Evidence of Differential Interaction in the Polygenic Hordeum vulgare-Puccinia hordei Relation during Epidemic Development

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ABSTRACT

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The partial resistance of barley (*Hordeum vulgare*) to leaf rust (which is caused by *Puccinia hordei*) shows all characteristics of the so-called horizontal type of resistance. The partial resistance in the field, the latent period, and the infection frequency in young flag leaves were measured in three fairly resistant cultivars with five different isolates. The cultivars and the isolates differed significantly ($P = 0.001$) for all three variables. The cultivar-isolate interaction component was highly significant for latent period; the Julia-isolate 18 combination showed a differential interaction, but other combinations also contributed to the interaction variance. In the field, the partial resistance of the Julia-isolate 18 combination also appeared to interact differentially. The cultivar effect on latent period was shown earlier to be governed by polygenes. This indicates that differential interactions occur in a polygenic system and suggests that polygenic systems in the host can operate on a gene-for-gene basis with polygenic systems in the pathogen.

Additional key words: vertical resistance.

Van der Plank (9, 10) discriminated between vertical (VR) and horizontal (HR) resistance of plants to diseases. The former is characterized by differential interactions between host and pathogen, the latter by the absence of such differential interactions. He stated (12): "Characteristically, HR slows epidemics down. Sporulation is less abundant, or fewer spores infect, or the time taken from infection to sporulation is increased, or these effects occur together. And the effects derive, I believe, from a genetic basis distinct from that of VR".

The partial resistance of barley (*Hordeum vulgare* L.) to leaf rust (*Puccinia hordei* Otth.) is of the HR type. The barley cultivars Vada, Julia, and Berac show a high degree of partial resistance (slow rusting) due to a lower infection frequency, longer latent period, lower sporulation rate, and shorter infectious period (1, 3, 4, 6, 8). This resistance is governed by polygenes (5).

Differential interactions of small magnitude in barley-*P. hordei* were reported for infection frequency (2) and latent period (4). Whether such interactions could be observed in the field was the object of this study.

MATERIALS AND METHODS

The spring barley cultivars Vada, Julia, and Berac and five leaf rust monospore isolates (designated 11-1, 18, 1-2, 22 and 24) were used. The three cultivars and the isolates 1-2, 11-1, and 18 were used in an earlier (4) study. The results, especially on seedlings, indicated that isolate 11-1 was lost and that I was dealing with an unknown isolate. Isolates 22 and 24 were collected in 1974 near Les Settons, Burgundy, France, and near Aalten (50 km east of Wageningen), The Netherlands, respectively. Before the experiments started, the isolates again were single-spored. They were multiplied in plastic cages in different greenhouse compartments to minimize the chance of contamination.

The barley cultivars were sown on plots 2.5 × 3.0 m, isolated from each other by winter rape. Early in the spring of 1976, 6.0 × 6.0-m areas were cleared in a large rape field in the South-West Flevopolder and the soil prepared for barley culture. The barley was sown at a commercial seeding rate of 120 kg/ha. The sowing (end of April) was relatively late to enhance the development of leaf rust. The trial design was a split-plot in two replicates with isolates on main plots and cultivars on subplots. The main plots were separated from each other by 40 m of winter rape; the subplots by 12 m of winter rape. Four control (noninoculated) plots were sown with the very susceptible cultivar Sultan on the corners of the trial area and 50 m from the experimental plots. The epidemic in each plot was initiated by means of infected seedlings of Sultan. These were inoculated in the usual way (3, 7) with the five isolates and transplanted on 20 May into the plots when the lesions were tiny light green specks. Two spreader plants were planted per plot. Seedlings with about equal infection densities were used, except for the isolate 24 group, in which the seedlings had slightly fewer lesions. Twenty days after being planted, the spreader plants were removed.

The disease severity of each plot was assed on 7 and 15 July by collecting, at random, 40 tillers per plot per date. The amount of disease on the upper three leaves of each tiller was evaluated by means of the assessment key (7) described earlier. The scales of the key range from 1 (one uredosorus per 10 tillers) to 19 (leaves dead). A 0 indicates no sori. Each unit increase on the scale corresponds approximately with a twofold increase in amount of disease up to 13 units. Above 13 (10% leaf area affected),

the increase per unit becomes progressively less than a factor of two to correct for the leaf area already affected (9). Scale values of the 40 tillers were averaged to obtain a mean scale value per treatment. This mean scale value was used to obtain the mean percentage leaf area affected through back-transformation.

Plants of the same cultivars were grown in the greenhouse in black 12 × 12-cm square plastic pots and inoculated with the five isolates at heading. Eight well-developed plants per cultivar and per isolate were used. At inoculation, the eight plants of each cultivar were situated at random. Forty mg of uredospores per isolate were sprayed over the 24 plants (4), which then were placed at 100% RH for about 16 hr.

The latent period (LP) (i.e., the period between inoculation and uredosorus formation) was measured on two to four young flag leaves per plant as described (4). Tillers carrying these leaves were selected and marked before rust symptoms appeared. Very young and old flag leaves were not considered. The flag leaves used for measuring the LP also were used for measuring the number of uredosori per unit of leaf area (infection frequency) as described (6).

Analyses of variance and a partial regression and a multiple correlation calculation were used to evaluate the transformed field assessment (scale) values, latent periods and infection frequencies (IF). Analysis of variance for LP was allowed here in contradiction to (4) as the susceptible cultivars with a considerably shorter LP and a smaller variance were excluded.

TABLE 1. Disease severity expressed in transformed scale values and in percentage leaf area affected of three barley cultivars infected by five isolates of *Puccinia hordei* just prior to maturation

Barley cultivar	Disease severity in plants infected by *P. hordei* isolate: 11-1	18	1-2	22	24	Mean[b]
	Scale value[a]					
Berac	12.7	12.4	11.5	12.0	9.7	11.7
Julia	11.9	13.3[c]	10.9	10.1	9.2	11.1
Vada	9.7	9.1	9.2	8.1	6.2	8.5
Mean[d]	11.4	11.6	10.5	10.1	8.4	
	Leaf area affected (%)					
Berac	8.1	6.7	3.1	5.0	0.9	3.8
Julia	4.5	12.1[e]	1.8	1.1	0.6	2.1
Vada	0.8	0.5	0.6	0.22	0.06	0.3

[a]The scale values range from 1 (one uredosorus per 10 tillers) to 19 (leaves dead). Each unit increase on the scale corresponds approximately with a twofold increase in amount of disease. A 0 indicates no sori. The LSD ($P = 0.05$) for scale values is 1.6.
[b]Calculated LSD ($P = 0.05$) = 0.7 (mean of five isolates).
[c]In absence of interaction, 11.4.
[d]Calculated LSD ($P = 0.05$) = 1.0 (mean of three cultivars).
[e]In absence of interaction, 2.9%.

TABLE 2. Latent periods in days for uredosorus formation in young flag leaves of three barley cultivars inoculated with five isolates of *Puccinia hordei*

Cultivar	Latent period in days with *P. hordei* isolate:[a] 11-1	18	1-2	22	24	Mean[b]
Berac	13.6	14.4	12.7	13.9	13.8	13.7
Julia	13.8	13.3[d]	12.8	14.4	13.7	13.6
Vada	15.6	18.3	14.9	16.1	16.4	16.3
Mean[c]	14.3	15.3	13.5	14.8	14.6	

[a]The latent period is the period between inoculation and uredosorus formation. The LSD ($P = 0.05$) = 0.8 days.
[b]Calculated LSD ($P = 0.05$) = 0.35 (mean of five isolates).
[c]Calculated LSD ($P = 0.05$) = 0.50 (mean of three cultivars).
[d]Based on additive effects 14.6 days.

RESULTS

The field plots were well isolated from each other. From sowing until assessment the winter rape stood at least 60 cm above the barley. Three of the four control plots of Sultan showed less than one-tenth pustule per tiller on the first assessment date; the fourth and leeward plot averaged 0.7 pustules per tiller. In contrast, the most resistant cultivar, Vada, averaged nine pustules per tiller on the first assessment date. If the control plots had received primary inoculum at the same time or a little later than the experimental plots, an affected leaf area of 10-20% would be expected by extrapolating from the data of Parlevliet and van Ommeren (7).

Both cultivars and isolates contributed very highly significantly ($P = 0.001$) to the differences in disease severity (Table 1). The data in Table 1 were derived from the sampling; nevertheless, except for a higher level of severity, they parallel those of the first assessment. In the overall analysis of variance the interaction between cultivars and isolates was not significantly different from the error variance. Nevertheless, the Julia-isolate 18 combination showed a differential interaction significant at $P = 0.05$. The Julia-isolate 18 combination also showed a significantly differential interaction at the first sampling date ($P = 0.05$). Cultivar and isolate effects were highly significant for LP and IF, two of the components of partial resistance that determine the epidemic (Tables 2 and 3). The cultivar-isolate interactions for LP were also very highly significant ($P = 0.001$), but still small compared to the main effects. The Julia-isolate 18 combination showed a clear differential interaction that confirmed the earlier observations (4), but which did not explain all interaction variance. With IF, no cultivar-isolate interaction could be discerned. This was expected because the trial error in measuring IF was considerably larger

TABLE 3. Infection frequencies (uredosoi per square centimeter) on the young flag leaves of three barley cultivars inoculated with five isolates of *Puccinia hordei*

Cultivar	Infection frequency with *P. hordei* isolate:[a] 11-1	18	1-2	22	24	Mean[b]
Berac	11.5	3.9	8.9	7.7	3.1	7.0
Julia	15.1	7.7[c]	8.7	11.6	2.9	9.1
Vada	11.7	3.8	5.4	7.5	0.8	5.8
Mean[d]	12.8	5.1	7.7	8.9	2.2	

[a]Calculated LSD ($P = 0.05$) = 4.3.
[b]Calculated LSD ($P = 0.05$) = 1.9 (mean of five isolates).
[c]Based on additive effects 6.4.
[d]Calculated LSD ($P = 0.05$) = 2.5 (mean of three cultivars).

than the one for LP (the coefficients of variation were 21 and 1.9%, respectively). With a CV for LP of 21% no cultivar-isolate interactions could have been discerned either.

Although no cultivar-isolate interactions for IF could be discerned, the IF of the Julia-isolate 18 was higher than expected on a basis of additive effects between cultivar and isolates. Also, the uredosori were visibly larger than on other combinations, suggesting a higher spore production.

Disease severity as measured in the field is expected to depend on the components of partial resistance. The two components studied here, LP and IF, both correlated with the field assessment. The multiple correlation coefficient was 0.72. The partial regression equation was: Field assessment (scale values) = 0.17 IF − 0.67 LP + 18.9.

DISCUSSION

The differential interaction for partial resistance, as observed for the Julia-isolate 18 combination, appears to be caused primarily by its shorter LP and secondly by the possible increase in IF and spore production. An increase in IF with decreasing LP has been shown to exist in the barley - *P. hordei* relationship (6). A shorter LP also tends to increase spore production (Parlevliet, *unpublished*). The cultivar effect on LP is governed by polygenes. Barley cultivar Vada is supposed to possess five to six minor genes for a longer LP (5). Julia also carries several minor genes for a longer LP (Parlevliet, *unpublished*).

The partial resistance in this host-pathogen relationship is typical of the horizontal type as envisaged by van der Plank (11, 12); i.e., a polygenic, slow-rusting type. However, van der Plank stated explicitly that the definition for HR excludes any differential interactions and rules out the possibility of any gene-for-gene relation (11, pages 166-167).

Parlevliet (4) doubted the validity of the host-pathogen interaction test to evaluate the presence of HR, and later with Zadoks (8), investigated this problem more thoroughly. They compared two models in which resistance and pathogenicity were governed by five minor genes each. In model I (addition model) the genes in the host and the genes in the pathogen were acting in an additive way as, in fact, was envisaged by van der Plank (11, 12) by assuming that no gene-for-gene relations occur in HR. In the other model (interaction model) the five genes acted on a gene-for-gene basis with the five genes of the pathogen. In the addition model all variance was due to main effects; in the interaction model, most of the variance was caused by main effects and only a small proportion of the variance was derived from cultivar-isolate interactions. This proportion was so small, that only in experiments with a very small error variance one could hope to discern interaction effects from the error effects. This is exactly what has been found here. With LP, small cultivar-isolate interaction effects were found, not only attributed to the Julia-isolate 18 combination. The overall interaction component in the field assessment data was not significantly different from the error component, although the Julia-isolate 18 combination scored significantly higher than expected on the basis of no interaction effects. With the IF data, no interaction effects could be discerned. The coefficients of variation due to error for LP, field assessments, and IF were 1.9, 7.3, and 21%, respectively. It appears that LP is the most sensitive component of partial resistance, that it is polygenically controlled, and that it shows differential interactions. This suggests that minor genes controlling partial resistance to leaf rust in barley operate on a gene-for-gene basis with minor genes for pathogenicity in the pathogen.

For further studies, the LP may be the most suitable variable in this host-pathogen system since it can be measured more accurately than any of the other contributing variables. It is also the most important component determining the level of partial resistance. The study of a wide range of cultivars resulted in a correlation between field assessments and LP's in the young flag leaves of $r = -0.91$ (7). It was reduced here to $r = -0.65$, which could be expected, since the three cultivars with the longest LP were chosen and this reduced the variation for LP. Infection frequency, sporulation rate, and infectious period also contribute to partial resistance, but apparently to a considerably smaller extent than LP in the barley-leaf rust relationship.

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