Genetic Variation for Virulence and Resistance in the Wheat-*Mycosphaerella graminicola* Pathosystem I. Interactions Between Pathogen Isolates and Host Cultivars

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ABSTRACT

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Genetic variation for virulence in 63 *Mycosphaerella graminicola* isolates, originating from 13 countries, was studied in two seedling experiments. Each experiment was performed according to a partially balanced incomplete block design with four replications over time. The first experiment put emphasis on *M. graminicola* isolates that originated from bread wheat, and comprised 50 isolates that were inoculated on a set of testers containing 19 bread wheat cultivars, four durum wheat cultivars, and one triticale cultivar. In the second experiment more attention was paid to *M. graminicola* isolates that originated from durum wheat, and comprised 15 isolates that were inoculated on a set of testers containing 17 durum wheat cultivars, four bread wheat cultivars, one triticale cultivar, and a *Triticum turgidum* subsp. *dicoccoides* accession. Two disease parameters, the presence of necrosis (*N*) and pycnidia (*P*) estimated as percentages of primary leaves, were employed to measure disease severity. Genetic variation for virulence in the pathogen isolates and genetic variation for resistance in the host cultivars were estimated by analyses of covariance. The significance of cultivar × isolate interactions in both experiments and for each disease parameter suggested a gene-for-gene interaction between resistance and virulence loci in host and pathogen, respectively. An agglomerative hierarchical clustering procedure, that used one df component of interaction between isolates and cultivars as a proximity measure, was employed to study the similarity between isolates and cultivars. Discrepancies between *N* and *P* resulted in nonidentical clusters of isolates and cultivars when considering these parameters separately, which suggested that *N* and *P* were under different genetical control. Evidently, isolates of *M. graminicola* were specialized to either bread wheat or durum wheat. This was particularly evident when considering *P*. It is proposed, therefore, to designate two varieties in *M. graminicola* that refer to the host species specialization in this pathogen.

Additional keywords: pathogenic variation, pathotypes, races, *Septoria tritici, Triticum aestivum, Triticum turgidum* subsp. *durum.*

Septoria tritici leaf blotch is a fungal disease of bread wheat and durum wheat, *Triticum aestivum* L. and *T. turgidum* (L.) Thell. subsp. *durum* L., respectively. The disease is caused by *Septoria tritici* Roberge in Desmaz. or its teleomorph *Mycosphaerella graminicola* (Fuckel) J. Schröt. in Cohn, that has been reported in several wheat-producing areas of the world (14,34,36). It is particularly a major problem in regions characterized by a temperate, high rainfall environment during the wheat growing season, such as the Mediterranean Basin, Eastern and Central Africa, and the Southern Cone of South America (12,20,40). High incidences and disease severities were also reported in the United States and Mexico, as well as in some European countries, New Zealand, and Australia (12,29).

Relative humidity (RH) and temperature are considered to be key determinants for successful penetration of the host and its further colonization by the fungus (16). Based on interactions between temperature and leaf wetness periods, temperatures of 20 to 25°C were considered to promote infection (23). Pycnidia are produced under a RH range of 35 to 100% with an optimum at 85% (27), although Shaw and Royle (38) reported that for a susceptible cultivar, nonconducive weather conditions in the field did not seem to limit disease establishment.

Restriction fragment length polymorphism (RFLP) markers show extensive genetic variation in *M. graminicola* (24,25). Genetic variation for virulence, as expressed by interactions between host and pathogen genotypes, has been a questionable subject

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since physiologic specialization in this fungus was suggested, despite reported declines in effectiveness and an inconsistent expression of resistance in wheat to *M. graminicola* (10,12,17,26,30,33,43). The majority of studies on the wheat-*M. graminicola* pathosystem dealt with bread wheat and bread wheat-derived isolates (20,26). Tetraploid wheat species were reported to be more resistant to *M. graminicola* than bread wheat (6,44). Van Ginkel and Scharen (41,42,43) analyzed the resistance in durum wheat, and concluded that interactions between host and pathogen genotypes were of minor importance, since additive gene effects and general combining ability explained the greater part of the genetic variation for resistance that was revealed in their inheritance studies. Therefore, they suggested that host species specialization in *M. graminicola* was a much simpler explanation for reported physiologic specialization (10), and hence the absence of differential gene-for-gene relationships, thus variation for aggressiveness rather than virulence, among *M. graminicola* isolates. In addition, proportions of the total variance in analyses of variance that were attributable to interaction were low and not always significant (12,43,44). This ambiguity and the inconclusive reports on host-pathogen interactions, obviously thwart the development of effective breeding strategies, which has been the most widely adopted strategy to control *M. graminicola* (20,26,40). Inheritance of resistance in wheat to *M. graminicola* was reported to be conditioned by single or multiple dominant and recessive genes with major effects, as well as by additivity of resistance factors with a less pronounced effect (26,42).

The present contribution was part of a larger study that was undertaken to elucidate genetic variation for virulence in the wheat-*M. graminicola* pathosystem, and comprised two experiments. The first experiment largely dealt with isolates from bread wheat, whereas the second experiment mainly involved isolates that originated from durum wheat.

MATERIALS AND METHODS

Plant materials. In the first experiment, 19 bread wheat cultivars, four durum wheat cultivars, and one triticale (× *Triticosecale* Wittmack) cultivar were employed. In the second experiment, 17 durum wheat cultivars, one *T. turgidum* (L.) Thell. subsp. *dicoccoides* (Körn) Thell. (genomes AABB, $2n = 28$) accession, one triticale cultivar, and four bread wheat cultivars were utilized to study genetic variation for virulence and resistance (Table 1).

***M. graminicola* isolates.** Leaf samples were collected in 13 countries and originated from bread wheat and durum wheat cultivars. Monopycnidial *M. graminicola* isolates were obtained (18) that were used to inoculate susceptible wheat cultivars ('Inbar' and 'Lakhish' for durum wheat and bread wheat isolates, respectively). Desiccated colonized primary leaves were stored for short-term preservation. Sixty-three isolates were selected on a regional basis; hence, most countries were represented by accessions from several more or less distant locations (Table 2). Inoculum was prepared by inoculating 50 ml of liquid yeast-glucose medium in 100-ml Erlenmeyer flasks with fresh *M. graminicola* colonies from agar plates. For each experiment, two flasks per isolate were incubated for 5 days in a temperature-controlled reciprocal shaker at 15°C. The resultant spore suspensions were pelleted by centrifuging at 10,000 rpm (12,360 × *g*) for 10 min, resuspended in deionized water, and adjusted to a density of 10^7 spores/ml.

Experimental design. The experiments comprised sets of inoculations and were conducted according to a partially balanced incomplete block design with respect to pathogen isolates, which permitted the execution of four replicates over time. Experiment 1 involved 25 blocks of eight main plots (=isolates), and experiment 2 comprised 10 blocks of six main plots (5). The host cultivars were randomly allocated to 24 subplots in each main plot.

Experimental procedures and conditions. Ten to 15 seeds per accession were linearly sown in plastic pots (5 by 5 cm) with a peat/sand mixture. Plants were grown in controlled walk-in climate chambers with similar pre- and postinoculation conditions with respect to light intensity and day length (56 μE sec^{-1} m^{-2} for 16 h day^{-1}). Pre- and postinoculation temperature and RH conditions were 18/16°C (day/night rhythm) and 70% RH, and 22/21°C and ≥85% RH, respectively.

Quantitative inoculations were conducted by spraying spore suspensions, 30 ml/isolate supplemented with two drops of Tween 20 surfactant, on the test cultivars that were randomized on a turntable, adjusted at 15 rpm, in a closed inoculation cabinet equipped with interchangeable atomizers and a water cleaning device to avoid contamination. Incubation was conducted under polyethylene-covered aluminum frames, providing leaf wetness for 48 h at a light intensity of approximately 3 μE sec^{-1} m^{-2}. Fertilizer (Sporumix PG [Windmill Holland, Vlaardingen, Netherlands], 0.5 g $liter^{-1}$) was applied at 7 days after inoculation, and

TABLE 1. Experimental code and origin of each bread wheat and durum wheat cultivar, of an accession of *Triticum turgidum* subsp. *dicoccoides*, and of a triticale cultivar employed to study genetic variation for virulence in *Mycosphaerella graminicola*

EC[z]	Cultivars	Origin
Bread wheat		
An	Anza	Algeria
Ar	Arminda	Netherlands
BL	Beth Lehem	Israel
Bo	Bobwhite	Mexico
Ce	Ceeon	Israel
Co	Colotana	Brazil
Ge	Gerek 79	Turkey
Ia	Iassul 20	Brazil
K7	Kavkaz/7C	Mexico
KK	Kavkaz/K4500 l.6.a.4	Mexico
KT	Klein Titan	Argentina
KU	Kavkaz/UP301	Mexico
KZ	Kavkaz	USSR
La	Lakhish	Israel
Ob	Obelisk	Netherlands
Ol	Olaf	USA
Sh	Shafir	Israel
T29	Taichung 29	Japan
To	Toropi	Brazil
Ve	Veranopolis	Brazil
Durum wheat		
A65	Acsad 65	Algeria
B17	Bidi 17	Algeria
BD	BD2777	Morocco
Ca	Cakmak 79	Turkey
Cc	Cocorit	Morocco
Et	Etit 38	Israel
H3	Hedba 3	Algeria
I69	Inrat 69	Algeria
In	Inbar	Israel
Jo	Jori	Morocco
Ma	Marzak	Tunisia
MB	M. B. Bachir	Algeria
OR	Omrabi 5	Morocco
OZ	OZ 368	Algeria
Sa	Safir	Tunisia
Te	Tensift	Morocco
Vo	Volcani 447	Israel
Wa	Waha	Algeria
ZB	Zenati Bouteille	Algeria
ZP	Zenati Bouteille/*T. polonicum*	Algeria
T. turgidum subsp. *dicoccoides*		
G25	G25	Israel
Triticale		
Be	Beagle	Mexico

[z] EC = experimental code.

the emerging second leaves were clipped 14 days after inoculation in order to facilitate light penetration to the primary leaves and disease assessment. Disease severity was evaluated at 21 days after inoculation using two parameters; the presence of necrosis (*N*) and pycnidia (*P*), estimated as percentages of the total primary leaf area of individual seedlings. These values were averaged per pot for further analyses of the disease parameters *N* and *P*.

TABLE 2. Experimental code and origin of 63 *Mycosphaerella graminicola* isolates studied for genetic variation of virulence towards 23 wheat cultivars and one triticale cultivar

EC[x]	Isolate	Country	Location
AR1	*IPO*86063	Argentina	Balcarce
AR2	*IPO*86068	Argentina	Balcarce
AR3	*IPO*87022	Argentina	Pergamino
AR4	*IPO*87023	Argentina	Pergamino
AR5	*IPO*87024	Argentina	Pergamino
AR6	*IPO*86078	Argentina	Tres Arroyos
UR1	*IPO*87019	Uruguay	Colonia
UR2	*IPO*87021	Uruguay	Colonia
UR3	*IPO*87016	Uruguay	Dolores
UR4	*IPO*87018	Uruguay	Dolores
UR5	*IPO*87020	Uruguay	Dolores
ET1	*IPO*88005	Ethiopia	Assassa
ET2	*IPO*88010	Ethiopia	Bekoje
ET3	*IPO*88012[y]	Ethiopia	Bekoje
ET4	*IPO*88020	Ethiopia	Bekoje
ET5	*IPO*88013	Ethiopia	Blue Nile Valley
ET6	*IPO*88018	Ethiopia	Holetta
ET7	*IPO*88019	Ethiopia	Holetta
ET8	*IPO*88021	Ethiopia	Holetta
ET9	*IPO*88004	Ethiopia	Kulumsa
ET10	*IPO*88022	Ethiopia	Mota
ET11	*IPO*88027	Ethiopia	Sinana
KE1	*IPO*87000[z]	Kenya	Eldoret
KE2	*IPO*87011[z]	Kenya	Eldoret
KE3	*IPO*87008	Kenya	Eldoret
KE4	*IPO*87015	Kenya	Mou Narok
KE5	*IPO*87009	Kenya	Njoro
KE6	*IPO*87012	Kenya	Njoro
KE7	*IPO*87013	Kenya	Njoro
KE8	*IPO*86026	Kenya	Timau
BU1	*IPO*88023	Burundi	Tora
BU2	*IPO*88024	Burundi	Tora
RW1	*IPO*88037	Rwanda	Tamira
UG1	*IPO*88038	Uganda	Kalengyere
NL1	*IPO*235	Netherlands	Anjum
NL2	*IPO*89011	Netherlands	Barendrecht
NL3	*IPO*89013	Netherlands	Drenthe
NL4	*IPO*88025	Netherlands	Ebelsheerd
NL5	*IPO*89012	Netherlands	Wageningen
NL6	*IPO*89010	Netherlands	Zelder
TK1	*IPO*86013	Turkey	Adana
TK2	*IPO*88014	Turkey	Adana
TK3	*IPO*88015	Turkey	Adana
TK4	*IPO*88016	Turkey	Adana
TK5	*IPO*86022[y]	Turkey	Altinova
TK6	*IPO*86023	Turkey	Altinova
TK7	*IPO*86010	Turkey	Tasci
TK8	*IPO*86009	Turkey	Tasci
TK9	*IPO*86008	Turkey	Tasci
TK10	*IPO*88017	Turkey	Unknown
AL1	*IPO*90020	Algeria	Guelma
TN1	*IPO*91009[y]	Tunisia	Beja
TN2	*IPO*91010[y]	Tunisia	Beja
TN3	*IPO*91016[y]	Tunisia	Beja
TN4	*IPO*91011[y]	Tunisia	Tunis
TN5	*IPO*91012[y]	Tunisia	Sidi Ncir
TN6	*IPO*91014[y]	Tunisia	Mateur
TN7	*IPO*91015[y]	Tunisia	Fetissa
SY1	*IPO*91004[y]	Syria	Lattakia
MO1	*IPO*91017[y]	Morocco	O. Frej
MO2	*IPO*91018[y]	Morocco	J. Shaim
MO3	*IPO*91019[y]	Morocco	Meknes
MO4	*IPO*91020[y]	Morocco	Doukhala

[x] EC = experimental code.
[y] Collected from durum wheat.
[z] Collected from the same sample.

Data analyses. The experimental design was only partially balanced, which would imply that the corresponding statistical regression model would comprise more than a thousand parameters. This was computationally not feasible; hence, it appeared appropriate to subject the responses *N* and *P* to analyses of covariance (ANCOVAs). Block differences were fully accounted for by setting one covariate for each block. Since blocks were parts of the experiment that were carried out sequentially, block effects were confounded with possible time effects. These effects could only be partly disentangled; hence, block effects were used to adjust the responses. A consequence of this procedure was that tables of means might contain some negative values, after adjusting for covariates (Tables 3 to 6). Statistical analyses were conducted using the Genstat 5 package (15) on transformed (arc-sin) and untransformed data sets. Since transformations did not substantially stabilize the residual variance and did not influence the conclusions, untransformed data are presented here.

In order to reveal structures of the interactions between host and pathogen genotypes, the tables of means were subjected to a hierarchical agglomerative clustering procedure as described by Corsten and Denis (8). The procedure groups rows and columns in the tables to identify a minimum number of groups that account for the overall interaction. The groups are internally homogeneous. In each step of this sequential procedure, the mean square for interaction (MS_{int}) is calculated for all possible subtables consisting of a pair of rows or a pair of columns of the full table. The pair of rows or columns with minimal MS_{int} is merged, giving an updated table, and the process is repeated. Thus, a sequence of amalgamations of rows and columns is produced, eventually leading to a two-by-two table. In this way the total sum of squares for interaction (*S*) is built up from orthogonal increments, each connected with a merge as described, to obtain insight into a possible structure of the interaction. Corsten and Denis (8) formulated an *F*-test procedure to stop clustering just before *S* exceeds the critical value $c(x) = ns^2F(n, f, \alpha)$, in which n = (number of isolates − 1)(number of cultivars − 1), s^2 = an estimate of the residual variance obtained independently from the two-way tables subjected to the cluster analysis, and $F(n, f, \alpha)$ = the upper α point of the *F* distribution with n and f degrees of freedom. This procedure determined the probability of stopping too early, i.e., ending up with too many groups, under the H_0 hypothesis of no interaction between isolates and cultivars. Thus, it determined which isolates or cultivars were significantly different from each other.

RESULTS

Symptom development. The inoculated leaves remained green during the first 8 to 9 days after inoculation. The first symptoms were generally observed as necrosis, starting at the leaf tips. In susceptible cultivars, necrotic blotches quickly coalesced and eventually resulted in high *N* levels with maxima of 90% in experiment 1 and 100% in experiment 2 at 21 days after inoculation. Necrosis was generally straw-colored, though characteristic straw-reddish to greyish black phenotypes were observed in responses with few and abundant pycnidia, respectively. Pycnidia were almost entirely produced in the necrotic area, and appeared somewhat later than the necrosis. In very susceptible responses, pycnidia sometimes occurred in still green, but collapsing, tissue. Resistance was expressed as low *P*, which was not necessarily concurrent with *N* (Tables 3 to 6, data adjusted for block effects). Low *N* was sometimes confined to small necrotic spots, which resembled those developed in hypersensitive responses towards obligate parasites such as the cereal rusts and powdery mildews, that were particularly evident in the responses of bread wheat cultivars towards durum wheat-derived isolates.

Bread wheat-derived isolates almost exclusively produced pycnidia in the bread wheat cultivars, whereas pycnidial production by durum wheat-derived isolates was almost entirely restricted to the durum wheat cultivars. A few cultivars, particularly 'Inbar', allowed some pycnidia production of isolates that originated from both wheat species (Tables 4 and 6). The discrimination between isolates that were derived from either bread wheat or durum wheat was less evident when considering *N* (Tables 3 and 5, Figs. 1 to 4), since bread wheat isolates usually induced abundant necrosis in the durum wheat cultivars, whereas durum wheat isolates generally caused little necrosis in the bread wheat and triticale cultivars (Tables 3 and 5). Isolates *IPO*88012-ET3 (Tables 3 and 4), *IPO*91010-TN2, and *IPO*91019-MO3 (Tables 5 and 6) were derived from durum wheat, but appeared to be adapted to bread wheat as evidenced by high *P* levels in bread wheat, but low *P* levels in durum wheat. *N* levels were high in both species. A few durum wheat cultivars, such as 'Inbar' and 'Omrabi 5', allowed some pycnidia formation, whereas the triticale cultivar Beagle was not affected by either type of *M. graminicola* isolate (Tables 4 and 6).

Interactions between isolates and cultivars. ANCOVAs were conducted on both complete and restricted response matrices for *N* and *P* (Table 7). The restricted analyses in each experiment merely included either bread wheat cultivars and bread wheat-derived isolates, or durum wheat cultivars and durum wheat-derived isolates. Such analyses were necessary to eliminate any contribution to the MS_{int} of the aforementioned observation that bread wheat and durum wheat isolates almost exclusively produced pycnidia in their respective host species. The interactions between cultivars and isolates were highly significant ($P < 0.01$) for parameters *N* and *P*. Numerous interactions were observed in the response matrices for *N* and *P* (Tables 3 to 6), though simultaneous consideration of these parameters revealed that they were not necessarily corresponding. Hence, the relationship between pathogen isolates and host cultivars could be categorized into four types. Responses could be noninteractive for both *N* and *P*, interactive for both *N* and *P*, interactive for *N* and noninteractive for *P*, or vice versa, which suggested that *N* and *P* were under dissimilar genetic control (Table 8).

Cluster analyses. The response matrices of *N* and *P* of both experiments were subjected to cluster analyses, which resulted in dendrograms for isolates and cultivars for each disease parameter (Figs. 1 to 4).

Experiment 1. The analysis for *N* resulted in 31 and 21 significantly different clusters for isolates and cultivars, respectively (Fig. 1). For *P*, the analysis resulted in 21 and 18 significantly different clusters for isolates and cultivars, respectively (Fig. 2). Each cluster, considering both *N* and *P*, contained less than five accessions. The number of significantly different clusters per country was substantial. For example, 10 Turkish isolates (Table 2) were attributed to nine significantly different clusters for both *N* and *P*. Similar comparisons for the other countries indicated extensive genetic variation for virulence not only between, but also within, local populations of the fungus, since most countries were represented by multiple accessions from several locations (Table 2). For example, 11 isolates were sampled at seven locations in Ethiopia, which were assigned to seven significantly different clusters for *N*, though the isolate from Assassa (*IPO*88005-ET1) was merged with the three isolates from Bekoje, representing the central southern part of the country. The three isolates from Holetta were placed in two clusters for *N*. For *P*, only four isolates were clustered, one cluster comprised two isolates from Bekoje (*IPO*88010-ET2 and *IPO*88012-ET3) and the other cluster combined an isolate from Bekoje and the isolate from Assassa (*IPO*88020-ET4 and *IPO*88005-ET1). The remaining seven isolates were significantly different at $P = 0.01$; thus they were not clustered, which implied considerable genetic variation for virulence within a location such as Holetta, in which the cultivars Colotana, Kavkaz/K4500 l.6.a.4, and Veranopolis differentiated the isolates. Analogous comparisons revealed similar results for isolates from other countries. The Kenyan isolates showed the least variation, though two isolates from Eldoret that were derived from the same leaf sample (*IPO*87000-KE1 and *IPO*87011-KE2) proved to be significantly different, particularly for *P*.

Isolate *IPO*89012-NL5 was isolated from a field plot that was inoculated with *IPO*235-NL1; hence, these two isolates were considered to be similar, as reflected in the cluster analysis that clustered them in the first step for *N* and in the fifth step for *P*.

Dissimilarities among the cultivars were particularly evident in the dendrogram for *P* (Fig. 2). The durum wheat accessions were in one cluster, as were 'Kavkaz' and one of its derivatives, 'Kavkaz/UP301'. A third cluster comprised the cultivars Iassul 20, Beagle, and Bet Lehem, which were very resistant to most isolates. The majority of the cultivars, however, was not clustered for *P* and *N*, indicating considerable genetic variation for resistance to *M. graminicola* in the tested cultivars.

Experiment 2. The 15 isolates were separated into eight and ten significantly different clusters for *N* and *P*, respectively (Figs. 3 and 4). The four isolates that were particularly pathogenic on bread wheat cultivars (*IPO*88018-ET6, *IPO*90020-AL1, *IPO*91019-MO3, and *IPO*91010-TN2) were separated for both *N* and *P* from the remaining 11 isolates that were clearly adapted to the durum wheat cultivars. Because of the virulence differences, *IPO*88018-ET6, *IPO*90020-AL1, *IPO*91019-MO3, and *IPO*91010-TN2 were placed in two significantly different clusters. Isolate *IPO*91019-MO3 only produced high *P* levels in 'Anza', whereas the others were also virulent on 'Lakhish' and to a lesser extent on 'Bobwhite'. Considering *P* of the durum wheat-derived isolates, those originating from Tunisia were all significantly different from each other, which implied ample genetic variation for virulence among and within locations (e.g., Beja, isolates *IPO*91009-TN1, *IPO*91010-TN2, and *IPO*91016-TN3). Considering *P*, isolate *IPO*91014-TN6 was grouped with *IPO*91004-SY1 from Syria, whereas *IPO*91016-TN3 showed most similarity to two Moroccan isolates (*IPO*91020-MO4 and *IPO*91018-MO2). The remaining Moroccan isolate (*IPO*91017-MO1) was unique.

The arrangement of the cultivars over the clusters was not analogous for the two disease parameters (eight and twelve significantly different clusters for *N* and *P*, respectively; Figs. 3 and 4). For instance, 'Marzak' and *T. dicoccoides* 'G25' were in the same cluster for *N*, but for *P* the latter was clustered with the highly resistant bread wheat cultivars Kavkaz-K4500 l.6.a.4 and Bobwhite, and with the triticale cultivar Beagle, because of the low *P* levels in these accessions; hence, the absence of interactions. However, 'Marzak' was clustered with 'Tensift', and displayed a highly susceptible response with the majority of the durum wheat-derived isolates. Similarity for *P* was observed between 'OZ 368', 'Bidi 17', 'Hedba 3', and 'M. B. Bachir' that are land races cultivated in Algeria. These cultivars occurred in one cluster for *N*, but were pairwise separated for *P*, particularly because of the responses with isolates *IPO*91011-TN4, *IPO*91012-TN5, and *IPO*91015-TN7 (Table 6). For *P*, the bread wheat cultivars Lakhish and Anza were individually separated from cultivars Kavkaz/K4500 l.6.a.4 and Bobwhite, because of their susceptibility for isolates *IPO*88018-ET6, *IPO*91010-TN2, and *IPO*90020-AL1, and the differential response to *IPO*91019-MO3 (Table 6).

The dendrograms of isolates and cultivars (Figs. 1 to 4), indicate ample genetic variation for virulence and resistance in the wheat-*M. graminicola* pathosystem. Comparison of these dendrograms revealed significant discrepancies between isolate and cultivar clusters for the two response parameters, i.e., entries that constitute a cluster for *N* do not necessarily form a similar cluster for *P* (Tables 3 to 6 and 8).

Efficacy of resistance. The bread wheat and durum wheat cultivars were grouped according to their response to the bread

wheat- and durum wheat-derived isolates in experiments 1 and 2, respectively. Three response classes, generally susceptible, generally resistant, and differentially resistant, were composed to evaluate the efficacy of the resistance in those cultivars in the different countries, since the number of isolates per country was too small for reliable virulence frequency calculations (Table 9). Apparently, the majority of the bread wheat isolates, except those from Dutch origin, carried virulence for the cultivars Lakhish, Shafir, Gerek 79, and Ceeon. The cultivars Kavkaz, Beth Lehem, Bobwhite, Kavkaz/K4500 l.6.a.4, and Iassul 20 were considered to be appropriate sources for resistance, though their efficacy for Turkey, Kenya, Argentina, and Netherlands may be limited since virulent isolates were found in these countries. Comparison of the responses of 'Kavkaz' and its derivatives 'Kavkaz/K4500 l.6.a.4', 'Kavkaz/7C', and 'Kavkaz/UP301' revealed generally decreased resistance levels in the two latter derivatives, whereas the cross between 'Kavkaz' and 'K4500 l.6.a.4', which resulted in the line 'Kavkaz/K4500 l.6.a.4', had a significantly increased level of resistance (Tables 3, 4, and 9). When considering the number of cultivars that were generally resistant, isolates originating from Burundi, Rwanda, Uganda, and Uruguay were virulent on fewer cultivars than isolates from other countries (Table 9). However, this may well have coincided with the small number of isolates that originated from these countries, since an increase of isolates would probably result in more cultivars with a differential response. Observed virulences and the origin of the cultivars were

TABLE 3. Adjusted necrosis (*N*) response matrix of experiment 1; 24 host accessions and 50 *Mycosphaerella graminicola* isolates, arranged according to the clusters of Figure 1[v]

	Cultivars[x]																							
	B	B	B	B	B	B	B	D	D	B	D	T	B	D	B	B	B	B	B	B	B	B	B	B
EC[w]	La	Sh	Co	To	Ge	Ia	Ce	Vo	Et	BL	Ca	Be	Bo	In	T29	KT	Ol	Ob	Ar	Ve	KK	KU	KZ	K7
TK3	69	64	49	56	76	50	55	45	64	57	49	30	22	63	84	83	60	51	67	40	20	13	29	21
TK4	78	57	62	64	78	55	67	41	61	52	58	35	25	63	83	87	58	59	52	55	20	7	22	27
UR5	59	71	53	66	77	54	63	50	43	47	57	18	39	69	71	79	72	62	33	49	15	9	23	12
UR3	51	67	32	47	65	56	58	30	45	43	58	26	26	54	79	79	54	36	24	19	5	5	16	14
ET11	44	38	39	31	72	27	28	38	51	18	34	18	6	35	77	67	29	49	48	23	5	6	3	23
AR6	50	57	28	54	75	48	54	44	16	49	48	7	10	52	72	76	53	54	50	69	1	1	10	6
AR3	62	72	21	32	82	65	65	31	32	44	44	7	9	5	82	81	56	56	53	68	–1[y]	0	8	5
AR4	59	69	41	36	80	57	62	25	42	38	49	24	27	5	74	79	63	43	49	52	1	0	8	18
UR2	42	64	31	38	77	52	57	29	45	34	45	16	21	19	81	88	56	71	58	40	12	6	5	17
TK9	71	73	30	30	83	35	63	21	26	21	53	18	12	18	90	85	49	35	38	38	7	9	11	13
AR1	53	71	64	60	79	58	63	41	54	43	61	57	38	5	76	80	60	51	44	38	13	7	5	24
TK6	47	61	58	53	79	42	27	46	34	37	60	10	15	25	74	83	60	53	21	45	8	6	11	9
UR1	54	66	30	48	82	37	50	35	20	38	50	10	40	18	87	86	69	29	14	11	3	9	12	10
ET5	46	67	52	63	77	52	31	34	50	28	42	13	13	42	43	23	16	21	11	17	15	15	20	13
ET10	55	67	58	79	79	58	48	67	47	35	55	31	14	64	78	55	8	64	17	74	19	10	26	19
TK5[z]	7	14	47	15	33	29	2	68	71	17	70	23	14	61	54	28	7	10	3	41	5	9	8	10
NL5	24	33	41	50	23	22	21	11	16	25	35	6	22	33	75	79	29	56	43	17	18	19	54	5
NL1	38	46	48	55	25	26	43	23	44	32	59	13	33	42	85	84	49	67	57	32	34	28	73	10
ET3[z]	68	62	44	69	69	52	42	41	37	34	50	9	34	40	80	75	39	69	35	52	51	40	61	60
ET2	65	69	66	69	77	52	56	44	50	36	55	30	34	55	80	84	51	81	56	50	58	50	72	65
ET4	49	62	36	65	73	47	21	32	22	30	50	9	22	43	78	76	47	68	26	46	29	37	49	51
ET1	72	72	62	87	86	57	54	50	40	47	59	27	37	69	89	82	63	85	76	69	34	45	70	83
ET7	58	64	65	64	71	62	45	48	51	32	63	0	33	56	78	79	57	54	45	73	37	58	71	73
NL2	43	55	50	76	43	42	41	37	15	31	50	29	42	57	84	84	58	71	65	69	36	47	81	68
BU2	59	69	69	74	75	74	59	24	35	37	50	26	7	50	83	75	65	65	60	73	28	25	46	55
TK1	66	66	49	67	65	47	66	27	47	44	49	13	12	33	80	81	46	68	65	51	5	21	30	42
NL4	45	36	35	15	78	58	31	21	53	31	38	–5	13	48	78	78	37	62	60	–4	26	42	43	56
NL6	48	57	42	80	78	35	47	27	56	42	62	24	43	80	88	87	52	82	75	14	36	61	87	67
NL3	6	14	33	4	81	57	60	28	44	11	36	–1	2	13	71	39	5	85	83	0	0	6	38	7
UR4	52	68	55	62	74	34	52	14	19	44	47	11	17	39	62	79	47	53	30	5	14	41	61	51
KE4	53	56	56	71	62	27	45	28	15	33	48	6	26	44	60	63	63	59	31	5	22	34	63	48
KE5	57	61	41	62	77	40	41	27	19	23	51	2	24	34	70	86	51	51	38	7	10	32	74	58
KE2	57	73	52	65	77	48	59	41	25	40	43	8	28	48	85	75	71	71	37	32	42	41	73	80
KE6	49	68	58	80	71	58	48	34	22	51	50	6	11	56	71	78	61	55	34	–6	24	14	58	69
KE8	75	74	65	85	86	55	70	26	35	39	74	22	21	64	85	91	56	66	39	11	25	38	61	62
RW1	47	71	66	88	69	32	61	16	24	39	48	9	25	66	86	86	60	62	12	10	17	56	81	69
KE7	61	66	66	75	70	46	55	21	50	42	57	28	22	59	69	71	48	53	28	10	29	31	71	62
KE1	63	70	66	80	82	53	57	38	39	47	57	41	39	52	76	80	66	46	30	8	26	29	66	64
UG1	68	68	61	70	74	37	55	28	57	31	54	23	28	41	80	76	67	56	23	10	27	52	63	69
TK2	81	67	66	75	77	47	50	31	47	45	52	25	36	43	81	76	62	62	32	42	26	26	51	56
BU1	65	69	56	63	80	38	62	24	35	34	35	18	26	45	81	72	55	43	20	34	10	30	56	61
KE3	64	74	59	78	86	35	50	21	53	36	61	25	15	41	61	74	49	60	26	17	20	25	56	25
ET8	69	78	68	78	80	45	73	45	59	39	59	32	47	71	87	80	59	64	15	39	66	51	70	79
ET6	52	56	54	78	60	20	46	59	34	32	54	9	34	55	73	75	48	57	13	10	30	44	71	61
TK7	67	53	38	62	69	49	55	56	44	51	60	24	23	61	75	69	54	42	22	45	33	33	66	60
TK10	51	51	54	62	69	38	38	47	39	42	60	25	14	54	80	42	7	74	10	51	64	33	71	59
TK8	64	53	41	73	70	55	39	60	58	53	63	27	18	59	72	81	12	69	29	16	31	40	73	57
ET9	55	79	59	75	84	56	59	60	37	36	43	14	16	46	66	52	13	29	10	15	63	46	58	70
AR2	67	64	50	60	71	56	61	62	42	48	63	53	38	30	75	79	59	59	29	28	67	46	59	64
AR5	79	80	41	51	75	38	55	74	32	50	42	28	62	32	86	75	70	60	26	12	68	68	67	73

[v] $LSD_{0.01} = 29$, $LSD_{0.05} = 22$.
[w] Experimental codes for isolates according to Table 2.
[x] Experimental codes for cultivars according to Table 1; B = bread wheat, D = durum wheat, and T = triticale.
[y] Negative values are because of block adjustments.
[z] Durum wheat-derived isolates.

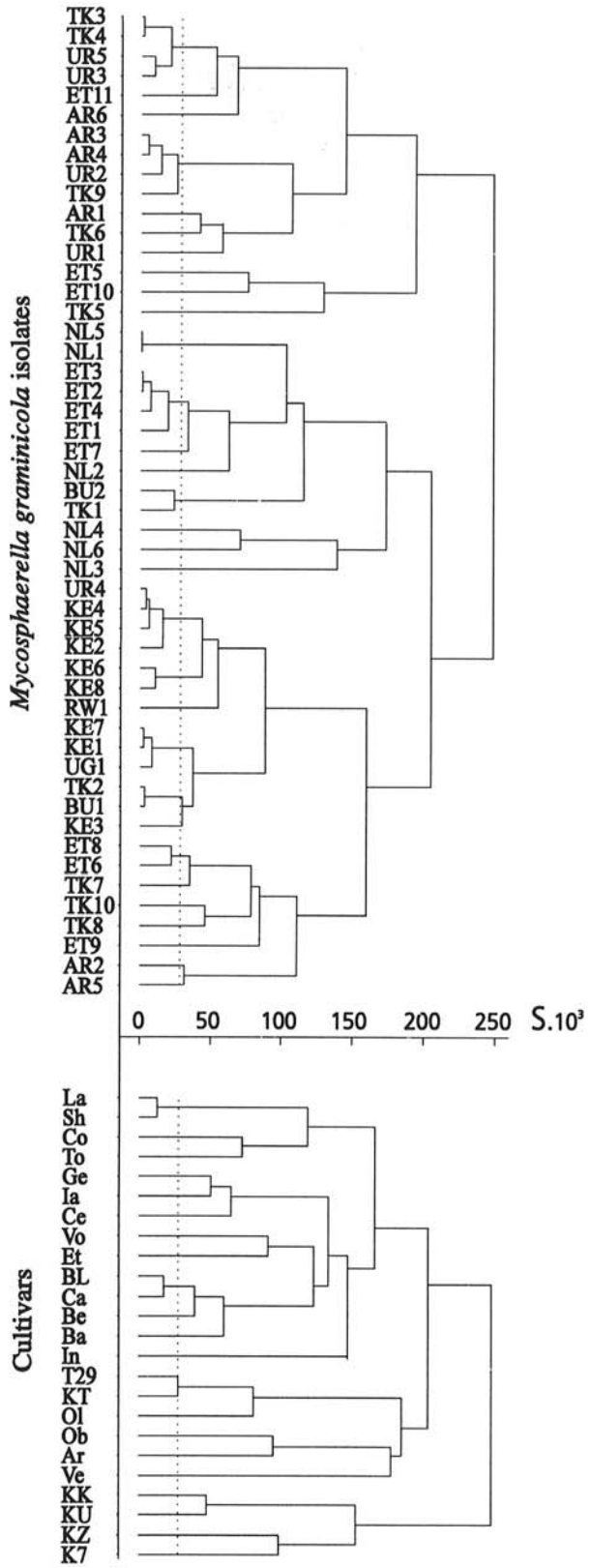

Fig. 1. Dendrograms of simultaneously clustered genotypes of wheat (23) and triticale (1), and *Mycosphaerella graminicola* isolates (50), based on *N* in experiment 1. The positions of the nodes correspond with the cumulative sum of squares for interaction between cultivars and isolates (*S*) on the horizontal axis. The area at the left of the vertical dotted line represents nonsignificant differences at $P = 0.05$, $S = 28.12 \times 10^3$ (for $P = 0.01$, $S = 29.78 \times 10^3$).

not always evident. The resistance in the Dutch cultivars Arminda and Obelisk, for example, was circumvented by several isolates from Turkey, Ethiopia, Uruguay, and Argentina, though these cultivars were never exposed to *M. graminicola* outside Europe by commercial cultivation. The efficacy of resistance in the durum wheat cultivars appeared to be limited, particularly to the isolates from Morocco and Syria which were extremely virulent. The *T. turgidum* subsp. *dicoccoides* accession 'G25' was the only entry with a reasonable level of resistance to the majority of the durum wheat-derived isolates.

DISCUSSION

Genetic variation for virulence. Extensive genetic variation for virulence in *M. graminicola*, characterized by differential interactions between host and pathogen genotypes for both *N* and *P*, suggested the involvement of specific factors for virulence and resistance in this pathosystem. Specificity in necrotrophic pathogens, such as *Stagonospora nodorum*, *Pyrenophora tritici-repentis*, *Rhynchosporium secalis* (on barley), and *Setosphaeria turcica* (on maize), was reported previously (21,22,32,35) but was considered to be controversial in *M. graminicola*. While some experimental evidence supported its existence (2,10,11,12,33,44), other evidence did not (26,43). Hence, Johnson (17) made the statement that "it appears that a gene-for-gene interaction cannot be identified, at least with present techniques". A major element in this controversy seems to be the limited evidence for differential interactions between host and pathogen genotypes (10,11,12,17,31,44). The most obvious differential interactions reported so far occurred between bread and durum wheat and isolates secured from these species (10,33). An analogous observation was reported by Van Ginkel and Scharen (43), who, therefore, considered specialization in *M. graminicola* on bread or durum wheat to be of much greater importance than differential specificity on particular cultivars of these species. Indeed, when considering *P* in the present study, bread wheat and durum wheat isolates were particularly virulent on bread wheat and durum wheat cultivars, respectively. However, in addition, highly significant interactions within each of these systems were determined through analyses of restricted data matrices, either for bread wheat cultivars and bread wheat-adapted *M. graminicola* isolates or durum wheat cultivars and durum wheat-adapted isolates. This result was in contrast to that of Van Ginkel and Scharen (43), who did not find interactions between host and pathogen genotypes in their experiments that primarily dealt with durum wheat cultivars. They, therefore, suggested that interactions may disappear whenever considering a restricted system, i.e., bread wheat or durum wheat with their respective isolates. To strengthen the insignificance of isolate × cultivar interaction, they also discussed the relative proportion of the total variance that was attributed to main effects because of cultivars, isolates, and interaction in their own experiments and in the experiments of others (43). Kema et al. (19), however, discussed diverse statistical approaches that were also employed to analyze additional data, and considered it to be incorrect to question the proportion of the MS_{int} as long as it is statistically significant.

The wide genetic variation for virulence in *M. graminicola* complemented information on genetic variation revealed by RFLP analyses (4). McDonald and Martinez (24,25) observed a high frequency of RFLPs in a sample of *M. graminicola* isolates that was mainly secured from one limited area, indicating substantial genetic variation within local populations and even between isolates derived from lesions in the same leaf. Indeed, separation of two Kenyan isolates, that originated from the same leaf (*IPO*87000-KE1 and *IPO*87011-KE2), in significantly different clusters indicated the presence of genetic variation for virulence at micro levels. Boeger et al. (4) suggested that common alleles, as defined by probe/restriction enzyme combinations, in very

distant *M. graminicola* populations were either because of seed transmission of the pathogen or the employment of anonymous DNA probes that hybridize to conserved noncoding regions of the genome. In our study, certain cultivars were susceptible to *M. graminicola* isolates that originated from regions in which these cultivars were never exposed to the pathogen. Parallel evolution of the pathogen population to imported resistance factors might possibly explain such observations. Since pathogen populations may, at least partly, be structured by gene-for-gene coevolution (39), an integrated analysis of local pathogen populations using molecular markers and selectable markers such as virulence would reveal the most useful information for breeding programs with respect to the magnitude and stability of genetic variation for virulence. In that case, adult plant inoculation experiments should also be considered, since seedling responses do not necessarily correlate with adult plant responses because resistance factors may operate in only one of these physiological stages. Adult plant inoculation experiments in the field also showed big cultivar × isolate interactions, and thus confirmed one of the main inferences from the present study that specificity appeared to be an important aspect of the wheat-*M. graminicola* pathosystem (G. H. J. Kema, *unpublished data*).

Races in *M. graminicola*? The occurrence of differential interactions between host and pathogen genotypes suggested a gene-for-gene relationship between these genotypes. However, the pathosystem discussed in the present contribution was far from the

TABLE 4. Adjusted pycnidia (*P*) response matrix of experiment 1; 24 host accessions and 50 *Mycosphaerella graminicola* isolates, arranged according to the clusters of Figure 2[v]

	Cultivars[x]																							
	D	D	D	D	B	B	B	B	B	B	T	B	B	B	B	B	B	B	B	B	B	B	B	B
EC[w]	Vo	Et	Ca	In	KZ	KU	K7	Co	To	Ia	Be	BL	Ar	Bo	KK	Ve	Ob	T29	La	Ce	Ge	Sh	KT	Ol
TK5[y]	53	50	41	40	−2[z]	−2	−2	−2	−1	−2	−2	−2	−2	−2	−2	11	−2	0	−2	−2	−2	−2	0	−1
NL5	1	1	1	1	8	4	3	1	2	1	1	1	4	5	1	5	8	25	11	9	1	2	30	6
NL1	−1	−1	−1	0	8	0	0	−1	2	−1	−1	0	7	3	−1	7	10	21	20	14	−1	12	20	18
BU2	1	1	1	1	8	5	8	12	20	6	1	4	5	1	1	25	15	23	21	23	35	21	27	17
ET11	0	0	1	3	0	0	0	2	4	0	0	0	10	0	0	1	26	34	32	17	36	25	27	14
NL4	−2	−2	0	−2	−2	6	8	−2	−2	−1	−2	−2	5	−1	−1	−2	32	17	17	5	16	3	45	0
NL6	0	0	0	7	14	22	26	1	21	2	0	5	7	11	1	0	44	14	20	24	35	15	35	9
NL3	−1	−1	−1	0	2	0	0	−1	0	1	−1	−1	25	−1	−1	−1	38	4	1	32	48	0	18	0
ET10	20	9	10	21	0	1	0	14	53	19	1	9	0	0	2	59	38	57	39	34	64	55	9	1
ET9	4	2	1	5	21	26	39	28	35	8	1	5	1	1	19	2	9	14	40	41	54	55	14	2
ET5	2	2	12	11	1	2	1	9	21	13	1	4	1	1	6	1	2	4	39	21	52	52	7	2
KE7	3	4	3	3	14	8	21	21	21	4	3	5	3	5	4	3	7	11	23	30	34	29	29	26
KE3	5	4	4	5	11	7	9	17	20	5	4	4	4	4	4	4	14	8	34	28	45	28	27	27
KE1	2	3	2	4	4	3	9	16	9	2	2	6	2	6	2	2	6	7	29	18	32	27	36	20
KE6	1	0	1	1	12	5	10	14	15	0	0	8	0	0	7	0	21	6	33	35	12	34	33	33
UR4	0	−1	0	0	19	11	14	12	15	0	0	1	0	0	1	0	11	16	33	32	43	37	46	33
KE4	0	0	3	0	19	10	23	20	34	0	0	4	0	1	3	0	18	14	35	31	36	36	37	45
KE8	0	0	1	1	4	11	16	22	36	0	0	5	10	2	1	0	10	22	50	40	40	47	44	35
KE5	−1	−1	−1	−1	16	12	42	19	27	1	−1	1	1	2	−1	0	20	13	43	28	39	43	64	30
ET6	10	0	5	14	23	28	39	31	32	1	0	4	0	22	4	1	14	38	41	37	39	43	38	32
UG1	0	0	0	0	19	25	44	30	36	2	0	0	0	0	6	0	9	27	36	35	38	51	34	25
RW1	0	−1	0	15	31	37	40	25	43	0	0	0	0	1	2	1	17	39	34	43	34	47	63	32
TK8	1	2	9	16	23	28	42	12	51	5	1	8	4	7	7	1	41	38	53	22	38	44	73	1
TK2	1	1	1	1	1	6	8	19	16	4	1	2	1	2	1	4	3	37	40	21	36	40	33	29
BU1	−1	−4	−1	−4	19	9	10	31	33	0	−1	0	−1	7	−1	18	10	38	43	43	49	47	42	35
ET7	1	0	10	12	10	24	14	30	24	7	−1	0	11	16	0	40	4	28	51	30	39	47	40	37
ET4	0	1	7	4	11	19	36	2	26	5	0	3	2	9	3	28	51	45	34	14	29	45	37	25
ET1	0	−1	1	6	14	14	39	9	22	8	0	1	9	7	1	45	43	52	43	25	41	43	44	30
ET3[y]	10	0	2	8	11	13	24	6	29	1	0	0	6	16	9	21	51	40	51	25	46	50	58	19
ET2	0	0	4	4	19	21	26	12	16	17	0	1	4	20	22	30	39	40	54	44	43	57	52	23
TK10	0	0	3	1	14	12	47	17	14	4	0	0	0	0	30	35	53	40	22	31	58	33	0	0
NL2	−3	0	1	6	42	25	44	6	28	1	0	5	30	27	8	45	54	55	14	31	9	43	61	45
TK7	1	1	2	14	22	19	31	5	16	1	1	26	1	2	3	15	16	41	54	42	37	36	52	30
KE2	0	0	0	0	31	22	39	2	18	0	0	10	7	7	7	12	37	36	47	38	46	42	42	41
ET8	0	3	1	1	10	19	21	1	16	2	1	1	1	20	22	12	23	25	40	39	34	26	34	29
AR5	7	0	0	7	8	38	30	4	3	2	1	15	0	38	20	1	20	40	41	35	26	48	35	43
AR2	2	2	2	2	10	32	41	3	12	6	4	12	3	29	35	7	37	46	64	56	53	60	54	54
TK3	1	0	1	21	1	1	1	12	20	8	2	19	26	9	1	1	34	66	55	43	54	47	72	29
TK4	4	4	5	28	4	4	4	19	31	17	3	33	23	9	3	6	38	62	69	59	67	53	66	33
AR3	−1	−1	0	−1	−1	−1	−1	0	0	16	−1	10	27	1	−1	40	24	58	57	54	65	52	64	51
AR4	2	2	2	2	2	2	2	8	10	28	2	9	24	18	2	41	22	48	44	54	68	54	57	48
TK9	0	0	0	1	0	0	0	1	5	2	0	0	7	1	0	22	20	57	54	47	57	52	57	29
AR6	−2	−2	−2	−1	−2	−1	1	0	0	14	−2	4	6	−1	−2	16	27	12	30	36	43	42	44	27
UR2	3	6	3	8	3	3	3	5	3	12	3	4	24	8	3	13	42	41	32	41	50	50	47	30
TK1	3	1	14	16	11	8	7	11	37	9	2	13	31	2	2	32	46	33	54	57	50	49	51	33
TK6	2	2	5	4	2	2	2	14	12	10	2	6	3	9	2	28	33	44	22	15	52	51	61	54
AR1	4	3	3	3	4	4	3	4	4	4	3	15	5	25	3	7	12	23	26	34	45	43	39	38
UR1	0	0	0	4	0	0	0	0	1	0	0	3	0	18	0	0	0	26	30	36	45	48	61	47
UR5	−3	−3	−3	5	−3	−3	−3	14	5	−3	−3	0	−3	15	−3	16	20	25	21	39	34	48	60	45
UR3	2	1	1	13	1	1	1	8	11	1	6	25	2	20	1	8	22	45	45	52	43	66	62	44

[v] $LSD_{0.01} = 20$, $LSD_{0.05} = 15$.
[w] Experimental codes for isolates according to Table 2.
[x] Experimental codes for cultivars according to Table 1; B = bread wheat, D = durum wheat, and T = triticale.
[y] Durum wheat-derived isolates.
[z] Negative values are because of block adjustments.

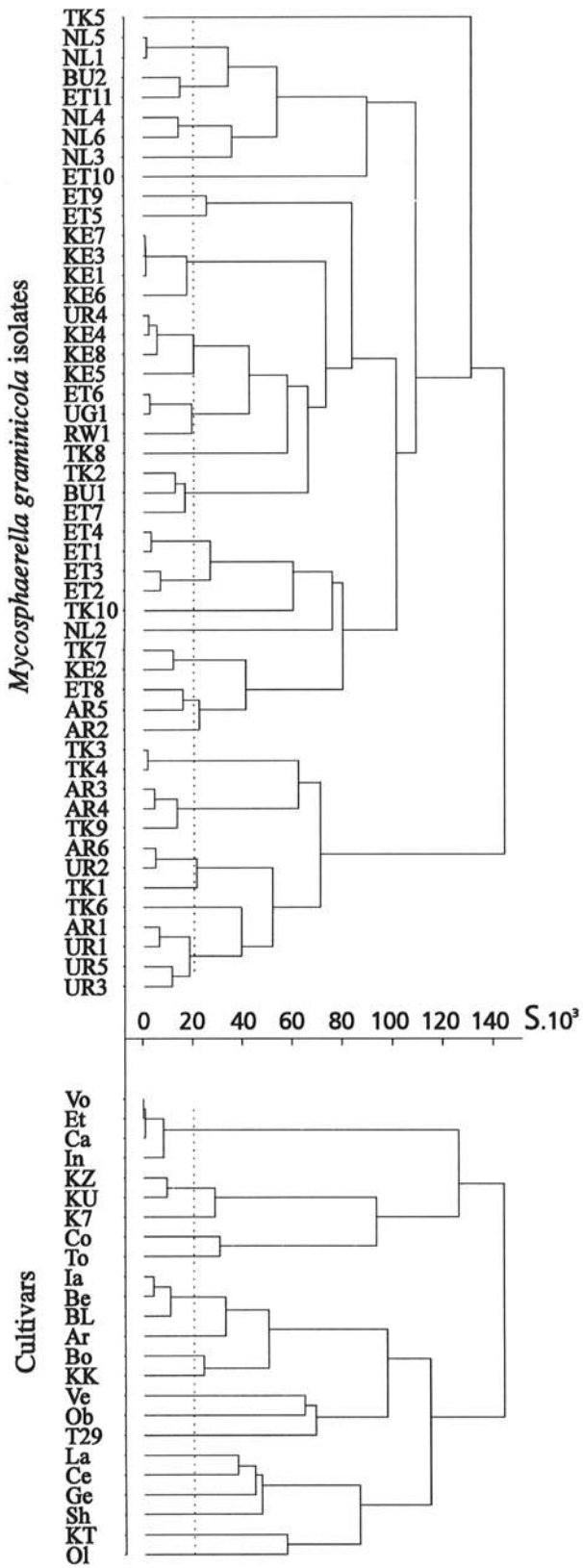

Fig. 2. Dendrograms of simultaneously clustered genotypes of wheat (23) and triticale (1), and *Mycosphaerella graminicola* isolates (50), based on *P* in experiment 1. The positions of the nodes correspond with the cumulative sum of squares for interaction between cultivars and isolates (*S*) on the horizontal axis. The area at the left of the vertical dotted line represents nonsignificant differences at $P = 0.05$, $S = 20.12 \times 10^3$ (for $P = 0.01$, $S = 21.17 \times 10^3$).

ideal gene-for-gene system as discussed by Person (28), which requires a locus in the host that governs either a resistant or a susceptible response, and a locus in the pathogen that governs a virulent or an avirulent response. Hence, the analysis of data for *M. graminicola* that has a more quantitative character as compared with cereal rusts and powdery mildews, for example, cannot be performed sufficiently using the method proposed by Person (28). In the current study, gene-for-gene interaction in the *M. graminicola*-wheat pathosystem was inferred from significant MS_{int} values in ANCOVAs, from cluster analyses that employed such values as proximity measures for isolates and cultivars in consecutive analyses of variance, and from other statistical procedures (19). Eyal and coworkers (11,12,44) developed an elaborate statistical procedure, that was also adopted by Van Ginkel and Scharen (42,43), to calculate cutpoints in order to assign qualitative descriptors, resistant or susceptible, to quantitative data, either *N* or *P*. This procedure enabled the designation of hypothetical resistance and virulence genes to host cultivars and pathogen isolates, respectively, and analysis of the data as suggested by Person (28). However, the procedure disregarded additive modes of action in virulence and resistance, which results in inadequate assignment of hypothetical virulence and resistance factors. Therefore, this procedure was not considered.

The occurrence of differential interactions justified the recognition of physiological races in plant pathology. In case resistance in the host is largely quantitatively inherited, virulence differences among fungal strains might be of insufficient magnitude to distinguish distinct pathogen races. Race designation would be even more complicated, if not impossible, if such pathogens had a functional generative stage combined with a relatively efficient dissemination mechanism as compared with the dispersal of asexual propagules, as in *M. graminicola*. Caten (7) argued that extensive genetic variation for virulence and gene flow between populations of the pathogen would lead to the designation of a separate race identity to virtually each individual isolate. Indeed, such a situation is conceivable for *M. graminicola*, particularly when considering recombination during ascosporogenesis. Hence, nomenclature of races in *M. graminicola* is fairly trivial. However, the designation of a bread wheat and a durum wheat variant in *M. graminicola* is of importance. Both types could be easily recognized in inoculation experiments, but did not differ morphologically and were not geographically isolated. In addition, the two *M. graminicola* variants could not be distinguished by amplification and digestion of nuclear and mitochondrial internally transcribed spacer ribosomal DNA (ITS rDNA) (E. C. P. Verstappen and G. H. J. Kema, *unpublished data*). The sequences of amplified ITS fragments of both variants appeared to be identical (E. C. P. Verstappen, A. Lever, J. Keijer, and G. H. J. Kema, *unpublished data*) and were also similar to the sequence of *M. graminicola* isolate ATCC 26517 (American Type Culture Collection accession) as was recently published (3), which supported the idea that both variants were from a similar taxonomic rank. A similar situation was recently described for wheat leaf rust, in which the durum wheat and bread wheat types clearly differ in pathogenicity and also appear to be sexually isolated, but could not be distinguished by molecular markers (1,13,45). The presence of both *M. graminicola* variants at the same location (e.g., TK5 and TK6 in Altinova, Turkey; and TN1, TN2, and TN3 in Beja, Tunisia) emphasized the importance of population dynamics studies (4), particularly since *M. graminicola* is of increasing importance in the region.

Although the major inference of the present study was the specificity of the host-pathogen interaction, which was irrespective of the proposed variants, the suggested gene-for-gene relationship as the underlying mechanism for this requires further evidence through crossing experiments among accessions of host and pathogen.

Genetic variation for resistance. The resistance in the host cultivars varied widely in both experiments. In the first experi-

ment, the durum wheat cultivars were in one group, since the majority of the isolates hardly produced pycnidia in them. In contrast, most of the bread wheat cultivars were significantly different for *N* and *P*. However, the relationship between some cultivars was evident from composed clusters, such as 'Kavkaz' and its derivatives 'Kavkaz/UP301' and 'Kavkaz/7C'. Another composed cluster contained 'Iassul 20', 'Bet Lehem', and the triticale cultivar Beagle, which were apparently unrelated but had low *P* levels with the majority of the isolates. Cultivars such as 'Kavkaz', 'Bobwhite', 'Kavkaz/K4500 l.6.a.4', and 'Iassul 20' proved to be highly effective against the majority of the isolates, in accord with Eyal et al. (12).

In the second experiment, 'OZ 368' and 'Bidi 17' had a similar differential response to the *M. graminicola* isolates and were clustered. The land race 'OZ 368' was selected by Ducellier in 1936 from the land race population 'Bidi' in the region Oued Zenati, 40 km west of Guelma in Algeria, whereas 'Bidi 17' was selected by Perrot from the same land race population in 1938 in Guelma (9). A parallel inference was evident for 'Hedba 3' and 'M. B. Bachir', that were selected in 1907 from unknown, but probably similar, land race populations in the region of Setif, 300 km southeast of Algers (9).

The relation between *N* and *P*. Our study considered two disease parameters, *N* and *P*, whereas other reports on pathological variation in *M. graminicola* considered either one or the other of these parameters (2,10,11,12,33,43). The smaller standard error of the mean for *P* provided a better resolution of genetic variation than *N*, which was also evident from the cluster analyses. *P* resulted in more pronounced differences between isolates or cultivars. Therefore, *P* appeared, apart from its epidemiological relevance, to be most appropriate to characterize isolates or cultivars. The controversy about host-pathogen interactions in the wheat-*M. graminicola* pathosystem might be partly because of the analysis of just one parameter, which obviously mitigates the complexity

TABLE 5. Adjusted necrosis (*N*) response matrix of experiment 2; 23 host accessions and 15 *Mycosphaerella graminicola* isolates, arranged according to the clusters of Figure 3[w]

	Cultivars[y]																						
	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	B	B	B	B	D	T
EC[x]	I69	ZB	BD	OZ	B17	H3	ZP	MB	In	Wa	OR	Te	Ma	G25	Jo	A65	Sa	La	An	Bo	KK	Cc	Be
TN5	84	82	85	82	69	83	83	72	69	68	62	76	72	63	72	69	71	1	−2[z]	−1	−4	77	1
TN1	77	95	83	100	86	93	78	78	87	78	66	78	76	66	74	67	77	8	8	5	3	78	8
MO2	86	85	75	78	74	71	76	68	69	54	55	70	69	59	59	49	63	6	5	3	0	62	9
TN3	97	97	92	86	74	90	83	84	78	80	72	84	90	54	61	72	68	7	7	2	4	90	5
SY1	84	80	66	85	65	85	79	68	82	64	56	81	82	44	65	83	75	10	5	6	2	62	7
TN4	80	79	75	88	82	84	88	90	89	81	70	83	86	65	83	84	82	28	29	3	4	84	4
MO1	74	82	94	98	90	95	85	94	90	64	62	75	81	53	90	87	90	11	7	9	3	95	32
TN7	51	65	87	54	31	79	50	65	68	49	48	60	72	44	47	31	47	3	5	5	4	59	10
MO4	82	101[z]	103	98	72	95	86	80	63	47	55	79	99	57	74	52	49	11	8	6	7	83	5
TN6	85	104	83	92	78	98	80	79	58	60	36	63	86	55	66	46	47	10	9	6	6	66	8
TK5	77	73	58	42	32	33	43	29	57	59	60	67	70	56	43	35	47	11	12	14	9	71	26
MO3	18	35	19	41	16	14	20	17	13	11	8	9	43	37	11	14	37	9	62	11	8	58	6
ET6	49	55	63	60	37	53	63	56	66	69	63	76	71	64	66	51	56	68	74	52	53	80	36
TN2	48	65	44	51	28	43	54	40	87	61	71	82	70	40	60	39	52	78	91	19	26	86	23
AL1	14	31	43	42	13	32	24	15	68	68	39	57	43	21	24	24	8	75	71	37	5	80	11

[w] $LSD_{0.01} = 29$, $LSD_{0.05} = 20$.
[x] Experimental codes for isolates according to Table 2.
[y] Experimental codes for cultivars according to Table 1; B = bread wheat, D = durum wheat, and T = triticale.
[z] Values >100 and <0 are because of block adjustments.

TABLE 6. Adjusted pycnidia (*P*) response matrix of experiment 2; 23 host accessions and 15 *Mycosphaerella graminicola* isolates, arranged according to the clusters of Figure 2[w]

	Cultivars[y]																						
	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D	B	T	B	B	B
EC[x]	Wa	OR	Ma	Te	ZB	OZ	B17	ZP	H3	MB	I69	BD	In	Cc	Jo	A65	Sa	G25	Bo	Be	KK	La	An
MO1	56	53	53	48	46	51	57	35	58	65	46	61	68	68	62	62	67	20	0	0	0	0	1
TN1	34	40	31	32	61	48	59	29	60	51	28	36	75	63	58	65	62	20	0	0	0	0	0
TN5	14	13	62	69	33	13	19	59	54	48	32	46	76	70	60	67	56	6	−1[z]	−1	−1	−1	−1
MO4	47	48	71	66	45	51	53	54	60	55	33	41	73	58	66	38	39	17	2	2	2	2	2
MO2	42	57	67	67	57	55	69	68	66	56	26	25	74	47	54	46	56	16	−1	−1	−1	−1	−1
TN3	73	70	67	69	72	49	55	54	67	62	33	45	73	50	63	57	60	18	−1	−1	−1	−1	0
TN4	70	61	76	77	63	31	23	69	83	70	24	20	72	59	61	66	48	7	−1	−1	−1	−1	−1
SY1	34	34	63	69	58	58	49	68	67	62	44	38	76	55	59	52	51	12	0	0	0	0	0
TN6	32	16	58	55	53	55	50	60	63	58	40	32	51	48	59	37	45	6	0	0	0	0	0
TN7	47	54	69	64	8	6	6	12	49	57	6	14	62	53	54	32	43	4	2	2	2	2	2
TK5	60	61	34	64	18	5	3	4	5	3	5	4	63	14	24	20	35	5	3	3	3	3	3
TN2	0	13	−2	1	0	−2	−2	−2	−2	−2	−2	−1	10	−2	−1	−2	−2	3	5	−2	0	67	47
ET6	3	21	5	6	1	0	1	1	1	1	0	9	13	0	3	0	8	8	20	0	4	61	54
AL1	15	20	0	1	2	1	1	0	3	1	0	2	39	13	0	4	1	2	21	0	0	61	45
MO3	1	0	3	0	3	2	2	1	0	2	0	0	0	2	0	0	10	3	0	0	0	0	42

[w] $LSD_{0.01} = 17$, $LSD_{0.05} = 12$.
[x] Experimental codes for isolates according to Table 2.
[y] Experimental codes for cultivars according to Table 1; B = bread wheat, D = durum wheat, T = triticale.
[z] Negative values are because of block adjustments.

of the pathosystem. Suboptimal experimental conditions, in particular inadequate RH levels (23,37), may lead to a consideration of *N* as disease parameter, since pycnidia development will be severely hampered if RH levels are ≤75% or insufficiently controlled. Eyal et al. (12) and Van Ginkel and Scharen (41,42,43) considered *N* as their main disease parameter. In addition, the frequency of plants showing pycnidia was determined, sometimes 4 weeks after inoculation. Yechilevich-Auster et al. (44) reported a mean value of $P = 3.8$ on the resistant cultivar Zenati Bouteille, whereas it had a differential response ranging from $P = 7$ to $P = 72$ with merely durum wheat isolates in our experiments. Similarly, the susceptible cultivar Inbar, had a mean value of $P = 27.8$, whereas in our experiments it ranged from $P = 50$ to $P = 75$ for various isolates. We observed pycnidium formation already at 10 days after inoculation, and final observations were conducted at 21 days after inoculation. Although these discrepancies may be conferred by differences in virulence of the pathogen isolates, it was not surprising that dissimilar experimental conditions produced conflicting data, thus obscuring the discussion on specificity in this pathosystem (10,11,12,17,26,43).

Observed cluster discrepancies for *N* and *P* suggested that *N* and *P* were under different genetical control. Extensive leaf necrosis with no or a few pycnidia occurred frequently. High necrosis levels with varying pycnidial densities were also observed in field experiments (G. H. J. Kema, *unpublished data*). Therefore, Rosielle (30) introduced an assessment scale with six discrete host-response classes representing immunity and varying levels of necrosis and pycnidial density. The reason for dissimilarities between *N* and *P* classifications has, to our knowledge, not been addressed. Histological studies showed that phenotypes with high *N* but low *P* levels were not profusely colonized (G. H. J. Kema and D. Yu, *unpublished data*). These observations suggest that high *N* levels may be provoked by the pathogen, but apparently could imply avirulence rather than virulence. Therefore, *P* appears to be the most reliable disease parameter until histological and physiological aspects of the pathogenesis of *M. graminicola* in compatible and incompatible interactions have been resolved.

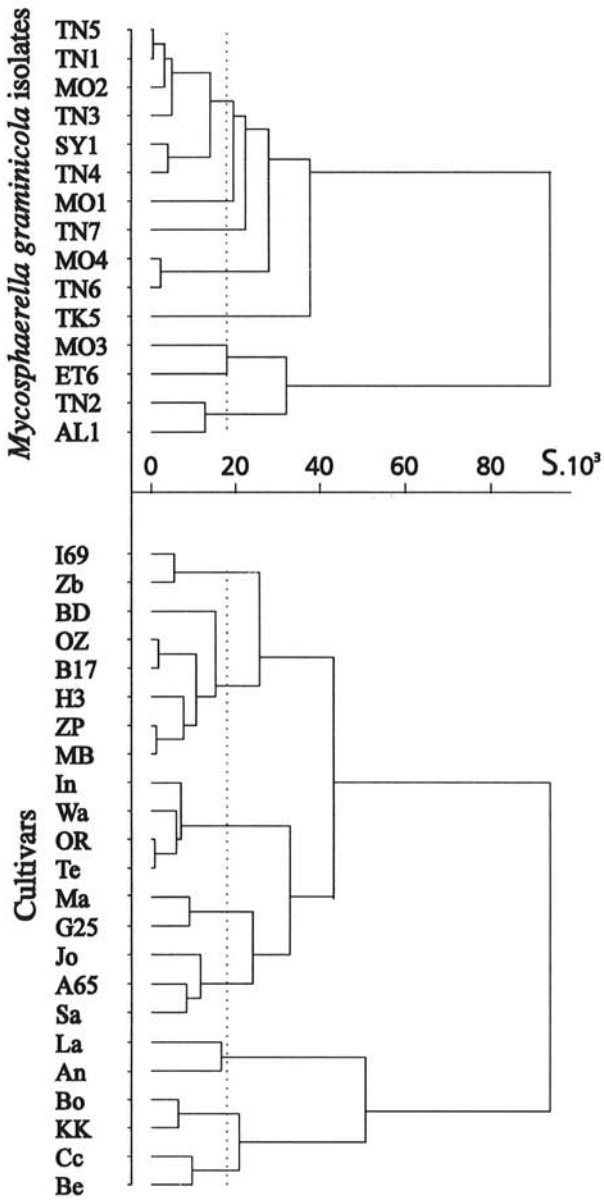

Fig. 3. Dendrograms of simultaneously clustered genotypes of wheat (22) and triticale (1), and *Mycosphaerella graminicola* isolates (15), based on *N* in experiment 2. The positions of the nodes correspond with the cumulative sum of squares for interaction between cultivars and isolates (*S*) on the horizontal axis. The area at the left of the vertical dotted line represents nonsignificant differences at $P = 0.05$, $S = 18 \times 10^3$ (for $P = 0.01$, $S = 20 \times 10^3$).

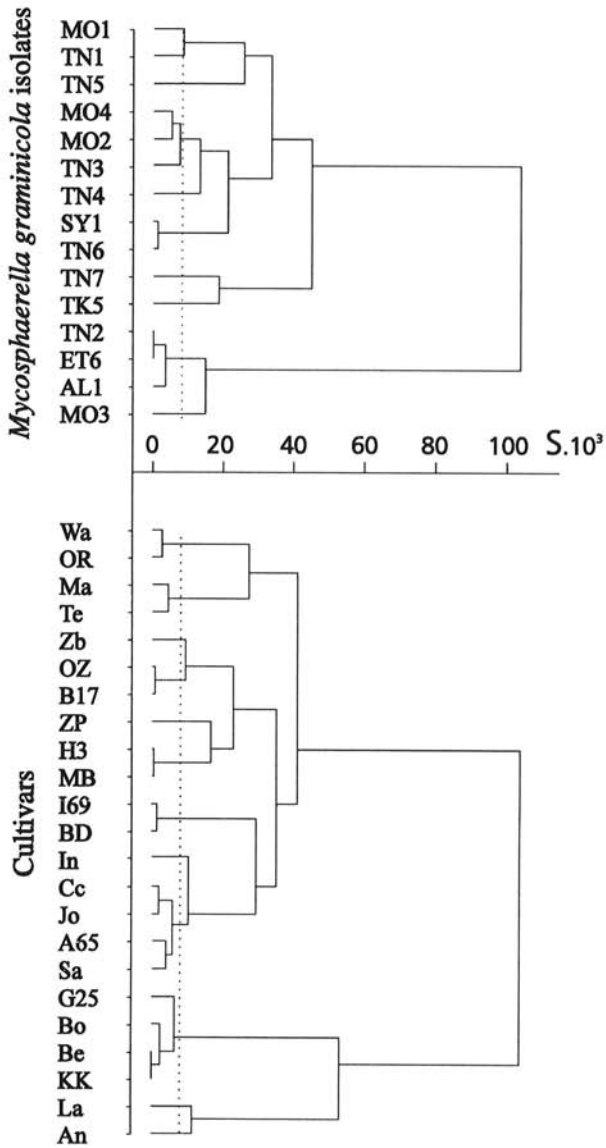

Fig. 4. Dendrograms of simultaneously clustered genotypes of wheat (22) and triticale (1), and *Mycosphaerella graminicola* isolates (15), based on *P* in experiment 2. The positions of the nodes correspond with the cumulative sum of squares for interaction between cultivars and isolates (*S*) on the horizontal axis. The area at the left of the vertical dotted line represents nonsignificant differences at $P = 0.05$, $S = 8 \times 10^3$ (for $P = 0.01$, $S = 8.5 \times 10^3$).

TABLE 7. Analyses of covariance of the necrosis (*N*) and pycnidia (*P*) disease parameters, on total and restricted response matrices in two experiments[p]

	Total response matrix			Restricted response matrix		
		N	*P*		*N*	*P*
Source of variation	df	MS[q]	MS	df	MS	MS
Experiment 1						
Isolates	49	5,440.5[r]	2,587.9[r]	47	4,317.2[r]	2,872.2[r]
Covariates	24	13,578.6	2,364.4	24	9,965.7	2,801.6
Mainplot error	124	1,324.3	331.7	118	1,098.7	377.7
Cultivars	23	47,725.6[r]	38,408.1[r]	18	49,551.2[r]	35,187.5[r]
Cultivars × isolates	1,127	864.0[r,s]	506.4[r,t]	846	882.5[r,u]	542.6[r,v]
Subplot error	3,468	197.9	108.5	2,556	182.5	125.0
Experiment 2						
Isolates	14	12,907.5[r]	20,110.0[r]	10	5,697.0[r]	5,173.2[r]
Covariates	9	14,054.8	1,144.0	9	6,811.5	1,746.6
Mainplot error	36	2,687.2	378.7	24	1,199.4	513.3
Cultivars	22	28,722.2[r]	14,530.0[r]	17	3,549.1[r]	7,864.8[r]
Cultivars × isolates	308	1,220.0[r,w]	1,347.0[r,x]	170	416.5[r,y]	730.4[r,z]
Subplot error	990	191.0	87.1	561	192.1	118.0

[p] The total response matrix of experiment 1 comprised 24 host accessions and 50 *Mycosphaerella graminicola* isolates, and the restricted response matrix was confined to 19 bread wheat accessions and 48 isolates from bread wheat. The total response matrix of experiment 2 comprised 23 host accessions and 15 *Mycosphaerella graminicola* isolates, and the restricted response matrix was confined to 18 durum wheat accessions and 11 isolates from durum wheat.
[q] MS = mean square.
[r] *F* value highly significant ($P < 0.01$).
[s] Percentage of the total variance is 2.04.
[t] Percentage of the total variance is 3.58.
[u] Percentage of the total variance is 2.33.
[v] Percentage of the total variance is 4.52.
[w] Percentage of the total variance is 1.24.
[x] Percentage of the total variance is 1.14.
[y] Percentage of the total variance is 1.33.
[z] Percentage of the total variance is 1.29.

TABLE 8. Categories of relationships between *Mycosphaerella graminicola* isolates and wheat cultivars[x]

			Response of cultivars[y,z]			
Category	Experiment	Isolate[y]	*N*		*P*	
Interactive *N*-interactive *P*	1		Ve	KU	Ve	KU
		TK8	15 a	70 b	1 a	42 b
		AR3	68 b	5 a	40 b	−1 a
	2		Te	MB	Te	MB
		SY1	81 a	68 a	69 a	62 a
		TK5	67 a	29 b	64 a	3 b
Noninteractive *N*-interactive *P*	1		T29	Ob	T29	Ob
		NL3	71 a	85 a	4 a	38 b
		TK2	85 a	62 a	37 b	3 a
	2		Wa	Cc	Wa	Cc
		TK5	59 a	71 a	60 a	14 b
		TN5	68 a	77 a	14 b	70 a
Interactive *N*-noninteractive *P*	1		Ve	KZ	Ve	KZ
		AR1	38 b	5 a	7 a	4 a
		NL3	0 a	38 b	−1 a	2 a
	2		I69	B17	I69	B17
		TN4	80 a	77 a	24 a	23 a
		TK5	77 a	32 b	5 a	3 a
Noninteractive *N*-noninteractive *P*	1		Ce	Ge	Ce	Ge
		TK4	67 a	78 a	59 a	67 a
		AR4	62 a	80 a	54 a	68 a
	2		Te	Ma	Te	Ma
		TN4	83 a	86 a	77 a	76 a
		MO4	79 a	99 a	66 a	71 a

[x] Values are taken from Tables 3 to 6.
[y] Experimental codes for cultivars according to Table 1 and experimental codes for isolates according to Table 2.
[z] Values followed by different letters are significantly different at $P < 0.01$ (experiment 1: $LSD_N = 29$ and $LSD_P = 20$; experiment 2: $LSD_N = 29$ and $LSD_P = 17$).

TABLE 9. Generalized efficacy of the resistance in bread wheat and durum wheat cultivars, with respect to the pycnidia parameter (*P*), to *Mycosphaerella graminicola* isolates that originated from these respective species

Country and number of isolates	Responses[x,y] of bread wheat cultivars[z] to bread wheat-derived isolates																		
	La	Sh	Ge	Ce	KT	Ol	T29	To	K7	KU	Co	KZ	Ob	Ve	Bo	Ar	BL	KK	Ia
Argentina (6)	●	●	●	●	●	●	○	R	○	○	R	R	○	○	○	○	R	○	○
Uruguay (5)	●	●	●	●	●	●	●	R	R	R	R	R	○	R	○	R	○	R	R
Ethiopia (11)	●	●	●	○	○	○	R	○	○	○	○	R	○	○	○	R	R	○	R
Kenya (8)	●	○	○	●	●	●	○	○	○	○	○	○	○	R	R	R	R	R	R
Burundi (2)	●	●	●	●	●	○	●	●	R	R	○	R	R	○	R	R	R	R	R
Rwanda (1)	●	●	●	●	●	●	●	●	●	●	●	●	R	R	R	R	R	R	R
Uganda (1)	●	●	●	●	●	●	●	●	●	●	●	R	R	R	R	R	R	R	R
Turkey (10)	●	●	●	●	○	○	●	○	○	○	R	○	○	○	R	○	○	R	R
Netherlands (6)	○	○	○	○	●	○	○	○	○	R	R	○	○	○	○	○	R	R	R

	Responses[x,y] of durum wheat cultivars[z] to durum wheat-derived isolates																	
	Ma	Te	In	Jo	A65	Sa	ZB	OR	Wa	OZ	B17	ZP	I69	BD	Cc	MB	H3	G25
Morocco (3)	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	○
Tunisia (6)	●	●	●	●	●	●	○	○	○	○	○	○	○	○	●	●	●	○
Syria (1)	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	R
Turkey (1)	●	●	●	●	●	●	●	●	●	●	R	R	R	R	R	R	R	R

[x] The restricted response matrix for bread wheat and bread wheat-derived isolates comprised 19 cultivars and 48 isolates. The restricted response matrix for durum wheat and durum wheat-derived isolates comprised 18 cultivars and 11 isolates.

[y] R = no virulent isolates encountered, ○ = virulent isolates encountered, and ● = all isolates carried virulence.

[z] Experimental codes for cultivars according to Table 1.

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