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Estimation of combining abilities and heterosis of *Septoria tritici* blotch resistance in wheat genotypes

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Abstract

Septoria tritici blotch (STB), caused by *Mycosphaerella graminicola* (anamorph *S. tritici*) is currently the most serious foliar disease of wheat worldwide. An eight-parent half diallel set of crosses between wheat genotypes were planted in the field of agricultural research center of Gorgan, Golestan in a randomized complete block design (RCBD) with three replicates. Plants were inoculated in tillering, long stem and appearance of flag leaf stages. Disease rating was visually recorded by using the double-digit scale (00–99). Disease severity and its AUDPC (sAUDPC) were calculated. The analysis of variance for combining ability showed the significant variation for both characters, indicating a wide range of variability among the genotypes. High significant variation due to general combining ability and also specific combining ability indicated the importance of additive as well as non additive types of gene action in inheritance of these characters; however Baker ratio showed the more importance of additive effects than non-additive effects of genes for both traits. Some negative and significant heterosis and heterobeltiosis effects were emphasized the existing of dominance gene effects to control resistance to STB.

Keywords: STB, Triticum, GCA, SCA, gene effect

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 regions 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, 2001a,b; 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 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, 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). Jlibene and El 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 (Vakili et al, 2010; Van

Ginkel and Scharen, 1987; Danon and Eyal, 1990; Jlibene *et al.*, 1994; Simon and Cordo, 1997, 1998). The seedling stage study of resistance to STB indicated that GCA was more important than SCA and additive gene effects played the major role in host response to STB (Vakili *et al*, 2010). Resistant cultivars provide an effective and economical way to control the disease. A better understanding of the relative importance of general and specific combining abilities (GCA/SCA) of resistance to STB would potentially leads to more efficient development of resistant cultivars and deployment of germplasm resources. Therefore, the objective of the present research was to estimate the combining abilities in several wheat genotypes exhibiting various levels of STB resistance and also to evaluate the heterosis values for better understanding of dominance gene effects.

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 promising lines while the rest were cultivar. Line pedigrees and STB infection responses of all genotypes are presented in Table 1. F1 crosses were obtained by hand emasculation and pollination in the field of agricultural research center of Gorgan, Golestan in 2008. Thirty-six genotypes including parents and F1 were planted in the field under randomized complete block design with three replications under mist irrigation in 2009. Tajan cultivar was used as a susceptible check, Figure 1 showed symptoms on Tajan as highly susceptible cultivars (a) and Chamran as a moderately susceptible cultivar (b). Each experimental unit was consisted of double lines with 100 and 30 cm interval between and within rows, respectively.

 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.



Fig 1. *Septoria tritici* blotch disease symptoms on two different cultivars used in diallel crossing; (a) Tajan cultivar as a susceptible check, (b) Chamran a moderately susceptible cultivar

 Table 2. Mean squars of general/specific combining abilities and their ratio

SOV	Mean square		
5.0. v	Disease Severity	sAUDPC	
GCA	1128.15**	2045.49^{**}	
SCA	60.96**	355.68**	
Error	1.88	20.67	
2GCA	0.07	0.04	
$\overline{2\text{GCA} + \text{SCA}}$	0.97	0.94	

Preparation of spore and disease evaluation

One isolate of *S. tritici* originating from field collections of Gorgan was used. For extracting the pathogen, direct method of Eyal *et al* (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 $20 \pm 2^{\circ}$ C. After a week, small pink colonies moved to PDA media without antibiotic and kept in incubator at $20 \pm 2^{\circ}$ 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 10⁶ -10⁷ mL⁻¹ of conidia as determined by hemacytometer counts. Plants were inoculated in tillering stage, long stem stage and appearance of flag leaf stage. Disease rating was visually recorded as soon as the first symptoms appearance on the lowest leaves 8 times with 4 interval day using the double-digit scale (00-99) developed as a modification of Saari and Prescott's severity scale to assess wheat foliar diseases (Saari and Prescott, 1975; Eyal et al., 1987). The first digit (D_1) indicates vertical disease progress on the plant and the second digit (D_2) refers to severity measured as diseased leaf area. Four plants in each replication were selected randomly to record disease rating and the mean of them was applied. For each score, disease severity percentage was calculated based on the following formula (Sharma and Duveiller, 2007)

% severity = $(D_1 / 9)(D_2 / 9)100$

The area under disease progress curve (AUDPC) was calculated using severity percentage estimates based on Moldovan et al (2005) according to the following function.

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

Where y_i = disease severity on the *i*th date, t_i = *i*th day, and *n* = number of dates on which septoria tritici blotch was recorded. Disease severity of the last assessment while genotype as susceptible check was severely diseased (90% or more disease severity) and sAUDPC (disease severity area under the disease progress curve) were used for analysis.

All data were normal based on Kolmogorov Smirnov's test in SPSS software. Data analysis was performed using D2 genetic software.

Results

For estimating combining ability effects, method 2, model 1 Griffing (Griffing, 1956) that contains parents and F1 crosses was used. The analysis of variance for combining ability (Table 2) showed the significant variation for both characters, indicating a wide range of variability for resistance to STB among the genotypes. Highly significant variation due to general combining ability (GCA) and also specific combining ability (SCA) indicated the importance of additive as well as non additive types of gene action in inheritance of these characters. Vakili et al (2010) by estimatting genetic components of STB resistance in seedling stage suggested that all traits related to STB resistance were under the control of both additive and dominance gene effects. High value of Baker ratio (Baker, 1978) for both traits, showed the more importance of additive effects than non-additive effects of genes. Same results reported by Vakili et al (2010), Zhang et al (2001), Van Ginkel and Scharen (1987), Danon and Eyal (1990) and Jlibene et al (1994). To select the most resistant genotypes, those with less value of 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 3) to reduce Disease Severity and sAUDPC (increasing resistance) and also GCA of N-81-18, N-80-19 and Moghan3 genotypes for both traits were negative and highly significant (P, 0.01). In Chamran cultivar GCA of sAUDPC was negative and significant (P, 0.05) too. Negative GCA values in mentioned genotypes indicating that resistance to STB was consistently inherited in crosses with these parents. Through crosses, nine genotypes showed

Line#10	N-80-19	Koohdasht	N-81-18	Moghan3	Zagros	Tajan	Chamran	
			Disease severity					
-12.48**	-6.41 [*]	0.49	-10.77***	-2.12	5.10^{*}	7.35**	2.74^{**}	Chamran
-0.09	-2.66	-10.65**	1.66	4.51*	0.16	16.28**		Tajan
-5.27*	-2.11	-12.50**	-0.66	2.26	9.93**			Zagros
-9.62**	-3.56	12.02**	-5.01*	-3.02**				Moghan3
-3.84	2.22	3.33	-5.00**					N-81-18
4.52^{*}	7.69^{**}	3.03**						Koohdasht
-7.19**	-4.46**							N-80-19
-18.59**								Line#10
			sAUDPC					
-17.97**	-11.53**	11.94**	-18.45**	-13.49**	17.26**	24.63**	-3.15*	Chamran
-19.22**	-17.47**	-26.50**	10.60^{*}	-7.24	-8.02	54.63**		Tajan
-8.06*	-10.81**	-40.67**	-12.03**	20.73**	26.00^{**}			Zagros
-13.67**	-6.16	35.81**	-10.01*	-11.38**				Moghan3
-5.70	5.32	2.22	-14.42**					N-81-18
9.03*	13.28**	5.38**						Koohdasht
2.29	-12.75**							N-80-19
-44.30**								Line#10

Table 3. General combining ability effects (diagonal values) and specific combining ability effects (above diagonal) for studied characters.

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

Table 4. Estimates of mid parent and better parent heterosis for studied characters.

CDOSSES	Disease Severity		sAUDPC		
CRUSSES	%MP	%BP	%MP	%BP	
Chamran*Tajan	4.27^{**}	19.89**	6.64**	69.57**	
Chamran*Zagros	-4.52**	4.97^{**}	3.29^{**}	37.71**	
Chamran*Moghan3	-15.18^{**}	7.65^{**}	-13.72**	-2.53**	
Chamran*N-81-18	-39.43**	-23.13**	-25.62**	-21.06**	
Chamran*Koohdasht	-5.53**	6.29^{**}	9.56^{**}	14.75^{**}	
Chamran*N-80-19	-29.36**	-14.21**	-18.27**	-14.08^{**}	
Chamran*Line#10	-64.30**	-37.48**	-39.89**	-6.48**	
Tajan*Zagros	-4.03**	0.00	-13.45*	-0.11**	
Tajan*Moghan3	7.68^{**}	61.48^{**}	-9.84**	68.86^{**}	
Tajan*N-81-18	-2.52^{**}	46.17^{**}	-4.15**	65.22^{**}	
Tajan*Koohdasht	-14.21**	12.58^{**}	-19.47**	20.46^{**}	
Tajan*N-80-19	-9.92**	28.68^{**}	-19.75**	36.56**	
Tajan*Line#10	-17.63**	75.11^{**}	-28.91**	99.19 ^{**}	
Zagros*Moghan3	-2.60**	38.52**	8.50^{**}	68.11^{**}	
Zagros*N-81-18	-13.48**	23.04**	-20.39**	14.28^{**}	
Zagros*Koohdasht	-25.00^{**}	-6.29**	-31.93**	-14.15^{**}	
Zagros*N-80-19	-15.75	14.30**	-18.90**	15.06^{**}	
Zagros*Line#10	-37.48**	25.03^{**}	-26.29**	66.54**	
Moghan3*N-81-18	-23.13**	-23.13	-16.32**	-11.22**	
Moghan3*Koohdasht	31.07**	46.17**	36.16**	61.94**	
Moghan3*N-80-19	-18.51**	-15.39**	-11.36**	-5.05**	
Moghan3*Line#10	-61.98**	-50.07**	-35.61**	-13.83**	
N-81-18*Koohdasht	3.39**	15.30**	-3.13**	7.95**	
N-81-18*N-80-19	-11.05**	-7.65**	-7.83**	-6.99**	
N-81-18*Line#10	-52.40**	-37.48**	-33.78**	-4.61**	
Koohdasht*N-80-19	13.38 ^{**}	21.49 ^{**}	7.34**	18.44	
Koohdasht*Line#10	-8.29**	37.63**	-3.40**	59.93**	
N-80-19*Line#10	-63.70**	-50.08**	-22.16**	13.46**	

*= Significant at the 0.05 level of probability **= Significant at the 0.01 level of probability better parent heterosis : BP mid parent heterosis: MP

significant negative SCA values for Disease Severity and thirteen crosses for sAUDPC. For Severity two crosses, Zagross * Koohdasht and Chamran*Line#10 had the best SCA values to reduce symptoms of disease and increasing resistance. Also the best SCA combination for sAUDPC belongs to hybrid between Zagross and Koohdasht. Both genotypes Zagross and Koohdasht are known as susceptible genotypes to *Septoria tritici* blotch. It is not at all unusual for susceptible parent of a cross to contribute alleles for increasing resistance to diverse diseases and pests of many crops (Cherif and Harrabi, 1993; Dirlewanger *et al.*, 1994; Dixon *et al.*, 1991; Pernet *et al.*, 1999; Thomas *et al.*, 1995). This may leads to transgressive segregation, with progeny lines that combine resistance genes from both parents and hence have better resistance than either parents (Chartrain *et al.*, 2004b). Twenty-five out of twenty-eight crosses showed

significant negative mid parent heterosis (Table 4) for Severity. For this trait just ten crosses showed significant negative better parent heterosis. Maximum decrease over the mid parent heterosis observed in crosses of Line#10 with Chamran, N-80-19 and Moghan3, respectively. Also crosses of Line#10 with N-80-19 and Moghan3 genotypes showed the highest negative better parent heterosis. Heterotic studies for sAUDPC revealed that twenty-two crosses showed negative mid parent heterosis. Maximum decrease over the mid parent and highest negative better parent heterosis were recorded in Chamran* Line#10 and Chamran* N-81-18, respectively.

Discussion

These results suggested that among the genotypes promising Line#10 is an excellent source of resistance to STB. Major sources of resistance to STB used in world breeding programmes for decades were such as Kavkaz-K4500, Veranopolis, Catbird and TE9111 (Chartrain et al., 2004a). Talebi et al (2010) introduced another cultivar for STB resistance, they suggested Wangshuibai' as a valuable source of resistance to STB for wheat breeding, especially in Mediterranean environments. High negative GCA value in promising Line#10 for both studied traits indicating that this genotype carrying resistant additive genes and so have potential for obtaining superior lineages in selection programmes for STB resistance. With the observation of predominant GCA effects for enhanced resistance, improvement of STB resistance can be achieved by crossing 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 for resistance to STB in wheat. Sharma and Duveiller (2007) suggested N-81-18 (Milan/Shang-hi#7) cultivar is the most stable cultivar for spot blotch resistance that is in agreement with our finding for STB. In this study results showed GCA effect of this genotype was high and negative significantly, exhibiting the additive effect of resistant genes, so justify the stability of resistance to STB. Crossing between promising Line#10 and N-81-18 because of additive nature inheriting resistance consistently and through selection program can accumulate resistant in one genotype. Same results obteined in our previous study that conducted in seedling stage (Vakili et al, 2010). Results insisted that it's not reasonable to use just resistant genotypes in breeding programes because of transgressive segregation nature while can obtain progenies carreing new resistant genes from susceptible genotypes, so we proposed to apply vary different rang of genotypes in disease resistance breeding program.

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