Generation Mean Analysis and Heritabilities of Resistance to *Septoria tritici* in Durum Wheat

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ABSTRACT

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Thirteen durum wheats used in breeding programs in North Africa and the Middle East were crossed in all combinations except reciprocals. Backcrosses were made and generations P_1, P_2, F_1, F_2, BC_1, and BC_2 were tested as seedlings in the greenhouse. Seedlings were quantitatively inoculated with an isolate of *Septoria tritici* at the second-leaf stage, and the percentage of necrotic tissue of the first leaf was assessed 21 days later. Additive, dominance, and epistatic gene effects were estimated by a generation mean analysis on each of the 65 crosses. Significant additive and dominance gene effects were found in about one-half and one-third of the crosses, respectively. Estimates of broad-sense heritability ranged from 0 to 78%, with a mean of 38%. The proportion of variance explained by models generally involving only additive and dominance gene effects, was $R^2 \geqslant 0.88$. Thus, we concluded that epistatic effects were of minimal importance. The additive gene effects component was of prime importance, but the dominance component also was often significant. We found heritability estimates to be of an intermediate magnitude and thus conclude that selecting for resistance to *S. tritici* on a single-plant basis could be successful but probably slow.

Additional key words: quantitative inheritance, *Mycosphaerella graminicola*, Septoria tritici blotch, speckled leaf blotch.

Mycosphaerella graminicola (Fückel) Schroeter (anamorph: *Septoria tritici* Rob. ex. Desm.) incites the disease Septoria tritici blotch on wheat. Yield losses from slight to 60% have been attributed to natural infection (7,22). During the last 25 yr, attention to *S. tritici* on cereals has intensified. Reports of increased occurrence and disease levels have led to a prominent place for *S. tritici* in a number of research and crop improvement programs worldwide (3,6,7,13). Severe epidemics in the late 1960s and early 1970s have caused the disease to be considered one of prime importance in national wheat improvement programs in several North African and Middle Eastern countries bordering the Mediterranean Sea (5,20,25). More than half the world's durum wheat is grown in the Mediterranean region, where in a number of countries, durum wheat is cultivated on more than two-thirds of the wheat acreage.

Studies on the inheritance of resistance to *S. tritici* have largely concentrated on bread wheat (*Triticum aestivum*) (4,16,17,21,26). Often, seedlings were assessed for disease reactions. Brokenshire (2) has shown large positive correlations between seedling and adult plant reactions and between greenhouse and field disease responses. Most published research has emphasized the study of single genes.

The objective of the present study was to investigate the inheritance of resistance to *S. tritici* in durum wheats from Mediterranean origin using a quantitative genetic approach.

MATERIALS AND METHODS

Thirteen durum wheats used in breeding programs in North Africa and the Middle East were selected: Kyperounda, Badri, BD 2131, BD 2127, 65150-Lds, D 75-9-6B-5B-4B-10B, D 75-40-11B-4B-2B, Ben Bechir 79, Karim 80, Maghrebi 72, Etit 38, Volcani 447, and Zenati Bouteille. These cultivars and lines represent varying levels of disease resistance, yielding ability, agronomic desirability, local adaptation, and combining ability as parents.

The Tunisian *S. tritici* isolate, TUN 8204-1, was selected because it caused a wide range of infection levels on the cultivars involved, reproduced consistently on artificial media, and had an above-average ability to produce pycnidia in the leaf lesions under relatively dry greenhouse conditions.

The isolate was maintained on yeast-malt agar and increased in liquid yeast extract medium for inoculation purposes according to Krupinsky (12).

All possible crosses and related backcrosses between the first 10 cultivars and lines listed were made, except for reciprocals. F_2 generations were subsequently obtained. Thus, P_1, P_2, F_1, F_2, BC_1, and BC_2 generations for each cross were available for seedling testing in the greenhouse. Additionally, these six generations were produced for 20 other crosses involving the 10 cultivars and lines and the last three entries listed.

The six generations for each cross were planted in a square aluminum tray 21 × 21 × 6 cm; 5, 5, 5, 20, 7, and 7 seeds were sown for each of the six generations per replicate, respectively. The amount of seed obtained from the 65 crosses plus progenies varied, and thus, more replicates could be tested for certain crosses than

TABLE 1. Estimates, standard errors, and levels of significance of gene effects for the 45 nonreciprocal crosses possible among 10 durum wheat parents, plus the *P* value associated with the chi-square value of the deviations sum of squares for the model and the R^2 values for percent necrotic leaf area of durum wheat seedlings due to infection by *Septoria tritici* isolate TUN 8204-1

Cross	*m*	*a*	*d*	*aa*	*ad*	*dd*	*P*	R^2
Kyperounda ×								
Badri	84.3 ± 7.9***[z]	−16.7 ± 10.8	−72.7 ± 42.7	−78.8 ± 41.3$^+$	···	153.3 ± 65.7*	>0.95	1.00
BD 2131	64.3 ± 3.5***	10.5 ± 5.1$^+$	−115.1 ± 2.7***	−109.1 ± 27.4***	···	148.1 ± 57.0*	0.83	1.00
BD 2127	59.0 ± 5.4***	28.4 ± 8.3***	−2.3 ± 17.8	···	···	···	0.30	0.98
65150-Lds	64.9 ± 3.6***	−13.2 ± 6.8$^+$	22.0 ± 11.1$^+$	···	···	···	0.21	0.99
D75-9-6B-5B-4B-10B	45.6 ± 7.1***	5.7 ± 8.0	−9.0 ± 19.7	···	···	···	0.23	0.93
D75-40-11B-4B-2B	72.7 ± 3.2***	−8.1 ± 4.7	−10.3 ± 9.3	···	···	···	0.84	1.00
Ben Bechir 79	50.8 ± 5.7***	1.5 ± 6.8	−4.4 ± 11.9	···	···	99.7 ± 32.4**	0.29	0.98
Karim 80	51.7 ± 2.7***	15.1 ± 4.8**	15.9 ± 7.1*	···	···	···	0.41	0.99
Maghrebi 72	58.8 ± 3.9***	17.9 ± 7.3*	21.2 ± 10.9$^+$	···	···	···	0.23	0.96
Badri ×								
BD 2131	60.3 ± 13.7***	18.3 ± 59.3	−109.7 ± 168.1	−91.4 ± 131.8	···	199.4 ± 322.5	>0.95	1.00
BD 2127	50.4 ± 2.7***	14.9 ± 5.2*	9.3 ± 11.4	···	···	···	0.36	0.99
65150-Lds	89.6 ± 1.7***	−6.5 ± 3.4$^+$	−10.5 ± 10.5	···	···	···	0.87	1.00
D75-9-6B-5B-4B-10B	60.4 ± 2.9***	16.4 ± 8.0$^+$	−12.5 ± 12.3	···	···	···	0.08	0.97
D75-40-11B-4B-2B	68.4 ± 3.6***	−4.9 ± 5.1	−18.0 ± 11.5	···	···	···	0.57	1.00
Ben Bechir 79	63.5 ± 6.4***	20.9 ± 27.3	23.6 ± 55.1	···	···	···	0.64	1.00
Karim 80	45.3 ± 14.5*	29.6 ± 28.7	−18.0 ± 73.9	···	···	···	0.29	1.00
Maghrebi 72	63.3 ± 3.9***	27.1 ± 6.1***	18.3 ± 11.4	···	···	···	0.27	0.97
BD 2131 ×								
BD 2127	49.2 ± 3.2***	−11.1 ± 6.8	−31.2 ± 13.2*	···	···	···	0.24	0.96
65150-Lds	81.7 ± 3.4***	−39.7 ± 3.5***	−40.4 ± 15.8*	−45.5 ± 11.2***	···	···	0.16	0.99
D75-9-6B-5B-4B-10B	18.3 ± 1.8***	12.1 ± 5.5$^+$	−7.7 ± 4.6	···	21.3 ± 6.3**	···	0.08	0.93
D75-40-11B-4B-2B	52.4 ± 3.6***	−26.7 ± 6.2***	−9.7 ± 10.0	···	···	···	0.40	0.97
Ben Bechir 79	18.7 ± 3.6***	1.0 ± 6.9	−3.9 ± 13.2		···	···	0.68	0.97
Karim 80	21.3 ± 5.1***	0.6 ± 10.0	−29.0 ± 24.4	···	···	···	0.34	0.91
Maghrebi 72	21.8 ± 3.7***	−0.1 ± 8.8	−7.1 ± 9.4	···	···	···	0.49	0.95
BD 2127 ×								
65150-Lds	62.6 ± 3.7***	27.9 ± 6.5***	−2.5 ± 9.7	···	···	···	0.69	0.99
D75-9-6B-5B-4B-10B	31.8 ± 3.2***	0.6 ± 5.3	−25.6 ± 9.7*	···	···	···	0.64	0.96
D75-40-11B-4B-2B	66.6 ± 2.7***	27.7 ± 4.9***	−9.4 ± 6.9	···	···	···	0.13	0.98
Ben Bechir 79	45.3 ± 2.6***	−4.9 ± 2.9	−4.6 ± 6.7	···	···	67.9 ± 17.0***	0.36	0.99
Karim 80	32.7 ± 2.4***	3.6 ± 4.4	−17.3 ± 11.7	···	···	···	0.22	0.95
Maghrebi 72	49.9 ± 3.3***	−13.7 ± 6.7$^+$	−5.8 ± 11.3	···	···	···	0.32	0.97
65150-Lds ×								
D75-9-6B-5B-4B-10B	87.7 ± 8.5***	36.7 ± 8.8***	−162.9 ± 48.3**	−168.1 ± 40.6***	···	277.8 ± 76.7***	0.16	0.99
D75-40-11B-4B-2B	71.8 ± 4.4***	6.8 ± 7.4	7.9 ± 17.1	···	···	···	0.25	0.97
Ben Bechir 79	75.5 ± 2.9***	25.4 ± 5.3***	−13.4 ± 9.6	···	···	···	0.34	0.98
Karim 80	67.2 ± 4.7***	27.3 ± 6.7***	−2.4 ± 13.3	···	···	···	0.79	0.99
Maghrebi 72	97.4 ± 4.0***	21.4 ± 12.6	−6.4 ± 13.5	−22.0 ± 16.0	···	···	0.30	1.00
D75-9-6B-5B-4B-10B ×								
D75-40-11B-4B-2B	51.5 ± 4.2***	−21.2 ± 6.0***	−29.9 ± 12.6*	···	···	···	0.11	0.97
Ben Bechir 79	40.7 ± 6.7***	8.4 ± 9.5	−88.3 ± 38.1*	−90.4 ± 34.9*	···	172.7 ± 60.2*	0.88	1.00
Karim 80	36.4 ± 2.8***	−3.5 ± 4.7	−13.6 ± 8.1	···	···	···	0.15	0.93
Maghrebi 72	25.4 ± 3.9***	4.4 ± 7.6	−11.5 ± 20.9	···	···	···	0.16	0.92
D75-40-11B-4B-2B ×								
Ben Bechir 79	73.6 ± 5.9***	17.4 ± 11.7	12.5 ± 12.2	···	···	···	0.21	0.99
Karim 80	56.2 ± 6.0***	28.0 ± 12.3*	−16.0 ± 14.1	···	···	···	0.71	0.99
Maghrebi 72	81.3 ± 7.6***	31.0 ± 5.9***	−157.8 ± 47.6**	−161.9 ± 46.1**	···	278.2 ± 79.0**	0.74	1.00
Ben Bechir 79 ×								
Karim 80	53.7 ± 2.8***	8.6 ± 7.5	−13.4 ± 15.4	···	···	···	0.35	0.99
Maghrebi 72	30.0 ± 4.1***	1.8 ± 6.1	−5.4 ± 11.6	···	···	···	0.46	0.92
Karim 80 ×								
Maghrebi 72	52.8 ± 5.6***	−4.7 ± 4.4	−118.8 ± 27.3***	−105.8 ± 26.1***	···	168.1 ± 38.6***	>0.95	1.00

[z] +, *, **, ***: Significant at the 0.10, 0.05, 0.01, and 0.005 level of probability, respectively.

for others. The minimum number of replicates used for a few crosses was three and the maximum was six.

After full emergence of the first leaf and partial emergence of the second leaf (10–12 days after seeding), the seedlings were inoculated using the quantitative inoculation method described by Eyal and Scharen (9) with a suspension containing 10^7 spores per milliliter. Twenty-one days after inoculation, the infection of the first leaf of each seedling was visually assessed as percent necrotic tissue of the total leaf area (9).

A generation mean analysis was done on each of the 65 crosses to estimate additive, dominance, and epistatic gene effects, following Hayman's model (11). These gene effects were defined in Gamble's notation (10) as m = mean using the F_2 as a reference, a = pooled additive gene effects, d = pooled dominance gene effects, aa = pooled additive × additive epistatic gene effects, ad = pooled additive × dominance epistatic gene effects, and dd = pooled dominance × dominance epistatic gene effects.

The various generation means did not have equal variances and were therefore weighted using the reciprocals of the squared standard errors of the generation means (15). A weighted least squares analysis was used as described by Rowe and Alexander (19). The chi-square test for goodness of fit was applied to find the model best explaining the observed means. The weighted generation means were regressed on the variable subsets *m, a,* and *d* and subsequently on all seven extensions of this basic model, involving one or more of the epistatic effects, *aa, ad,* and *dd*. Assuming a chi-square distribution (15), the subset with the highest P value for the sum of squares deviations from regression best fits the data of a particular cross. Initially, such subsets were selected for all crosses. Subsequently, if simpler models were not significantly different at the 5% level of probability from the more complex models, the former were selected as sufficiently explanatory. The R^2 values of the model, standard errors, and significance levels of the individual gene effects were calculated according to Snedecor and Cochran (24).

Variances among plants within each generation were calculated on a single-plant basis and functioned as estimates of generation variance in the equations for broad-sense heritability and its standard error. Broad-sense heritability was estimated using Allard's approach (1):

$$h^2_{bs} = \frac{\sigma^2 F_2 - (\sigma^2 P_1 + \sigma^2 P_2 + \sigma^2 F_1)/3}{\sigma^2 F_2}$$

McNew (*personal communication*) supplied an equation to estimate the standard error of the h^2_{bs} equation:

$$\text{SE } h^2_{bs} = \left\{ \frac{1}{9} * \frac{2}{(\sigma^2 F_2)^2} * \left(\frac{(\sigma^2 P_1 + \sigma^2 P_2 + \sigma^2 F_1)^2}{dfF_2} + \frac{(\sigma^2 P_1)^2}{dfP_1} + \frac{(\sigma^2 P_2)^2}{dfP_2} + \frac{(\sigma^2 F_1)^2}{dfF_1} \right) \right\}^{1/2}$$

dfP_1, dfP_2, dfF_1, and dfF_2 = Degrees of freedom of the P_1, P_2, F_1, and F_2 populations, respectively.

RESULTS

The simplest subsets of variables *m, a, d, aa, ad,* and *dd* showing the best fit were selected for each cross and are presented in Tables 1 and 2. The fit of the selected models was excellent in almost all cases as indicated by the high P and R^2 values. In very few cases were epistatic gene effects required to explain differences among generation means. Because the generation means in a number of crosses did not vary greatly, presumably due to similar components of resistance in the parents, significant gene effects could not always be detected.

Significant additive gene effects occurred more frequently than dominance gene effects. At the 10% level of significance, more than 50% of the crosses showed significant additive gene effects. In about one-third of the crosses, dominance effects were important. Dominance effects were negative in most cases. The negative sign associated with the dominance components indicated that, in these hybrid combinations, disease levels could be decreased relative to the midparent. The additive × additive epistatic gene effects operated in reducing disease infection levels, whereas the dominance × dominance component enhanced necrotic leaf area.

TABLE 2. Estimates, standard errors, and levels of significance of gene effects for 20 additional crosses involving Etit 38, Volcani 447, and Zenati Buouteille plus the P value associated with the chi-square value of the deviations sum of squares for the model and the R^2 values for percentage necrotic leaf area of durum wheat seedlings due to infection by *Septoria tritici* isolate TUN 8204-1

Cross	*m*	*a*	*d*	*aa*	*ad*	*dd*	*P*	R^2
Badri ×								
Etit 38	64.0 ± 28.2*[z]	4.5 ± 6.6	−7.0 ± 57.5	...	...	...	>0.95	1.00
Zenati Bouteille	69.3 ± 3.0***	−1.7 ± 5.7	−9.7 ± 12.8	...	...	...	0.50	1.00
BD 2131 ×								
Etit 38	60.3 ± 4.5***	−30.8 ± 8.5***	10.6 ± 12.6	...	...	...	0.49	0.98
Zenati Bouteille	40.7 ± 2.5***	−30.2 ± 4.4***	−17.3 ± 5.8**	...	...	...	0.26	0.97
65150-Lds ×								
Etit 38	68.2 ± 5.1***	22.8 ± 7.2**	−14.1 ± 12.0	...	...	...	0.77	0.99
Zenati Bouteille	77.1 ± 3.6***	13.3 ± 6.2*	−23.3 ± 16.6	...	...	...	0.80	1.00
D75-9-6B-5B-4B-10B ×								
Etit 38	64.5 ± 4.8***	−10.3 ± 6.0	−2.5 ± 13.5	...	...	...	0.49	0.98
Volcani 447	29.4 ± 3.1***	6.9 ± 4.8	−19.6 ± 9.6[+]	...	...	...	0.28	0.88
Zenati Bouteille	67.8 ± 7.4***	−20.1 ± 8.1*	−112.2 ± 44.0*	−105.8 ± 36.0*	...	181.4 ± 71.4*	0.75	1.00
D75-40-11B-4B-2B ×								
Volcani 447	50.9 ± 5.2***	30.7 ± 7.4***	−14.3 ± 20.0	...	...	...	0.33	0.99
Zenati Bouteille	66.4 ± 5.0***	0.7 ± 9.9	−38.5 ± 11.9**	...	...	...	0.37	1.00
Ben Bechir 79 ×								
Etit 38	45.1 ± 5.3***	25.5 ± 10.6*	2.4 ± 22.6	...	...	...	0.36	0.95
Volcani 447	52.6 ± 4.0***	19.6 ± 10.9[+]	−145.9 ± 40.8***	−122.5 ± 35.4***	...	178.2 ± 76.7*	0.86	1.00
Zenati Bouteille	59.2 ± 3.3***	−20.0 ± 6.4**	−5.4 ± 10.9	...	...	...	0.53	0.99
Karim 80 ×								
Etit 38	65.3 ± 3.4***	−13.9 ± 10.7	−110.7 ± 47.9*	−105.2 ± 40.5*	...	148.1 ± 92.9	>0.95	1.00
Volcani 447	39.0 ± 3.4***	−7.2 ± 7.1	−31.6 ± 8.9***	...	...	...	0.79	0.98
Zenati Bouteille	54.9 ± 4.7***	−21.3 ± 7.3*	−30.9 ± 14.9[+]	...	...	...	0.56	0.99
Maghrebi 72 ×								
Etit 38	39.8 ± 2.8***	−9.7 ± 4.6[+]	−28.4 ± 9.5*	−24.5 ± 8.6*	...	...	0.81	1.00
Zenati Bouteille	63.2 ± 3.3***	−20.8 ± 8.0*	−5.0 ± 15.3	...	...	...	0.20	0.98
Etit 38 ×								
Volcani 447	50.0 ± 6.1***	14.4 ± 3.6***	−121.7 ± 28.7***	−110.7 ± 27.1***	...	179.4 ± 39.1***	0.02	0.96

[z] +, *, **, ***: Significant at the 0.10, 0.05, 0.01, and 0.005 level of probability, respectively.

TABLE 3. Broad-sense heritabilities (%) and standard errors (%) of reactions to infection by *Septoria tritici* isolate TUN 8204-1 for a 10 × 10 diallel plus 20 additional crosses of durum wheat cultivars

Parents		Cultivar code											
Code	Cultivar	2	3	4	5	6	7	8	9	10	11	12	13
1	Kyperounda	52 ± 14	66 ± 9	33 ± 18	52 ± 17	69 ± 11	23 ± 22	14 ± 23	44 ± 16	36 ± 17	···(1)[a]	···	···
2	Badri	···	77 ± 7	75 ± 7	27 ± 25	56 ± 12	52 ± 15	73 ± 8	49 ± 19	50 ± 13	28 ± 22	···	0 ± 35
3	BD 2131	···	···	70 ± 8	27 ± 18	62 ± 13	18 ± 22	0 ± 37	33 ± 19	0 ± 44	49 ± 16	···	66 ± 10
4	BD 2127	···	···	···	34 ± 22	25 ± 22	0 ± 33	22 ± 19	29 ± 16	42 ± 15	···	···	···
5	65150-Lds	···	···	···	···	64 ± 14	78 ± 8	3 ± 26	34 ± 23	0 ± 2	67 ± 11	···	47 ± 15
6	D75-9-6B-5B-4B-10B	···	···	···	···	···	49 ± 14	24 ± 24	53 ± 13	36 ± 18	23 ± 22	2 ± 26	29 ± 20
7	D75-40-11B-4B-2B	···	···	···	···	···	···	0 ± 51	0 ± 53	46 ± 23	···	49 ± 14	10 ± 30
8	Ben Bechir 79	···	···	···	···	···	···	···	20 ± 17	21 ± 25	46 ± 17	69 ± 8	58 ± 11
9	Karim 80	···	···	···	···	···	···	···	···	51 ± 12	41 ± 20	59 ± 10	19 ± 30
10	Maghrebi 72	···	···	···	···	···	···	···	···	···	22 ± 25	···	60 ± 11
11	Etit 38	···	···	···	···	···	···	···	···	···	···	37 ± 18	···
12	Volcani 447	···	···	···	···	···	···	···	···	···	···	···	···
13	Zenati Bouteille	···	···	···	···	···	···	···	···	···	···	···	···

[a](1) = No filial and/or related generations available.

The broad-sense heritability estimates and their standard errors are presented in Table 3. Because the additive variance is not clearly separated from the total genetic variance in the numerator of the broad-sense heritability equation, as is the case in the narrow-sense heritability formula, the former will generally have a larger value than the latter. If all of the genetic variance is additive, then both estimates of heritability are identical. Thus, the broad-sense heritability establishes an upper limit for the narrow-sense heritability.

Estimates of broad-sense heritability varied between 0 and 78% with a mean of 38%.

DISCUSSION

The proportion of variance explained by the models used was high ($R^2 \geq 0.88$). We concluded that overall epistatic effects were of minimal importance. Some gene effects may have been canceled due to the simultaneous presence of positive and negative components. The additive gene effects component, *a*, was of prime importance, but the dominance component, *d*, was also often significant and reduced disease levels.

One consequence of the different gene effects on the choice of a breeding strategy is that line selection following repeated self-fertilization would be expected to raise levels of resistance due to the predominant additive gene effects. The occasional additive × additive epistatic components increasing resistance are likewise fixable in pure inbred lines. Dominance effects may be exploited but only if hybrid durum wheat is the objective of the breeding program. In a few crosses, additive × dominance and dominance × dominance gene effects were present. These effects were signed positive and thus contributed to increased disease levels. Because neither the simple nor the epistatic dominance gene effects can be fixed in homozygous lines and operate in opposing directions, it may be necessary for selection pressure to be lenient in early selfed generations and be intensified when homozygosity is approached.

It has been observed that heritability estimates increase with larger disparities between the parents and are otherwise dependent on experimental design (23). In this study, 13 cultivars representing a broad series of disease reaction levels were intercrossed in an essentially, albeit incomplete, diallel fashion and tested in replicated trials. Therefore, we believe that a reasonable order of magnitude for the heritability of reaction to infection by *S. tritici* for the seedling material studied has been established. The heritabilities averaging 38% were, in general, somewhat lower than those estimated for three spring wheat crosses by Rosielle and Brown (17), which varied between 57 and 68%. The values here obtained, plus the knowledge that additive gene effects are predominant but nonadditive effects are sometimes present, would predict that selecting for resistance to *S. tritici* on a single-plant basis could be successful, be it at an intermediate pace. Practical experience in several successful wheat improvement programs around the world has given evidence that indeed incorporation of resistance to *S. tritici* into a high-yielding background is a slow process but that resistance levels can be increased (8,13,14,18).

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