia density, a positive correlation clearly depicted that the parents with more picnidia density level had larger values of Wr+Vr and thus, had lesser number of dominant alleles. So, dominant alleles decreasing picnidia density level and increasing resistance.

For all traits, the additive and dominance gene effects are significant; however, the magnitude variation is high. The Wr/Vr graph also shows additive gene control for these traits and with the observation of predominant GCA effects for enhanced resistance, improvement of STB resistance can be achieved by crossing two parents having good resistance, while selecting resistant progeny from particular crosses based on the direction of the crosses is also predictable. Chartrain et al. (2004a) suggested that 'pyramiding' several resistance genes in one cultivar may be an effective and durable strategy for breeding of resistance to STB in wheat. High negative GCA value in Line#10 and N-81-18 for all studied traits indicating that these genotypes carrying resistant additive genes and so have potential for obtaining superior lineages in selection programs for STB resistance. Crosses with these parents because of additive nature inheriting resistance consistently and through selection program can accumulate resistant in one genotype. Heritability of all traits is high. Therefore, early generation selection would be effective. Best negative SCA combinations and high negative heterosis values belongs to hybrids of N-80-19 with other genotypes, so in breeding programs while our objective is benefiting from heterosis and dominant effects , using genotype N-80-19 as a parent in crosses should be noticed.

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