# Host-Pathogen Relationships of Wheat and *Septoria tritici*

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## ABSTRACT

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A total of 65 crosses were made among 13 durum wheats obtained from crop improvement programs in North Africa and the Middle East. The parental generations, the $F_1$'s, and the $F_2$'s were evaluated for their reaction to a highly pathogenic isolate of *S. tritici* from Tunisia. Means and variances of these generations were used to establish the number of effective factors governing reaction to *S. tritici* in the parents when crossed. The values varied between 1 and 68, with the majority less than 20. Transgressive segregation was also observed. In separate trials, the parents were tested for reaction to 34 isolates of *S. tritici* from seven countries in the Mediterranean area. Differences due to cultivars and isolates were highly significant. The cultivar × isolate interaction component was relatively very small and not significant. The major role of fungal host-species specialization as opposed to cultivar specialization is proposed. Isolates would thus vary in aggressiveness and cultivars in horizontal resistance.

*Mycosphaerella graminicola* (Fückel) Schroeter (anamorph, *Septoria tritici*), causal organism of Septoria tritici blotch on wheat may cause losses of more than 60% of the grain yield (21).

Increased occurrence has elevated *S. tritici* to a pathogen of worldwide importance. Crop improvement programs are stepping up efforts to develop resistant germ plasm (2,6,12). Severe epidemics have made resistance an absolute requirement for advanced wheat lines in national crop improvement programs, especially in the Mediterranean basin (5,18).

Most published research on the reaction of wheat cultivars to *S. tritici* has involved the use of either a single isolate or a bulk of isolates for inoculation. However, the reaction of several diploid, tetraploid, and hexaploid *Triticum* species was studied with seven separate isolates of *S. tritici* (27), while Eyal et al (9) evaluated 35 wheat and triticale cultivars using 97 isolates of *S. tritici* originating from 22 countries. Since the report of physiologic specialization by Eyal et al (8), it would seem essential that any genetic study of resistance to Septoria tritici blotch be executed with a number of separately collected isolates. In the case of applied studies, isolates of regional importance should probably be used.

Resistance varies among *Triticum* species, with durum wheat often being reported as relatively resistant (1,7,). Durum wheats tested in Tunisia were, however, more susceptible to the local isolates of *S. tritici* than the bread wheat entries (5).

Depending on the size of the experiment and the approach used, the number of effective factors for resistance to *S. tritici* estimated varied between one and eight (4,9,17,25,27). The presence of dominant, recessive, incomplete dominant, and modifying genes has been reported (21).

The objective of this research was to study the nature of the host-pathogen relationship between durum wheat and *S. tritici* and to estimate the number of effective factors involved in disease expression.

## MATERIALS AND METHODS

**The host.** Durum wheat cultivars used in the Mediterranean region were obtained from the Tunisian crop improvement program through Dr. H. Ketata. Ten entries represented wide differences in resistance to *S. tritici*, yield potential, agronomic traits, adaptation, and combining ability. Cultivars were Kyperounda, Badri, BD 2131, BD 2127, 65150-Lds, D75-9-6B-5B-4B-10B, D75-40-11B-4B-2B, Ben Bechir 79, Karim 80, and Maghrebi 72. Three additional durum wheat cultivars that have been used in breeding programs in the Mediterranean basin were also tested: Etit 38, Volcani 447, and Zenati Bouteille.

Crosses were made as a diallel among the first 10 durum wheat cultivars listed, excluding reciprocals. An additional 20 crosses were produced involving these 10 entries and Etit 38, Volcani 447, and Zenati Bouteille. $F_2$ generations were obtained.

**The pathogen.** A total of 34 isolates originating from Tunisia, Turkey, Israel, Syria, Portugal, Italy, and Spain were studied. Fourteen isolates were collected from durum wheats, four from bread wheats, and 16 from unknown wheat hosts.

Each isolate was cultured from spores obtained from a single pycnidium on a leaf and maintained on solid yeast-malt agar under constant light and 15 C. Before inoculation of seedlings the isolates were increased in liquid yeast extract medium (11).

**Disease assessment.** Five seeds of the homogeneous generations, $P_1$, $P_2$, and $F_1$, and 20 seeds of the segregating $F_2$ populations per replication were planted in 21- × 21- × 6-cm aluminum trays filled with a sand-soil mixture. Because the amount of seed available from the 65 crosses varied, the number of replications ranged from three to six depending on the cross.

In a second set of experiments, 10 seeds each of the 13 cultivars and of the check bread wheat cultivar, Fortuna, were sown per replication. This trial was repeated four times consecutively.

Ten-day-old seedlings were inoculated with a $10^7$ spores per milliliter suspension by using a quantitative methodology (9). The first set of plants was inoculated with isolate TUN 8204-1 of *S. tritici*. In the second set of trials, each of the 34 isolates was used to inoculate seedlings in separate trays, each containing the 14 cultivars.

**Statistical analysis.** The mean value of each set of 10 seedlings representing a particular cultivar in a tray in the second set of trials was calculated. These values were used for an analysis of variance of the 14-cultivar × 34-isolate × 4-replications trial.

An equation for the estimation of the minimum number of effective factors operating in a cross has been published by Burton (3) and attributed to Sewall Wright.

$$n = 0.25(0.75 - h + h^2)\, D^2/\sigma^2 F_2 - \sigma^2 F_1$$

$n$ = minimum gene number;
$h = \overline{F_1} - \underline{P_1}/\underline{P_2} - \overline{P_1}$;
$D = \overline{P_2} - \overline{P_1}$;

$\bar{P}_1$ = mean of the parent with the lower disease reading;
$\bar{P}_2$ = mean of the parent with the higher disease reading;
$\bar{F}_1$ = mean of the $F_1$ population;
$\sigma^2F_1$ = variance among $F_1$ plants; and
$\sigma^2F_2$ = variance among $F_2$ plants.

An unbiased estimate of $n$ is obtained if the effective factors involved are not linked, are of equal effect, and are not epistatic, and if one parent supplies only plus factors. The other parent must supply only minus factors of those in which the two parents differ, while the degree of dominance is assumed to be the same for all plus factors. Gene number will be underestimated if assumptions do not hold, and $n$ is thus a conservative estimate (3).

In the above equation, the value of $\sigma^2F_1$ is used as an estimate of $\sigma^2E$. However, the environmental variance $\sigma^2E$ is more accurately estimated by $(\sigma^2P_1 + \sigma^2P_2 + \sigma^2F_1)/3$. The value $D$, also sometimes called $R$, represents the range between extreme genotypes. If both parents in a cross show some resistance, but at different levels, they each may contain plus factors not present in the other. Therefore, their differences are not identical to the extreme genotypes possible with the genes involved. The assumption of unidirectional gene distribution will be more closely adhered to if the extreme genotypes are selected from the $F_2$ and used to estimate $D$ (26). The modified equation for the total number of effective factors is:

$$n_T = 0.25(0.75 - h + h^2)\,(Y - X)^2/^2F_2 - [(\sigma^2P_1 + \sigma^2P_2 + \sigma^2F_1)/3]$$

$X$ = value of segregate in $F_2$ with lowest disease reaction.
$Y$ = value of segregate in $F_2$ with the highest disease reaction.

The total number of effective factors involved can be divided between the two parents of a cross using the suggested relation published by Lawrence and Frey (13):

$$nP_1 = [(Y - P_2) + (P_1 - X)]n_T/2(Y - X)$$
$$nP_2 = n_T - nP_1$$

The resulting values are minimum estimates.

For the first set of experiments, the means and variances among plants within each generation were calculated. The replication effects were separated, and the values of the two extreme phenotypes, $X$ and $Y$, in the $F_2$ were determined using the modified Wright's equation (26) and Lawrence and Frey's equation (13), the number of effective factors for each parent in each cross was calculated. The estimates, being minimum, have been rounded to the next highest integer.

The modified Wright's formula is very sensitive to increasing deviations of the $F_1$ from the midparent if the parents have similar values. Therefore, outlyers should be considered overestimates.

A quite different approach to calculating the number of effective factors in a host-pathogen system is to subject a cultivar isolate disease reaction matrix to a Person-analysis for incomplete Person schemes (16). A major assumption is that classical gene-for-gene interactions operate in the *S. tritici–T. durum* aegricorpus. For the analysis it is necessary to ascribe a single value to each cell in the matrix, either the reaction is resistant or it is susceptible. This requires that, in the case of an essentially continuous scale, such as percentage leaf area affected, a cut point must be established below which disease levels are designated resistant and above which they are classified as susceptible. Eyal et al (9) and Scharen et al (19) have used a cluster analysis of the cultivar × isolate matrix, based on Euclidean distance, to group cultivars in similar disease-response classes. The median between the two moderate response classes minus a standard error estimate obtained from the analysis of variance was used as the cut point.

In establishing the cut point in this study, we considered it important to use as large as possible a sampling of cultivars and isolates in the cluster analyses. Therefore, the 13 durum wheat cultivars were divided on the basis of their reaction to the 34 isolates as determined in the second set of experiments. Five separate cluster analyses were run dividing the 14 cultivars subsequently into two through six clusters.

The computer program GENEALOGY analyzes the cultivar × isolate matrix, with each cell containing either a susceptible or a resistant designation. Smaller complete matrices that represent interacting genes are identified from the entire matrix. The linked smaller matrices necessary to identify the entire matrix indicate the total number of resistance and virulence genes operating and how many genes each cultivar or isolate contains (10). The program has been used by Eyal and cooperators to estimate the number of components and their assignment to cultivars and isolates of *S. tritici* and *S. nodorum* (9,19,27).

After we established the cut point in the present study with cluster analysis, the coded data were also analyzed by the GENEALOGY computer program.

## RESULTS

The percentage of necrotic leaf area over four replications is summarized in Table 1. Highly significant differences were found between cultivars and between isolates for disease reaction. The cultivars covered a wide range of infection levels. No significant interaction was found between cultivars and isolates. The cultivar × isolate interaction mean square value, 198, was less than 0.3% of the sum of the mean square values due to cultivars and due to isolates, 70423. When the analysis was made separately for individual countries, the interaction component remained small and insignificant.

The number of effective plus factors for percentage of necrotic leaf area estimated by the use of the modified Wright's formula and Lawrence and Frey's formula, with the exclusion of the four outlying crosses, varied between 1 and 68, with most estimates being less than 20; the average was about seven (Table 2). With this analysis it was not possible to determine genes in common among cultivars. In most crosses, both parents contributed effective plus factors for reaction to disease. Transgressive segregation observed in many crosses also suggested the presence of plus factors in both parents.

Comparison of the different cluster analyses indicated a natural breakpoint somewhere between the second and third most resistant groups. It was decided that the mean of the cluster values of these two groups in the cluster analysis involving six groups would be used as a cut point. The mean was 26% necrotic area, which proved to be about 30% of the mean percentage assigned to the most susceptible cluster.

The components estimated on the assumption of a gene-for-gene system operating are presented in Table 3. Moseman's suggestion (14) is adopted and the gene designation Rst is used.

According to the GENEALOGY analysis, based on a separation of all disease reactions into just two classes, the number of genes for resistance in the cultivars tested varied from zero to eight. Total number of genes estimated to be involved in this cultivar-isolate matrix was 11. These values are similar to the range determined in other studies (9). However, this analysis is relevant only if the disease data indicate the presence of classical gene-for-gene relationships.

## DISCUSSION

Although tested with a large number of isolates originating from diverse locations, the cultivars showed distinct, often easily separable identities as to their overall reaction to infection by *S. tritici*. No complete resistance was observed, thus confirming a conclusion arrived at by many researchers. The lowest levels of infection were obtained on four recently developed cultivars, BD 2131, Karim 80, Volcani 447, and BD 2127, a testimony to successful breeding for resistance.

The susceptible bread wheat check cultivar, Fortuna, showed a moderately high level of infection, while three other durum wheat cultivars had obtained higher mean percentages of infection. As the majority of isolates of *S. tritici* originated from durum wheat cultivars, the intermediate reaction of Fortuna was not unexpected and confirmed earlier observations and research (5,9). Eyal et al (9), in fact, assigned one hypothetical gene for resistance to Fortuna after analyzing a 35-cultivar × 97-isolate trial, in which

most isolates were obtained from bread wheat cultivars.

On average, the isolates collected in Syria from durum wheats showed a 10% higher infection level than those from other countries. About two-thirds of the wheat area in Syria is devoted to durum wheat cultivation, and the fungus may have become specialized in respect to its host-species.

The cultivar × isolate interaction in this study in which mainly durum wheat cultivars and many durum wheat derived isolates were used was very small relative to the main effects of cultivars and isolates. Its proportion was less than 0.3%. In the data published by Eyal et al (9), the interaction component constituted an identical, small proportion. Their study included more bread wheat and durum wheat cultivars and isolates than the present study, and, as the authors indicated, the large number of trials and plants recorded would easily result in statements of significance, due to the many degrees of freedom.

In both studies, interaction explained only an extremely small proportion of the variance. These observations may also indicate some adaptation of certain isolates to the wheat species from which they were obtained.

If cultivar × isolate interaction is absent when only one species is studied but appears when two are used, this may mean that interspecific crosses can be exploited to raise resistance levels.

The absence of a clear cultivar × isolate interaction, the tendency of the Syrian durum wheat derived isolates to have a higher infection level on the durum wheats, the similar genetic action of distinct isolates of *S. tritici* (24), and the suggestion of associated evolution of septoria species and gramineous groups (20) taken together signify the existence of a definite host-species specialization as opposed to a cultivar specialization of isolates of *S. tritici*. The data presented by Eyal et al (9) also support this conclusion.

The isolates of the pathogen vary in the levels of disease brought about on cultivars within the host-species, in this case *T. durum*, but rankings of the cultivars are largely retained independent of the isolate used. This pathogenic character has been called aggressiveness and horizontal pathogenicity (15,22,23). Also, when there is no differential interaction between isolates and cultivars, any differences between cultivars are due to horizontal resistance (23).

The prime assumption of classical gene-for-gene relationships, differential interaction, on which the use of GENEALOGY is

TABLE 1. Mean percentage of necrotic leaf area of the 13 selected durum wheat cultivars and Fortuna infected with 34 isolates of *S. tritici*, averaged over four replications

| | | Cultivars | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Isolates | | Kyperounda | Badri | BD 2131 | BD 2127 | 65150-Lds | D75-9-6B-5B-4B-10B | D75-40-11B-4B-2B | Ben Bechir 79 | Karim 80 | Maghrebi 72 | Etit 38 | Volcani 447 | Zenati Bouteille | Fortuna | $\bar{x}$ |
| TUN | 8201 | 24 | 66 | 14 | 5 | 77 | 21 | 63 | 18 | 12 | 12 | 22 | 11 | 39 | 43 | 30 BCDEFGH[a] |
| | 8202 | 28 | 65 | 13 | 19 | 80 | 37 | 71 | 14 | 3 | 14 | 30 | 5 | 55 | 46 | 34 DEFGHIJ |
| | 8202-1 | 28 | 24 | 3 | 7 | 62 | 22 | 48 | 6 | 3 | 1 | 17 | 1 | 23 | 48 | 21 A |
| | 8204-1 | 20 | 64 | 2 | 3 | 69 | 23 | 65 | 23 | 1 | 8 | 20 | 4 | 20 | 32 | 25 ABC |
| | 8205 | 56 | 85 | 8 | 15 | 96 | 44 | 74 | 25 | 12 | 24 | 41 | 3 | 51 | 59 | 42 JKLM |
| | 8206 | 31 | 52 | 5 | 19 | 84 | 40 | 49 | 11 | 8 | 25 | 22 | 6 | 48 | 45 | 32 CDEFGHI |
| | 8206-2 | 38 | 66 | 20 | 32 | 79 | 26 | 68 | 30 | 28 | 40 | 31 | 23 | 55 | 51 | 42 JKLM |
| TKY | 81218 | 22 | 51 | 3 | 3 | 63 | 33 | 71 | 19 | 7 | 10 | 10 | 6 | 30 | 50 | 27 ABCDE |
| | 81262 | 31 | 47 | 4 | 13 | 70 | 41 | 46 | 19 | 3 | 15 | 21 | 2 | 32 | 41 | 27 ABCDE |
| | 81281 | 36 | 77 | 21 | 24 | 94 | 31 | 61 | 35 | 21 | 34 | 31 | 21 | 53 | 68 | 43 KLM |
| | 8201 | 39 | 53 | 7 | 4 | 93 | 13 | 61 | 5 | 6 | 19 | 21 | 6 | 44 | 43 | 29 BCDEFG |
| | 8202 | 25 | 40 | 6 | 9 | 71 | 22 | 51 | 4 | 5 | 5 | 18 | 4 | 21 | 39 | 23 AB |
| | 8205 | 40 | 69 | 4 | 28 | 73 | 33 | 54 | 33 | 16 | 32 | 37 | 12 | 47 | 41 | 37 GHIJKL |
| | 8209 | 40 | 77 | 5 | 22 | 79 | 29 | 51 | 27 | 5 | 27 | 29 | 31 | 55 | 55 | 38 HIJKL |
| ISR | B1R1 | 42 | 54 | 8 | 11 | 71 | 19 | 50 | 17 | 12 | 10 | 22 | 3 | 25 | 33 | 27 ABCD |
| | B1R2 | 38 | 56 | 3 | 5 | 71 | 27 | 69 | 7 | 7 | 27 | 31 | 11 | 44 | 49 | 32 CDEFGHI |
| | B1R3 | 22 | 53 | 3 | 4 | 72 | 9 | 57 | 19 | 1 | 8 | 22 | 5 | 32 | 42 | 25 ABC |
| | B1R4 | 23 | 67 | 18 | 20 | 92 | 24 | 74 | 23 | 14 | 15 | 24 | 18 | 42 | 52 | 36 FGHIJKL |
| | 80-6 | 40 | 51 | 12 | 14 | 66 | 33 | 61 | 29 | 4 | 26 | 27 | 2 | 35 | 48 | 32 CDEFGHI |
| | 80-8 | 43 | 51 | 9 | 8 | 86 | 39 | 73 | 17 | 6 | 32 | 30 | 3 | 47 | 50 | 35 EFGHIJK |
| | 80-11 | 45 | 74 | 9 | 4 | 89 | 21 | 64 | 17 | 2 | 28 | 29 | 3 | 54 | 51 | 35 DEFGHIJ |
| SYR | 8202 | 44 | 68 | 19 | 23 | 86 | 40 | 74 | 24 | 19 | 23 | 45 | 10 | 57 | 58 | 42 JKLM |
| | 8205 | 55 | 59 | 5 | 14 | 73 | 50 | 76 | 41 | 12 | 40 | 53 | 16 | 51 | 67 | 44 LM |
| | 8206 | 36 | 63 | 14 | 27 | 80 | 30 | 68 | 25 | 16 | 24 | 36 | 12 | 55 | 50 | 38 HIJKL |
| | 8207 | 42 | 79 | 30 | 26 | 91 | 43 | 83 | 43 | 25 | 41 | 39 | 26 | 55 | 74 | 50 M |
| | 8209 | 36 | 80 | 9 | 16 | 77 | 25 | 65 | 27 | 10 | 20 | 29 | 3 | 45 | 59 | 36 FGHIJKL |
| | 82-6 | 45 | 62 | 8 | 21 | 96 | 32 | 62 | 23 | 6 | 29 | 34 | 5 | 51 | 58 | 38 HIJKL |
| POR | 3 | 43 | 64 | 18 | 21 | 77 | 30 | 70 | 16 | 12 | 31 | 28 | 10 | 47 | 49 | 37 GHIJKL |
| | 8 | 24 | 64 | 4 | 8 | 85 | 24 | 68 | 17 | 7 | 6 | 20 | 3 | 32 | 51 | 29 BCDEFG |
| | 10 | 22 | 60 | 3 | 5 | 68 | 14 | 37 | 12 | 4 | 19 | 14 | 5 | 37 | 51 | 25 ABC |
| | 11 | 47 | 69 | 3 | 6 | 70 | 36 | 61 | 35 | 1 | 24 | 29 | 14 | 39 | 51 | 35 DEFGHIJ |
| | 81199 | 26 | 41 | 3 | 19 | 72 | 28 | 60 | 7 | 2 | 29 | 17 | 5 | 42 | 48 | 28 ABCDEF |
| ITL | 82024 | 56 | 66 | 7 | 6 | 88 | 38 | 73 | 30 | 8 | 24 | 33 | 14 | 59 | 46 | 39 IJKL |
| SPN | 81299 | 29 | 45 | 2 | 13 | 52 | 22 | 54 | 18 | 2 | 23 | 25 | 2 | 39 | 53 | 27 ABCD |
| | $\bar{x}$ | 36 | 61 | 9 | 14 | 78 | 29 | 63 | 21 | 9 | 22 | 27 | 9 | 43 | 50 | |
| | | D | G | A | A | H | C | G | B | A | B | C | A | E | F | |

[a] Means not followed by the same letter are significantly different as tested by LSD (0.05).

TABLE 2. Number of effective plus factors determining percentage of necrotic leaf area due to infection by isolate of *S. tritici*, TUN 8204-1, in crosses involving the 13 durum wheat cultivars

| Parents | | Cultivar code | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Code | Cultivar | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
| 1 | Kyperounda | | 2[a] | 2 | 3 | 1 | 6 | 4 | 7 | 3 | 4 | ...[c] | ... | ... |
| 2 | Badri | 3[b] | | 2 | 2 | 17 | 3 | 22 | 1 | 3 | 4 | 10 | ... | 0 |
| 3 | BD 2131 | 1 | 1 | | 4 | 1 | 2 | 3 | 0 | 16 | 0 | 1 | ... | 1 |
| 4 | BD 2127 | 2 | 1 | 6 | | 2 | 30 | 0 | 5 | 1,528 | 2 | ... | ... | ... |
| 5 | 65150-Lds | 2 | 21 | 7 | 6 | | 2 | 211 | 68 | 5 | 0 | 2 | ... | 15 |
| 6 | D75-9-6B-5B-4B-10B | 6 | 1 | 3 | 39 | 1 | | 1 | 5 | 4 | 50 | 11 | 127 | 2 |
| 7 | D75-40-11B-4B-2B | 6 | 26 | 11 | 0 | 202 | 3 | | 0 | 0 | 3 | ... | 3 | 182 |
| 8 | Ben Bechir 79 | 5 | 1 | 0 | 7 | 24 | 3 | 0 | | 17 | 3 | 1 | 3 | 1 |
| 9 | Karim 80 | 2 | 1 | 22 | 1,501 | 1 | 4 | 0 | 13 | | 7 | 2 | 15 | 15 |
| 10 | Maghrebi 72 | 2 | 1 | 0 | 3 | 0 | 45 | 1 | 3 | 8 | | 5 | ... | 1 |
| 11 | Etit 38 | ... | 8 | 3 | ... | 1 | 14 | ... | 2 | 3 | 6 | | 3 | ... |
| 12 | Volcani 447 | ... | ... | ... | ... | ... | 81 | 1 | 1 | 19 | ... | 1 | | ... |
| 13 | Zenati Bouteille | ... | 0 | 3 | ... | 11 | 5 | 127 | 2 | 27 | 2 | ... | ... | |

[a]Number of effective plus factors (for susceptibility) expressed in Kyperounda when crossed to Badri.
[b]Number of effective plus factors (for susceptibility) expressed in Badri when crossed to Kyperounda.
[c]...: No fillial and/or related generations available.

TABLE 3. Hypothetical components of resistance in 13 durum wheat cultivars estimated by GENEALOGY based on infection levels due to 34 isolates of *S. tritici*[a]

| Cultivar | Resistance components[b] | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Rst 1 | Rst 2 | Rst 3 | Rst 4 | Rst 5 | Rst 6 | Rst 7 | Rst 8 | Rst 9 | Rst 10 | Rst 11 | Total |
| Kyperounda | + | | | | | | | | | | | 1 |
| Badri | | + | | | | | | | | | | 1 |
| BD 2131 | + | + | + | + | + | + | + | | + | | | 8 |
| BD 2127 | + | + | + | | | | | + | + | + | | 6 |
| 65150-Lds | | | | | | | | | | | | 0 |
| D75-9-6B-5B-4B-10B | | + | | + | | | | | + | | | 3 |
| D75-40-11B-4B-2B | | | | | | | | | | | | 0 |
| Ben Bechir 79 | + | + | + | | + | | | | + | | | 5 |
| Karim 80[c] | | | | | | | | | | | | - |
| Maghrebi 72 | | + | | | | + | | | + | | | 3 |
| Etit 38 | + | + | + | | | | | | + | | | 4 |
| Volcani 447 | + | + | + | + | + | + | | | + | | + | 8 |
| Zenati Bouteille | | + | | | | | | | + | | | 2 |

[a]Cut point between resistant and susceptible was 26% necrotic leaf area.
[b]No statement can be made about the component determining specific resistance being dominant or recessive.
[c]This cultivar is resistant to all isolates; thus gene constitution cannot be determined.

based, could not be justified by these data. Because the premise is assumed to be incorrect, no value can be attached to the otherwise seemingly realistic gene estimates generated by the computer program. The presence of nonclassical gene-for-gene interactions on a microscale, involving very minor reductions in resistance related to very minor increases in pathogenicity cannot presently be excluded. This may in fact account for the very low interaction component observed.

The suggested host-species specialization by *S. tritici* for *T. aestivum*, *T. durum*, and probably other graminae, as opposed to cultivar specificity within species expressed as a clear cultivar isolate interaction, does not preclude dynamic aspects of the system: flexibility, change, and adaptation. New isolates may evolve locally or regionally that have increased levels of aggressiveness, leading to the demise of previously resistant cultivars.

Should the absence of within-species physiologic specialization for *S. tritici* indeed be consistently observed, then the presence of mainly additive effects and clearly demonstrated intermediate heritability values (24), implies a very effective and practical approach to raising resistance levels. By employing breeding and selection methods that stress mixing a large pool executed under disease pressure of highly aggressive isolates, many small effects may be accumulated in stable homozygous lines. If artificial inoculation is practiced, emphasis should be on using regional isolates of *S. tritici* collected from cultivars of the same species grown in areas that have recently shown an increase in field disease levels.

The recent decision by CIMMYT to require resistance to all prevalent Mexican isolates of *S. tritici* in bread wheat distributed internationally from their genetically broad-based wheat program should prove effective in increasing, on a worldwide basis, available resistant germ plasm. CIMMYT's global multi-location testing approach, its most important international strategy (6), would simultaneously signal occurrence of increased aggressiveness in the pathogen, thus pinpointing additional high priority screening locations. Constant watchful disease surveillance for the presence of true physiologic specialization, however, remains a necessity.

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