

Original Research Article

Gene effects of resistance to *Septoria tritici* in durum wheat under water stress level

Running title: Relationship between gene effects and water stress level in resistance to *septoria tritici* in durum

Abstract

Gene effects and narrow-sense heritability estimates for components of resistance to *Septoria tritici* Blotch (STB) assessed under three water stress levels were evaluated using two populations derived from crossing partial-resistant durum wheat inbred lines Agili and Jneh Kottifa and susceptible inbred lines Karim 80 and Razzek by the generation mean analysis. Populations included the resistant parents, susceptible parents, F₁ and F₂ generations and backcrossed BC₁Pr and BC₁Ps generations. The presence of an epistatic effect was dependent upon water stress level. In the absence of water stress, only additive and dominance effects were induced for the two populations and the two STB isolates. Therefore, in the presence of water stress levels, the variation of generation means was adequately explained by additive, dominance and digenic epistatic effects for the two populations. The change in the mode of the gene effect of STB resistance inheritance depending upon water stress level complicated the procedure of incorporating genes of resistance in rentably cultivars. In the absence of water stress, selection would be simpler, because only additive and dominance effects were implicated. However, the genetic mechanism will not be stable when the plant experiences water stress. For cultivars designed for exploitation in a wide range of environmental conditions, selection under water stress is suggested for the stability of resistance to STB. Narrow-sense heritability estimates varied between combinations of crosses–isolates–water stress levels and ranged from 51 to 72 %.

Keywords: durum wheat, , gene effects, stability, water stress

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1. Introduction

Septoria tritici Blotch (STB) caused by the ascomycete *Mycosphaerella graminicola* (Fuckel) J. Schröt in Cohn.(anamorph: *Septoria tritici* Roberge in Desmaz), is an important disease in many areas where wheat is grown (1,2,3). It can cause considerable yield losses of up to 50 % in the wheat growing areas with severe infections (4). In Tunisia, durum wheat is the primary cultivated crop and occupies more than half of the area dedicated to cereal crops (5), with biotic and abiotic stresses the major factors severely affecting crop production.

Resistance to STB is of high relevance in wheat breeding; and is quantitatively inherited and controlled by multiple genes (6,7,8). Both additive and dominance effects (9) play a significant role in the expression of resistance, and epistasis also has a substantial effect (10).The environmental problem caused by continuous use of chemicals for control of fungal makes breeding for resistance to STB an essential approach in managing this important foliar disease of wheat. The genetic architecture of complex traits consists of not only the action of genes in single loci but also the inter-locus interactions and gene $\times$ environment interactions. The complexity of the genetic architecture can be mainly attributed to epistasis, which plays a significant role in heterosis, inbreeding depression, adaptation, reproductive isolation and speciation (11). The efficiency of any methodology of selection is associated with the unbiased estimates of the additive genetic effect (12).The lack of empirical work on epistasis, in part, reflects the difficulty in the quantification of epistasis (13, 14). Nonetheless, many studies that attempt to identify the genetic basis of quantitative traits ignore the possibility that loci interact (15), as well as the effects of environment on the expression of gene effects and their interactions with the genetic background. It is important to note that most models of inheritance are simplifications of complex genetic phenomena. Epistasis is a significant component in the inheritance of quantitative characters, and geneticists must face the reality of complex genetic systems. A better understanding of the relative importance of water stress in genetic

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components of resistance to STB would potentially lead to the more efficient development of resistant varieties and deployment of germplasm resources. Therefore, the objective of the present research was to evaluate the role of water stress in the variation of gene effects in resistance to STB in durum wheat.

2. Materials and methods

2.1 Plant materials

Parental lines were selected depending on their differential reactions to STB. The partially resistant parents (Pr) were Agili and Jneh Kottifa, and susceptible parents (Ps) were Karim 80 and Razzek. F₁ and F₂ generations and BC₁Pr and BC₁Ps backcross generations from the crosses of Karim 80 × Agili and Razzek × Jneh Kottifa were used in this study. Plants were grown in a randomized complete block design with two replications. Two seeds of each genotype were sown in 1-L pots containing loam substrate with three water stress levels (LS0, LS1 and LS2 representing respectively 100 %, 70 % and 50 % of field capacity and placed in a growth chamber at 20 ± 2 °C with a 12-h photoperiod. Pots were weighed, and water was added to bring the pots to the required saturation if the water level was more than 10 % below the required level.

2.2 Pathogen production and disease evaluation

Two isolates (ISTB1 and ISTB2) of STB were collected from infected durum wheat plants from the northwest of Tunisia. Conidia of each isolate were streaked on PDA (39 g of potato dextrose agar per 1 L) Petri plates. Then the plates were placed in the growth chamber at 20 ± 2 °C with a 12-h photoperiod. When the edge of the pink colony began to darken, the conidia were ready to harvest. A conidial suspension was prepared using equal quantities of the two isolates, filtered through two layers of cheesecloth and adjusted to 10⁶ conidia mL⁻¹ using a hemocytometer. Plants (two-leaf stage) were inoculated evenly by spraying the fungal conidia

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suspensions (40 mL/100 plants) onto leaves with an atomizer. After inoculation, the plants were covered with black polyethylene film for 72 h; and 21 days later the disease symptoms were assessed.

Disease ratings of all leaves of each plant were recorded on a 1–9 scale, based on the infection response (amount of necrosis and chlorosis) and density of pycnidia (16). The scale was defined as follows: (1) no visible symptoms, and the leaves remained green and healthy; (2) a few chlorotic lesions were present, and the infection site was a tan-coloured spot; (3) extensive chlorotic lesions were present, lesions occasionally had necrosis at the infection; (4) extensive chlorotic lesions merged with each other, and individual lesions were identified by initial infection sites, and necrosis and chlorosis both existed on the leaf; (5) lesions were fully merged, and more than half of the leaf was desiccated by necrosis; (6) the entire leaf was desiccated, and water-soaked lesions occupied the entire leaf; (7) a few pycnidia were visible on the infected sites, and < 30 % of the leaf was occupied by pycnidia-covered lesions that remain dry and green; (8) pycnidia occupied 50–70 % of the leaf, and infected sites were dry yet still green; and (9) the entire leaf was covered by pycnidial lesions, and the leaf was dry yet still green. The number of plants evaluated varied depending on the generation and was greater in generations with greater segregation, such as F₂, BC₁Pr, and BC₁Ps.

2.3 Statistical analyses

Before analysis, transforming the data by log, square root, arc-sine, and arc-sine of the square root of the variable had no effect on data distribution or in removing epistatic effects. Analysis of variance by population, stress level and isolate using general linear model (GLM) procedures (17) indicated that the replication and generation × replication effects were not significant. Therefore, generation means analysis was conducted without adjusting data for replication.

2.3.1 Gene effects

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107 Generation means analysis was determined separately for each combination of cross–isolate–
 108 water stress level as follows: Weighted least squares regression analyses were used to solve for
 109 mid-parent [m] pooled additive [d], pooled dominance [h] and pooled digenic epistatic ([i], [l]
 110 and [j]) genetic effects, following the models and assumptions described in Mather and Jinks
 111 (18). A simple additive-dominance genetic model contains only the m, d and h effects was
 112 tested first, using the joint scaling test described in Rowe and Alexander (19). Adequacy of the
 113 genetic model was assessed using a chi-square goodness-of-fit statistic derived from deviations
 114 from this model. If statistically significant at $p < 0.05$, the genetic models containing digenic
 115 epistatic effects were then tested until the chi-square statistic was non-significant.

116 2.3.2 Heritability

117 Homogeneity of variances of non-segregating generations was tested using Bartlett's test (20),
 118 and when the variances were heterogeneous, the environmental variance (V_E) was replaced by
 119 an adequate number of separate parameters and pooled to produce a single environmental
 120 variance. Additive, dominance and environmental variance components were estimated using
 121 the maximum likelihood method with the observed variance of the six basic generations being
 122 used as the initial weights [$df/(2 \times S^2)$] until the chi-squared test value reached a minimum (21).
 123 Narrow-sense heritability was calculated as follows: $h^2_n = V_A/(V_A + V_D + V_E)$. Where V_A is the
 124 additive genetic component of variance, V_D the dominance genetic component of variance and
 125 V_E the environmental variance (22).

126 3. Results

127 Comparison of means using Duncan's multiple range test ($\alpha = 0.05$), indicated significant
 128 differences between generations in all combinations of cross–isolate–water stress level. As
 129 expected, the two parental lines in each cross were consistently different for the three
 130 experimental water levels (Table 1).
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132 The results of generation mean analysis revealed that in the absence of water stress (LS0), the
133 three-parameter model was sufficient to explain the genetic variation of generation means of
134 resistance to STB in the four combinations of cross-isolate and the probability of goodness-of-
135 fit varied from 34 to 89 %. Additive (d) type effect was significant, negative and more
136 pronounced than dominance (h) type effect. However, in the presence of water stress (LS1 and
137 LS2) the additive-dominance model did not adequately fit for all combinations, and so the
138 digenic epistatic model was applied and found appropriate. The probability of goodness-of-fit
139 of these models ranged from 27 to 99 %. In the majority of cases, dominance (h), additive ×
140 additive (i) and dominance × dominance (l) types' effects (when significant) contributed more
141 to the control of resistance than additive effects. In all cases additive and dominance effects
142 (when significant) were negative. The additive × dominance (j) effect was significant only in
143 the cross Karim 80 × Agili with isolate ISTB1, where the additive × additive (i) effect was not
144 significant (Table 2).

145 Variance components were estimates and used to calculate narrow-sense heritability (h^2_n) for
146 both crosses and isolate–water stress level combinations. For all cases, the additive variance
147 effect was significant and ranged from 2.15 to 11.45 in the cross Karim 80 × Agili, and from
148 3.45 to 7.33 in the cross Razzek × Jneh Kottifa. The dominance variance effect was smaller
149 than the additive variance and ranged from 0.8 to 4.13 in the cross Karim 80 × Agili, and from
150 0.55 to 2.86 in cross Razzek × Jneh Kottifa. Environmental variance estimates were smaller
151 than additive and dominance variances estimates and ranged from 0.36 to 1.12 in the two
152 crosses. Under the three levels of water stress, h^2_n was consistent between crosses and STB
153 isolates with a range from 51 to 72% (Table 3).

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155 4. Discussion

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Significant differences were observed among generation means for resistance to STB in all combinations of cross–STB isolate–water stress level. This indicates that the magnitude of differences in the two populations was sufficient for selecting genotypes to improve resistance to STB in presence and absence of water stress. Plant genetic adaptability is of primary importance in many aspects of genetic research and is a particular priority in the study of the main crops grown in a wide range of environments. Therefore, the variation of inheritance of quantitative traits with environmental conditions has infrequently been considered and there is little knowledge on this topic. Recently Bnejdi et al. (23) reported that inheritance of resistance to salt on durum wheat at germination stage was dependent upon salt stress level. The variation of gene effects with water stress levels indicated that the mechanism of genetic control was not stable and was dependent upon environmental conditions. In the absence of water stress (LS0), only additive and dominance effects were implicated in the genetic control of resistance to STB. Therefore, both additive and non-additive (i.e., dominance and epistasis) effects were induced by water stress levels (LS1 and LS2). The results from our experiments indicate that epistasis contributed consistently and substantially in resistance to STB under water stress. The significance of epistatic effects was reported for resistance to yellowberry in durum wheat (5), for resistance to *Phytophthora nicotianae* in pepper (24) and resistance *Puccinia coronata* in oats (25).

The moderate to high (51 to 72%) h^2_n values indicate that genetic effects were larger than environmental effects. A similar result was reported by Bastam et al. (26). The general stability of h^2_n noted between water stresses levels indicated that resistance to STB was not under separate genetic control. However, a genetic interaction was evident only when plants were cultivated under water stress. Wolf et al. (27) reported that epistasis contributes to a variety of evolutionary phenomena, including adaptation, outbreeding depression, heterosis, the evolution of sex, reproductive isolation, founder effects and hybrid zone dynamics.

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Breeding for resistance to STB in the absence of water stress is simple and efficient. In fact, the lack of epistatic effects and the preponderance of additive compared to dominance **i** type effects facilitates the fixation of favourable genes of resistance in the productive cultivars desired. In the presence of water stress, the breeding for resistance to STB is complicated by the presence of epistasis. So, the fixation of genes of resistance and suitable interactions such as additive $\times$ additive (i) type effects requires an appropriate schema of selection. Evolution research has established that the assembly and maintenance of favourable epistatic combinations adapted to a particular environment is a primary mechanism of adaptation in various plant species (28).

5. Conclusion

Our results suggest that inheritance of resistance to STB in durum wheat depended upon water stress level. Thus, for the improvement of resistance of cultivars designed for exploitation in a wide range of environmental water stress, the choice of the environment in which the selection is made is critical. Indeed, in the presence of water stress the genetic interactions are solicited and are considered by breeders. However, in the absence of water stress, genetic interactions are not induced and not found in the procedure of selection. Thus, the stability of selected cultivars in different environmental conditions is not guaranteed. To conclude, the moderate to higher estimates of h^2_n indicated that genetic effects were larger than environmental effects, and under water stress, gene interaction were solicited. Therefore, breeding for resistance to STB requires an adequate choice of the parent. Also, the selection of environmental conditions in which the genetic components of resistance are incorporated in agronomically acceptable cultivars is suggested for the efficiency of any breeding strategy.

References

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1. Shipton W.A., W.R.J. Boyd, A.A Rosielle and B.I. Shearer.. The common Septoria disease of wheat. Bot Rev. 1971;37: 231–262.

2. King, J.E., J.E.E. Jenkins and W.A. Morgan. The estimation of yield losses in wheat from severity of infection by Septoria diseases. Plant Patho. 1983;32: 239–249.

3. Eyal Z., A.L. Scharen Perescott and M. Van Ginkel. The Septoria Disease of Wheat. Concepts and Methods of Disease Management. CIMMYT, D.F.P., Mexico. 1987;1–28.

4. Eyal Z., E. Levy. Variation in pathogenicity patterns of *Mycosphaerella graminicola* within *Triticum spp.* in Israel. Euphytica. 1987;36: 237–250.

5. Bnejdi F. and M. El Gazzah. Inheritance of resistance to yellowberry in durum wheat. Euphytica. 2008;163: 225–230.

6. Schnurbusch T., S. D. Paillard Fossati, G.M. Messmer Schachermayr, M. Winzeler and B. Keller. Detection of QTLs for Stagonospora glume blotch resistance in Swiss winter wheat. Theo Appl Genet. 2003;107: 1226–1234.

7. Uphaus J., E. Walker, M. Shankar, H. Golzar, R. Loughman, M. Francki and H. Ohm. Quantitative trait loci for resistance to Stagonospora glume blotch in wheat in the USA and Australia. Crop Sci. 2007;47: 1813–1822.

8. Kelm C., S.M.T. Ghaffary, M.S.H. Bruelheide Roder, S. Miersch, W.E. Weber, G.H.J. Kema and B. Saal. The genetic architecture of seedling resistance to *Septoria tritici* blotch in the winter wheat doubled-haploid population Solitar x Mazurka. Mol. Breed., 2011;DOI:10.1007/s11032-011-9592-8

9. Van Ginkel M. and L.A. Scharen () genitic systems with digenic epistasis. Genet Mol Biol 23: 883–892.

10. Bnejdi F., M. Saadoun, M. El Gazzah. Genetic adaptability of the inheritance of the resistance to different levels of aggressiveness of *Septoria tritici* isolates in durum wheat. Crop Prot. 2011b; 30:1280–1284.

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11. Yang J, J Zhu. Methods for predicting superior genotypes under multiple environments based on QTL effects. *Theor Appl Genet.* 2005; 110: 1268–1274.

12. Viana, J. M. S.. Components of variation of polygenic systems with digenic epistasis. *Genet. Mol. Biol.* 2000;23:883-892.

13. Whitlock M.C., P.C. Phillips, FBG Moore, SJ Tonsor . Multiple fitness peaks and epistasis. *Ann Rev Ecol a Syst.* 1995;26: 601–629.

14. Fenster C.B., L.F. Galloway and L. Chao. Epistasis and its consequences for the evolution of natural populations. *Trends Ecol Evol.* 1997;12: 282–286.

15. Carlborg O., C.S. Haley. Epistasis: Too often neglected in complex trait studies?. *Nature Rev Genet.* 2004;5: 618–625.

16. Zhang X, SD Haley, Y Jin. Diallel analysis of *Septoria tritici* Blotch resistance in winter wheat. In: Van Ginkel M, et al. (Eds.). *Septoria and Stagonospora diseases of cereals: a compilation of global research.* CIMMYT, Mexico, 1999; pp56-58.

17. SAS. SAS/STAT user's guide, v. 6. 4th ed. SAS Institute, Cary. 1990.

18. Mather K., J.L. Jinks. *Biometrical genetics*, 3rd edn. Chapman and Hall, London.1982.

19. Rowe K.E. and W.L. Alexander. Computations for estimating the genetic parameter in joint-scaling tests. *Crop Sci.* 1980;20: 109-110.

20. Bartlett M.S. Some examples of statistical methods of research in agriculture and applied biology. *J Roy Stat Society.* 1937;4: 137–183.

21. Lynch M. and B. Walsh. *Genetics and analysis of quantitative traits.* Sinauer Associates, Massachusetts. 1998.

22. Kearsey M.J., H.S. Pooni. *The genetical analysis of quantitative traits*, 1st edition edn. Chapman and Hall, London. 1996.

23. Bnejdi F., N. Rassa, M. Saadoun, M. Naouari and M. El Gazzah. Genetic adaptability to salinity level at germination stage of durum wheat. *Afr. J. Biotechnol* 2011a.1; 4400–4004.

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257 24. Bnejdi F., M. Saadoun, M.B Allagui, M. El Gazzah. Epistasis and heritability of resistance
258 to *Phytophthora nicotianae* in pepper (*Capsicum annuum* L). Euphytica, 2009;167:39–42.

259 25. Bnejdi F., I. Hammami , M.B. Allagui and M. El Gazzah. Epistasis and maternal effect in
260 resistance to *Puccinia coronate* Cda. f. sp. Avenae Eriks in oats (*Avena sp.*). Agr Sci China.
261 2010;9:101–105.

262 26. Bastam S.V., S.S Ramezanpour Soltanloo, H.S. Kia, M. Kalate, and M.H. Pahlevani
263 Inheritance of resistance to *septoria tritici* blotch (STB) in some Iranian genotypes of wheat
264 (*Triticum aestivum* L.). Int J Genet Mol Biol. 2010; 2: 034-042.

265 27. Wolf J, B Brodie, MJ Wade. Epistasis and the Evolutionary Process. Oxford University
266 Press, New York. 2000.

267 28. Allard, R.W. Genetic basis of the evolution of adaptedness in plants. Euphytica. 1996;92:
268 1–11.

270 **Table 1.** Leaf means scores for two isolates of STB (ISTB1 and ISTB2) in parents and offspring populations
271 from two crosses (Karim 80 × Agili and Razzek × Jneh Kottifa) assessed at three water stress levels

Cross	Water stress level/isolate	Population					
		Ps(20) ^y	BC1Ps(30)	F1(20)	F2(50)	BC1Pr(30)	Pr(20)
Karim 80 (Ps) × Agili (Pr)	LS0/ISTB1	3.84±1.35a	3.90±2.14a	3.00±1.43ab	3.42±2.65a	2.1±1.19bc	1.44±0.53c
	LS1/ISTB1	6.10±2.39a	6.00±1.84a	4.46±1.30bc	5.06±2.27b	3.76±1.48c	2.55±0.61d
	LS2/ISTB1	7.05±0.64a	6.58±2.62ab	4.5±1.09c	5.93±2.19b	4.34±1.09c	3.90±0.62c
	LS0/ISTB2	4.07±1.36a	3.87±1.82a	2.90±1.37b	3.19±2.26ab	1.91±1.04c	1.22±0.38c
	LS1/ISTB2	6.42±0.45a	5.82±1.65ab	4.58±0.64c	5.14±1.88bc	3.44±0.87d	2.47±0.18e
	LS2/ISTB2	7.08±0.64a	5.99±2.13b	5.06±1.54c	5.95±2.13b	4.21±0.98c	3.23±0.60d
Razzek (Ps) × Jneh Kottifa (Pr)	LS0/ISTB1	4.43±0.76a	4.04±1.68a	2.98±1.33b	2.83±1.99b	2.18±1.72b	1.48±0.45c
	LS1/ISTB1	6.90±0.43a	6.22±2.34ab	4.86±2.23cd	5.64±2.78bc	4.06±1.28de	3.65±0.70e
	LS2/ISTB1	7.62±0.75a	7.05±2.56a	4.93±1.43bc	5.86±2.34b	5.41±1.25b	4.02±0.65c
	LS0/ISTB2	5.16±0.82a	4.27±1.41b	3.17±0.96c	3.43±1.83c	2.25±1.18d	1.36±0.31e
	LS1/ISTB2	7.03±0.39a	5.77±1.65b	4.93±1.48c	5.18±1.94bc	3.45±0.71d	2.73±0.14d
	LS2/ISTB2	7.77±0.81a	6.28±2.27b	5.18±1.26c	5.92±2.20bc	4.14±1.01d	3.27±0.50d

272 Pr: partially resistant parents, (Ps): susceptible parents, F₁ (Pr*Ps) and F₂ (F₁*F₁) generations
273 and BC₁Pr (F₁*Pr) and BC₁Ps (F₁*Ps) backcross generations. (LS0, LS1 and LS2 representing
274 respectively 100%, 70% and 50% of field capacity). For each cross, means within a line, bearing different letters
275 differ (P<0.05). ^y Number of plants evaluated in each generation

Table 2. Estimates of additive, dominance and epistatic effects for resistance to two isolates of STB (ISTB1 and ISTB2) in two crosses (Karim 80 × Agili and Razzek × Jneh Kottifa) evaluated at three water stress levels

C cross	water stress level/isolate	Best-fit model						P value
		m	D	h	i	l	j	
Karim 80 (Ps) × Agili (Pr)	LS0/ISTB1	2.7±0.1**	-1.3±0.1**	0.4±0.3	-----	-----	-----	0.34
	LS1/ISTB1	-5.1±1.6**	-1.9±0.2**	0.2±3.8	-0.7±0.1**	-0.9±0.1**	-----	0.31
	LS2/ISTB1	7.3±0.6**	-1.5±0.1**	-2.8±0.7**	-----	-1.8±0.6**	-1.32±0.9*	0.99
	LS0/ISTB2	2.9±0.1**	-1.5±0.1**	0.1±0.2	-----	-----	-----	0.73
	LS1/ISTB2	7.7±1.8**	-1.6±0.1**	-5.6±3.1*	-2.5±1.1*	2.7±1.4*	-----	0.29
	LS2/ISTB2	4.1±1.6**	-1.7±0.1**	6.0±3.8*	1.6±0.6**	-5.2±2.3**	-----	0.77
Razzek (Ps) × Jneh Kottifa (Pr)	LS0/ISTB1	2.7±0.2**	-1.5±0.2**	0.2±0.3	-----	-----	-----	0.42
	LS1/ISTB1	6.9±1.3**	-2.0±0.1**	-4.7±2.8*	-2.4±1.2**	2.38±1.7*	-----	0.27
	LS2/ISTB1	8.4±1.4**	-1.9±0.1**	-6.5±3.2**	-3.3±1.4**	3.14±2.2*	-----	0.73
	LS0/ISTB2	3.3±0.1**	-1.2±0.1**	-0.04±0.2	-----	-----	-----	0.89
	LS1/ISTB2	7.4±1.3**	-2.2±0.1**	-6.2±2.8**	-2.47±1.3*	3.85±1.7**	-----	0.63
	LS2/ISTB2	8.2±1.4**	-2.2±0.1**	-6.2±3.1**	-2.7±1.4**	3.2±2.0**	-----	0.82

m, d, h, i, l, j = mean, additive, dominance, additive × additive, dominance × dominance and additive × dominance genetic effects, respectively. P = probability of adequateness of the model. *p<0.05 and **p<0.01 compared to zero. LS0, LS1 and LS2 representing respectively 100%, 70% and 50% of field capacity.

Table 3. Estimates of additive (V_A), dominance (V_D) and environmental variances (V_E) and narrow-sense heritability (h^2_n) for resistance to two isolates of STB (ISTB1 and ISTB2) in two durum wheat crosses (Karim 80 × Agili and Razzek × Jneh Kottifa) assessed at three water stress levels.

Cross	Water stress level / isolate	V_E	V_A	V_D	$\chi^2(3\ df)$	h^2_n
Karim 80 (Ps) × Agili (Pr)	LS0/ISTB1	1.02±0.18**	10.37±3.00**	4.13±1.65	Ns	0.66
	LS1/ISTB1	1.12±0.27**	5.16±2.49**	0.80±1.6**	Ns	0.72
	LS2/ISTB1	0.76±0.23**	6.63±2.17**	2.37±1.27**	Ns	0.67
	LS0/ISTB2	0.53±0.09**	2.15±0.02**	1.49±1.41	Ns	0.51
	LS1/ISTB2	0.46±0.20**	11.45±3.30**	4.01±1.82*	Ns	0.71
	LS2/ISTB2	0.96±0.25**	7.01±2.52**	2.17±1.51*	Ns	0.69
Razzek (Ps) × Jneh Kottifa (Pr)	LS0/ISTB1???	0.85±0.24**	7.33±2.28**	2.86±1.31	Ns	0.66
	LS1/ISTB1	0.36±0.19**	4.94±1.69**	1.53±1.03*	Ns	0.72
	LS2/ISTB1	0.75±0.22**	6.45±2.05**	2.45±1.2**	Ns	0.66
	LS0/ISTB2	0.56±0.21**	3.45±1.62**	0.55±1.05	Ns	0.75
	LS1/ISTB2	0.44±0.19**	5.94±1.72**	2.40±0.99**	Ns	0.67
	LS2/ISTB2	0.79±0.23**	6.98±2.16**	2.72±1.25**	Ns	0.66

df = degrees of freedom, calculated as the number of generations minus the number of estimated variance parameters

ns = non-significant. * $p < 0.05$ and ** $p < 0.01$ compared to zero.