Genetic analysis and heritabilities of resistance to *Mycosphaerella graminicola* in wheat

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ABSTRACT

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Septoria tritici blotch is an important wheat disease in many areas of the world including Iran's warm, humid regions. In this study, crosses were made between resistant breeding line BOBWHITE#1/FENGKANG and commercial cultivars Moghan3, Koohdasht, Tajan and Morvarid, and derived F_1, F_2, BC_1 and BC_2 generations were used to determine the genetic control of necrosis, pycnidia traits and the area under disease progress curve (AUDPC) in seedlings under greenhouse conditions using generation mean analysis. The cultivars and derived populations were evaluated using a randomized complete block design with three replications. Significant differences among generations were found for all traits, and genetic analyses of those traits were performed. Results showed that additive, non-additive and epistatic effects played roles in controlling all traits. The role of dominance effects and dominance × dominance interaction was much greater in controlling the studied traits. Therefore, hybridization methods and selection in the final generation are recommended for improving resistance to septoria tritici blotch.

Key words: *Mycosphaerella graminicola*, dominance effect, heterosis

INTRODUCTION

Wheat, an essential food crop for human societies, is grown in wide range of environments. The wheat plant is exposed to different biotic and abiotic stresses at all growth stages, and wheat production and quality are affected by these factors. Foliar fungal pathogens are significant threats to wheat crop production. Septoria tritici blotch of wheat (*Mycosphaerella graminicola*, anamorph *Septoria tritici)* causes economically significant yield losses in most wheat growing areas of the world. Yield losses can range from 31 to 54% in climates conducive to disease development (Eyal *et al.,* 1985). The Middle East is probably the origin of *M. graminicola* (McDonald *et al*., 1999), but this fungus is now a worldwide problem affecting wheat-growing areas in Europe (Halama, 1996), Australia (Loughman and Thomas, 1992), Canada (Chungu *et al.,* 2001), and the United States (Garcia and Marshall, 1992, Mundt *et al*., 1998). Foliar fungicides are an option for controlling Septoria tritici blotch (STB); however, the application of fungicides to control STB is expensive, not entirely reliable and not environmentally friendly. Resistant cultivars provide an effective and economical way to control the disease, but little is known about the genetics of STB resistance in wheat, and breeders have to rely on unknown genes when breeding for resistance (Eyal, 1999). Genetic analysis of virulence and resistance in the wheat-*M. graminicola* pathosystem suggests that pathogenicity is controlled by several loci and is likely inherited as a quantitative trait (Kema *et al.,* 1996; Zhan *et al*., 2005).

Despite the high economic importance of this disease, as of 2000 only four resistance genes had been identified (Rillo and Caldwell, 1966; Somasco *et al*., 1996; Wilson, 1985), none had been mapped and no molecular markers were available for marker-assisted selection (Goodwin, 2007). Fortunately, this situation has changed rapidly. During the past decade, 13 STB resistance genes have been identified and mapped in the wheat genome (Adhikari *et al*., 2004abc; Arraiano and Brown, 2006; Arraiano *et al*., 2001, 2007; Brading *et al*., 2002; Chartrain *et al*., 2005ab, 2009; McCartney *et al*., 2003), and several molecular markers have been developed that can be used in marker-assisted selection. Additional resistance genes have been identified recently (Tabib Ghaffary *et al*., 2010),

which should soon bring the catalogue of mapped *Stb* genes to at least 17.

An understanding of the inheritance of STB resistance in wheat is needed for designing an effective breeding program. However, studies of the inheritance of host resistance have reported contradictory results. Early reports on the specificity of diseases were controversial; hence, the wheat-*M. graminicola* pathosystem was traditionally thought to be controlled by quantitative trait loci in both host and pathogen (Eyal *et al*., 1973, 1985; van Ginkel and Scharen, 1988; Johnson, 1992; Parlevliet, 1993). It is a testimony to fungal genetics that this hypothesis was eventually disproved when experimental evidence showed that host and pathogen interact according to the gene-for-gene hypothesis (Brading *et al*., 2002; Kema *et al*., 2002). Given that different types of gene actions are important in different crosses, the breeding strategy for developing a desirable genotype should be based on the gene action involved in that particular cross.

Quantitative resistance has been found in different genotypes (Jlibene *et al*., 1994; Brown *et al*., 1999; Simon *et al*., 2001) and most commercially grown cultivars range from moderately resistant to susceptible, indicating that minor gene effects are also present. However, most investigations have concentrated on studying major gene effects. Major genes are interesting because of the high level of resistance they confer, resulting in an almost complete absence of disease symptoms in the host. Partial resistance, however, is very important due to its putative durability and its expression under a broad spectrum of pathogen isolates. A few genes may be enough to confer resistance that will hold up in farmers' fields (Dubin and Rajaram, 1996).

Some of the components of partial resistance to *M. graminicola* may be controlled by just a few genes (Jlibene and El Bouami, 1995). Components that are genetically different could be combined into the same genetic background by crossing (van Ginkel and Rajaram, 1999). However, significant non-additive effects have often been identified (van Ginkel and Scharen, 1987; Bruno and Nelson, 1990; Danon and Eyal, 1990; Jonsson, 1991; Jlibene *et al*., 1994; Simon and Cordo, 1997, 1998). Heritability tends to be only moderate (Simon *et al*., 1998), but progress in breeding for resistance may still be possible.

Studies on the inheritance of resistance to Septoria tritici blotch have largely concentrated on bread wheat (*Triticum aestivum*). Often, seedlings have been assessed for disease reaction. The objective of this study was to investigate the inheritance of *S. tritici* resistance in wheat at the seedling stage using a quantitative genetic approach.

MATERIALS AND METHODS

The experimental materials consisted of six generations (P_1, P_2, F_1, F_2, BC_1 and BC_2) derived from crosses between resistant breeding line BOBWHITE#1/FENGKANG and commercial cultivars Moghan3, Koohdasht and Tajan Morvarid. The names and parentage/cross names of bread wheat genotypes used in this study and their response to *Septoria tritici* are presented in Table 1. Seven seeds of each genotype were planted in plastic pots in the greenhouse using a completely randomized design with three replications. Greenhouse temperature and humidity were controlled at 22.5 ± 2.5°C and 80±5%, respectively. Following full emergence of the second leaf, seedlings were inoculated using the direct method of Eyal *et al.* (1987) with a single isolate of STB (Gorgan isolate). Moisture was maintained by spraying water with an atomizer several times a day for three days. Fourteen days after inoculation, the susceptible check was severely infected (≥ 90% of lesions bearing pycnidia), and STB symptoms were visually rated on the second leaf four times at four-day intervals.

Table 1. Bread wheat genotypes and their response to *Septoria tritici*.

Genotypes	Parentage/Cross name	Response
Breeding line	BOBWHITE#1/FENGKANG	R
Morvarid	MILAN/SHA7	MR
Moghan3	LUAN/4/V7632.23/3/V879.ABC9//PWN/PICUS	MS
Koohdasht	TR8010200	S
Tajan	BOW"s"/NKT"s"	S

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

Percentage of necrotic leaf area (necrosis) and percentage of pycnidial coverage (pycnidia) on each experimental unit were assessed and scored. These data were used to calculate the area under the disease progress curve (AUDPC) for each trait (nAUDPC and pAUDPC) using the approach of Moldovan *et al*. (2005). AUDPC data were used for statistical analysis.

Generation mean analysis was performed using the Mather and Jinks (1982) method, in which the mean of each trait is estimated as follows:

$$Y = m + \alpha [d] + \beta [h] + \alpha^2 [i] + 2 \alpha \beta [j] + \beta^2 [l]$$

where Y= the mean of generation, m= the mean of all generations, [d] = the sum of additive effects, [h]= the sum of dominance effects, [i]= the sum of additive × additive interactions, [j]= the sum of additive × dominance interactions, [l]= the sum of dominance × dominance interactions, and α, β, α^2, 2 α β and β^2 are the coefficients of genetic parameters. The genetic parameters were tested for significance using the t-test. The adequacy of the additive-dominance model was determined by the χ^2 - test. Broad- and narrow-sense heritabilities were estimated using following formulas:

$$h_b^2 = \frac{(V_D + V_H)}{(V_D + V_H + V_E)} \qquad h_b^2 = \frac{V_D}{(V_D + V_H + V_E)}$$

$$V_E = \frac{V_{P1} + V_{P2} + 2V_{F1}}{4}$$

The components of F_2 variance were obtained using the Mather and Jinks method (1982):

$D = 4\,V_{F2} - 2\,(V_{Bc1} + V_{Bc2})$ $\quad H = 4\,(V_{Bc1} + V_{Bc2} - V_{F2} - E_W)$

$F = V_{Bc2} - V_{Bc1}$ $\quad E_W = 1/4\,(V_{P1} + V_{P2} + 2V_{F1})$

where V= variance.

Mid parent heterosis, superior parent heterosis and inbreeding depression were calculated as follows:

Mid parent hetrosis = $[(F_1\text{-MP})/MP]\times 100$

$t=(F_1\text{-MP})/[(3/8V_e)^{1/2}$

Superior parent heterosis = $[F_1\text{-BP} / BP] \times 100$

$t= (F_1\text{-BP}) /[(1/2\,V_e)^{1/2}]$

Inbreeding depression = $[F_1\text{-}F_2 / F_1] \times 100$

$t= (F_1\text{-}F_2) / [(2\,EMS / r)^{1/2}]$

SAS (v.9.1) and Minitab (v.11) software were used for analysis of variance and generation mean analysis, respectively.

RESULTS

Significant differences were found among means of generations' means for all traits indicating the presence of sufficient genetic variability. The summary of the analysis of variance, mean values and their respective standard errors for different traits are presented in Tables 2, 3, 4 and 5. The joint scaling test (Mather and Jinks, 1982) was employed to estimate gene effects. The best dominance–additive model was selected using the lower Chi-square test and significant t-test for each parameter. All the genetic components of the mean for all crosses and traits were significant (Table 6). For four traits, the five-parameter model [mdhil] was the best fitted for observing the expected generation means of cross Morvarid × Tajan. In cross BOBWHITE#1/FENGKANG × Koohdasht, the best fitted model for necrosis, nAUDPC and pycnidia was [mdhil], but for pAUDPC, the four-parameter model [mdhi] was the best. For necrosis, nAUDPC, pAUDPC and pycnidia, the four-parameter model [mdhj] was the best fitted for observing the expected generation means in cross BOBWHITE#1/FENGKANG × Moghan3.

The dominance, additive and epistatic effects were significant in all crosses, but in general the dominance effects were greater than the additive components. Results of Zhang *et al*. (2001), van Ginkel and Scharen (1987), Ramezanpour *et al.* (2010) and Vakili Bastam *et al*. (2010) support these findings. They also found significant additive, dominant and epistatic genetic effects for these traits, but the additive genetic effects were more important. The combined [h] + [l] component was larger than the additive component. This may be due to overdominance or unidirectional effect or dispersion of genes in the parents which are responsible for this reduced estimation of additive component [d] compared to dominance component [h]. This also suggests that selection should be delayed for several generations until a high level of gene fixation is attained; therefore, utilizing the heterosis method for inbreeding is useful. The signs associated with estimates of [i], [j] and [l] types of epistasis indicate the direction in which the gene effect influences the mean of the population. For [i] and [j], the sign also provides information on the association or dispersion of genes in the parents (Mather and Jinks, 1982). In all crosses and for all traits, the [d] and [l] parameters were significantly different from zero and had opposite signs; thus duplicate epistasis is indicated (Mather and Jinks, 1982). The opposite signs of [h] and [l] also indicated duplicate epistasis. This interaction increases the variation between generations and in the population (Farshadfar *et al.*, 2008).

The sign of the additive effect in all crosses and for all traits was negative, but the additive variance (D**)** was positive, except for nAUDPC in cross Morvarid × Tajan and for pAUDPC in cross BOBWHITE#1/FENGKANG × Moghan3. It is suggested that generation mean analysis estimates of the sum of genetic effects was biased by equilibrium of all gene loci. The values of scaling A, B, and C, except in pycnidia in BOBWHITE#1/FENGKANG × Moghan3, differed from zero, indicating epistatic gene effects (Mather and Jinks, 1982). Since four generations were used in the scaling test, the result of pycnidia in BOBWHITE#1/FENGKANG × Moghan3 cross was not similar to that of the joint scaling test (Table 7).

In Table 8; D, H, E_W and F are additive,

dominant, environmental and covariance between d and h in all loci components, respectively. The dominance variance analyses, except for nAUDPC in Morvarid × Tajan, were negative, because the variance of backcrosses was smaller than the F2 progeny and environment variances. Similar results were reported by Zahravi *et al.* (2007) for resistance to *Puccinia striiformis* f.sp. *tritici* in bread wheat and by Vaezi *et al*. (1999) for yield and yield components in maize.

The F value for most of the traits in each cross was negative and near to unit, and showed that the value and sign of the dominance deviation in all loci were not fixed and that most of the dominant genes were present in susceptible parents. The estimates of variance components, degree of dominance mean, dominance deviation and potency ratio are presented in Table 8.

The degree of dominance mean ($\sqrt{H/D}$) is a suitable estimator of dominance. The ratio of ($\sqrt{H/D}$) in most traits revealed the role of dominance and overdominance in controlling these traits. The value of the dominance deviation suggested that the sign and value of the gene effect in different loci were different. The degree of dominance was close to 1 for all traits, indicating the presence of the overdominance effect (Table 8). Different results were reported by Ramezanpour *et al*. (2010) and Vakili Bastam *et al.* (2010), who suggested the role of partial dominance with additive gene effect. The potency ratio indicated the overdominance effect in F1 progeny for these traits. Naghavi *et al.* (2002) found similar results in a study of genetic analysis for resistance to powdery mildew in barley.

Heterosis and heterobeltiosis were also assessed for all traits (Table 9). All crosses showed negative heterosis, but most was not significant. Also, most crosses showed negative and non-significant heterobeltiosis. Negative and significant heterosis and heterobeltiosis showed that the F_1 generations were superior to the mid parent and better parent. These findings are supported by Ramezanpour *et al*. (2010) and Vakili Bastam *et al*. (2010).

Positive inbreeding depression implied expression of undesirable traits (Table 9). Broad-sense heritability estimates the genetic proportion (additive + dominant + interaction) of the total phenotypic variation, while narrow-sense heritability estimates only the additive portion. In crosses Morvarid × Tajan, BOBWHITE#1/FENGKANG × Koohdasht and BOBWHITE#1/FENGKANG × Moghan3, necrosis, pAUDPC and pycnidia had larger broad-sense heritability and smaller narrow-sense heritability (Table 10). Considerable differences were observed between broad-sense and narrow-sense heritabilities in all crosses. These results suggest that dominance gene action was primarily responsible for the inheritance of *Septoria tritici* resistance in these crosses. A similar result was reported by Zahravi *et al.* (2007) for resistance to *Puccinia striiformis* f.sp. *tritici* in bread wheat.

Table 2. Analysis of variance of *Septoria tritici* blotch scores for different traits in cross Morvarid ×Tajan.

Sources of variation	Mean square				
	df	necrosis	nAUDPC	pycnidia	pAUDPC
Generation	5	0.250**	20.10**	0.260**	2.89**
Experimental error	12	0.035	4.60	0.006	0.43
CV (%)	-	9.300	5.50	7.910	4.60

** Significant at the 0.01 level of probability.

Table 3. Analysis of variance of Septoria tritici blotch scores for different traits in cross BOBWHITE#1/FENGKANG ×Moghan3.

Sources of variation	Mean square				
	df	necrosis	nAUDPC	pycnidia	pAUDPC
Generation	5	0.049**	4.35+	0.024**	0.82**
Experimental error	12	0.006	1.48	0.003	0.17
CV (%)	-	10.4	4.4	6.3	3.2

** Significant at the 0.01 level of probability.
+ Significant at the 0. 10 level of probability.

Table 4. Analysis of variance of Septoria tritici blotch scores for different traits in BOBWHITE#1/FENGKANG × Koohdasht.

Sources of variation	Mean square				
	df	necrosis	nAUDPC	pycnidia	pAUDPC
Generation	5	0.43**	46.62**	0.033**	2.80**
Experimental error	12	0.043	7.33	0.006	0.44
CV (%)	-	9.90	3.50	6.70	4.40

** Significant at the 0.01 level of probability.

Table 5. Estimates of generation means and standard errors for traits in three crosses.

Crosses	Traits	P_1	P_2	F_1	F_2	BC_1	BC_2
Morvarid ×Tajan	necrosis	0.95±0.08	1.10±0.13	0.89±0.08	1.10±0.13	0.81±0.09	0.89±0.09
	nAUDPC	16.70±1.10	18.40±1.40	16.10±0.83	17.30±0.83	15.60±0.92	16.30±0.98
	pycnidia	0.94±0.13	0.95±0.15	0.85±0.08	1.00±0.15	0.84±0.07	0.95±0.13
	pAUDPC	10.23±0.73	11.04±0.97	9.80±0.47	11.30±1.2	9.70±0.33	10.60±0.74
BOBWHITE#1/FENGKANG × Koohdasht	necrosis	0.84±0.09	1.15±0.10	0.82±0.11	1.10±0.14	0.86±0.12	0.94±0.10
	nAUDPC	15.10±0.81	9.20±7.10	15.30±1.10	17.80±0.86	15.80±0.59	17.00±0.82
	pycnidia	0.71±0.02	0.81±0.07	0.76±0.06	0.82±0.10	0.75±0.05	0.77±0.05
	pAUDPC	8.60±0.13	9.50±0.51	8.90±0.48	9.40±0.47	8.90±0.41	9.10±0.5
BOBWHITE#1/FENGKANG ×Moghan3	necrosis	0.89±0.07	0.94±0.12	0.75±0.07	0.96±0.11	0.91±0.07	0.99±0.050
	nAUDPC	15.50±0.70	15.90±0.82	14.60±0.54	16.70±0.91	16.20±0.74	16.30±0.71
	pycnidia	0.71±0.02	0.81±0.06	0.75±0.10	0.80±0.05	0.75±0.04	0.78±0.06
	pAUDPC	8.50±0.13	9.20±0.33	8.80±0.44	8.90±0.3	8.90±0.30	8.90±0.40

Table 6. Estimates of components of generation mean analysis for different traits in three crosses.

Crosses	Traits	m	d	h	i	j	L	χ^2
Morvarid×Tajan	necrosis	1.86^{**}	-0.08^{**}	-2.24^{**}	-0.83^{**}	-	1.27^{**}	0.06^{ns}
	nAUDPC	22.88^{**}	-0.82^{**}	-15.59^{**}	-5.37^{**}	-	8.78^{**}	0.06^{ns}
	pycnidia	1.46^{**}	-0.04^{*}	-1.23^{**}	-0.52^{**}	-	0.61^{**}	5.83^{ns}
	pAUDPC	15.80^{**}	-0.56^{**}	-11.80^{**}	-5.10^{**}	-	5.84^{**}	4.18^{ns}
BOBWHITE#1/FENGKANG ×Koohdasht	necrosis	1.84^{**}	-0.15^{**}	-1.87^{**}	-0.84^{**}	-	0.85^{**}	3.46^{ns}
	nAUDPC	21.80^{**}	-1.62^{**}	-9.59^{**}	-5.00^{**}	-	3.10^{**}	5.22^{ns}
	pycnidia	0.99^{**}	-0.04^{**}	-0.45^{**}	-0.24^{**}	-	0.21^{**}	2.64^{ns}
	pAUDPC	9.79^{**}	-0.46^{**}	-0.95^{**}	-0.80^{**}	-	-	5.80^{ns}
BOBWHITE#1/FENGKANG ×Moghan3	necrosis	0.92^{**}	-0.041^{**}	0.29^{**}	-	-	-0.46^{**}	2.80^{ns}
	nAUDPC	15.71^{**}	-0.20^{ns}	3.72^{**}	-	-	-4.80^{**}	2.01^{ns}
	pycnidia	0.85^{**}	-0.05^{**}	-0.11^{**}	-0.09^{*}	-	-	1.30^{ns}
	pAUDPC	8.82^{**}	-0.28^{**}	0.72^{**}	-	-	-0.78^{*}	7.50^{ns}

*** and ** Significant at the 0.05 and 0.01 levels of probability.**
ns: not significant.
m =Mean; d=Additive gene effect; h= Dominant gene effect; i=additive × additive gene effect; j= additive × dominant gene effect; l=dominant ×dominant gene effect.

Table 7. A, B and C scaling test for different traits in three crosses.

Crosses	Traits	A	B	C
Morvarid×Tajan	necrosis	-0.23^{**}	-0.211^{**}	0.40^{**}
	nAUDPC	-1.63^{**}	-1.810^{**}	-30.2^{**}
	pycnidia	-0.10^{*}	0.100^{ns}	0.43^{ns}
	pAUDPC	-0.60^{*}	0.400^{ns}	4.40^{ns}
BOBWHITE#1/FENGKANG × Koohdasht	necrosis	0.05^{ns}	-0.100^{ns}	0.82^{**}
	nAUDPC	1.30^{**}	-0.200^{ns}	6.53^{**}
	pycnidia	0.03^{ns}	-0.030^{ns}	0.25^{**}
	pAUDPC	0.32^{ns}	-0.240^{ns}	1.80^{**}
BOBWHITE#1/FENGKANG × Moghan3	necrosis	0.18^{**}	1.990^{**}	0.52^{**}
	nAUDPC	2.20^{**}	2.100^{**}	5.90^{**}
	pycnidia	0.04^{ns}	-0.002^{ns}	0.18^{ns}
	pAUDPC	0.60^{**}	-0.110^{ns}	0.50^{ns}

*** and ** Significant at the 0.05 and 0.01 levels of probability.**
ns: not significant.

Table 8. Estimates of components of additive (D), dominance (H) and environmental (E) variances, degree of dominance mean, dominance deviation, degree of dominance ([h]/[d]) and potency ratio (PI) for different traits in four crosses.

Crosses	Traits	D	H	F	EW	√H/D	F/√H.D	[h]/[d]	PI
Morvarid×Tajan	necrosis	0.030	-0.03	0.000	0.009	1.00	0.00	28.00	1.74
	nAUDPC	-0.950	0.16	0.120	1.090	0.40	0.30	19.01	1.73
	pycnidia	0.050	-0.06	0.010	0.010	1.10	0.20	32.40	10.72
	pAUDPC	4.180	-4.77	0.450	0.480	1.10	0.10	21.10	2.05
BOBWHITE#1/FENGKANG × Koohdasht	necrosis	0.030	-0.03	0.000	0.010	1.00	0.00	12.50	1.13
	nAUDPC	0.950	-4.86	0.320	1.490	2.30	0.15	5.92	0.88
	pycnidia	0.000	-0.01	0.000	0.003	0.00	0.00	10.22	0.07
	pAUDPC	0.140	-0.12	0.050	0.180	0.90	0.40	2.10	0.30
BOBWHITE#1/FENGKANG ×Moghan3	necrosis	0.031	-0.04	-0.003	0.010	1.13	0.10	-7.10	6.80
	nAUDPC	1.200	-0.79	-0.040	0.430	0.81	0.04	18.60	5.50
	pycnidia	0.000	0.00	0.002	0.003	0.00	0.00	2.20	0.30
	pAUDPC	-0.060	-0.02	0.057	0.130	0.60	1.90	-2.57	0.12

Table 9. Estimates of heterosis (MP), heterobeltiosis (BP) and inbreeding depression (ID) for different traits in three crosses.

Crosses	Traits	MP	BP	ID
Morvarid×Tajan	necrosis	-0.130**	-0.060 ns	0.180**
	nAUDPC	-0.130ns	-0.040 ns	0.070**
	pycnidia	-0.104*	-0.090 ns	0.180**
	pAUDPC	-0.080**	-0.040 ns	0.150**
BOBWHITE#1/FENGKANG × Koohdasht	necrosis	-0.180ns	-0.024 ns	0.360**
	nAUDPC	-1.100 ns	0.015 ns	0.160 ns
	pycnidia	-0.004 ns	0.060 ns	0.070 ns
	pAUDPC	0.015 ns	0.040 ns	0.300 ns
BOBWHITE#1/FENGKANG × Moghan3	necrosis	-0.200**	-0.150**	0.280**
	nAUDPC	-0.070 ns	-0.060 ns	0.140**
	pycnidia	-0.004 ns	0.070 ns	0.050 ns
	pAUDPC	-0.007 ns	0.040 ns	0.002 ns

**** Significant at the 0.01 level of probability.**
ns: not significant.

Table 10. Estimates of broad-sense (h2b) and narrow-sense heritabilities (h2n) for traits in three crosses.

Crosses	Trait	h^2_b	h^2_n
Morvarid × Tajan	necrosis	0.99	0.002
	nAUDPC	0.98	0.005
	pycnidia	0.96	0.430
	pAUDPC	0.98	0.004
BOBWHITE#1/FENGKANG × Koohdasht	necrosis	0.98	0.010
	nAUDPC	0.94	0.050
	pycnidia	0.94	0.020
	pAUDPC	0.64	0.200
BOBWHITE#1/FENGKANG × Moghan3	necrosis	0.77	0.030
	nAUDPC	0.89	0.005
	pycnidia	0.54	0.140
	pAUDPC	0.57	0.130

DISCUSSION

Resistance to Septoria tritici blotch is controlled by additive, dominance and epistatic gene action, but the role of the dominance gene effect is greater than the others. Estimated narrow-sense heritability for the crosses was 57-99%, suggesting that resistance to STB could not be readily selected for in early segregating generations. Also, estimates of the degree of dominance mean, dominance deviation and potency ratio revealed the role of dominance and over-dominance effects in the heritability of STB resistance. Considering the role of dominance effects and dominance × dominance interaction in controlling these traits, it is suggested that selection be delayed until later generations for improving resistance to Septoria trtici blotch.

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