Inheritance of Resistance to *Pyrenophora graminea* in Barley

MOHAMMED BOULIF, Professor, Department of Plant Pathology, National School of Agriculture, Meknes, Morocco, and ROY D. WILCOXSON, Professor, Department of Plant Pathology, University of Minnesota, St. Paul, MN 55108

ABSTRACT

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Barley cultivars and lines adapted for cultivation in Morocco and considered to be resistant or susceptible to the stripe disease caused by *Pyrenophora graminea* were crossed and progenies were tested for resistance to three virulent isolates of the pathogen obtained in Morocco. Progenies in the F_1, BC_1, BC_2, F_2, and F_3 generations were tested with isolate 17. In the cross Minnesota 23 × Atlas 68, resistance of Minnesota 23 was conditioned by a single dominant gene. This also was true in the cross Minnesota 23 × Merzaga 077. In the cross Arig 8 × Merzaga 077, resistance was heritable, but its genetic control was not determined because of the intermediate level of resistance in Arig 8. In the cross CI 9737 × Merzaga 077, resistance of CI 9737 was conditioned by two genes, one of which appeared to have a recessive epistatic effect on the other. In the cross CI 9737 × Atlas 68, resistance was controlled by recessive genes, but the number could not be estimated. In the cross Arig 8 × Atlas 68, resistance was also controlled by recessive genes, but again gene numbers could not be estimated. Progenies of Minnesota 23 × Atlas 68 were tested for resistance to *P. graminea* isolates 16 and 25, which were more virulent than isolate 17. With isolate 16, resistance of Minnesota 23 appeared to be due to two recessive genes with cumulative effects. With isolate 25, the resistance of Minnesota 23 probably was governed by several genes.

The stripe disease in barley (*Hordeum vulgare* L.) caused by *Pyrenophora graminea* Ito & Kuribayashi is widespread in Morocco (2,6,20), and resistance appears to be the most practical way to control it. Fungicides are not likely to be useful because the barley crop is grown over a large area from farmer-produced seed and farmers have not been educated to use seed treatments. Sources of resistance to barley stripe are known (10,12–14,19,21,24), and resistance should be incorporated into cultivars that are being grown in Morocco or that will be released in the near future.

To facilitate the development of stripe-resistant barley cultivars, current information is needed on the genetics and inheritance of resistance. The literature indicates that resistance is a heritable trait but genetic control is complex. Isenbeck (11) concluded that resistance was conditioned by several dominant genes. Arny (3) concluded that it was dominant and conditioned by three genetic factors in some crosses and incompletely dominant and conditioned by many factors in other crosses. Suneson (23) reported that resistance was completely dominant in the cultivar Trebi and partially dominant in Male Sterile, whereas susceptibility was dominant in Club Mariout. He suggested that resistance was due to six genes. Recently, Konak (15) studied resistance in F_2 populations of eight crosses between American and Turkish barleys and found that resistance was dominant and conditioned by one or two genes.

The objective of our work was to study the inheritance and genetics of resistance to barley stripe using cultivars that were adapted for use in Morocco and that varied in their reaction to the disease.

MATERIALS AND METHODS

Five barley genotypes were studied because their reactions to stripe had been observed in previous studies (4,6,8) (Table 1). Atlas 68, developed in California and adapted to Morocco, is susceptible to stripe (7). Merzaga 077, a Moroccan barley developed in the 1930s from indigenous barley populations (9), is also susceptible to stripe (6,8). Minnesota 23 and CI 9737, of American and Ethiopian origin, respectively, and obtained from John Caddel, formerly of the Institute of Agronomy and Veterinary Science, Rabat, Morocco, showed resistance to Moroccan isolates of *P. graminea* (8). Arig 8, an Italian selection adapted to Moroccan conditions (5), is moderately resistant to stripe.

The susceptible genotypes, Atlas 68 and Merzaga 077, were crossed to the resistant Minnesota 23 and CI 9737 and to the moderately resistant Arig 8. F_1 plants were grown in the greenhouse in 1982, and backcrosses were made to parental cultivars. F_2 populations produced in the greenhouse were space-planted in an irrigated field in summer 1982. The seed for F_3 populations was harvested from individual F_2 plants.

Testing of the progenies. Progenies of the F_1, F_2, F_3, and backcross generations

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Table 1. Reported percentage of stripe-infected plants and classification of disease reaction of barley genotypes used as parents

Stripe range (%) and reaction[a]	Genotypes				
	Atlas 68	Merzaga 077	Arig 8	CI 9737	Minnesota 23
Range	64–72[b]	40–62	19–33	0	0–5
	9–40[c]	5–20	11–5	0	0
Reaction	S	S	MR	R	R

[a] S = susceptible, MR = moderately resistant, R = resistant.
[b] Artificial inoculation (8).
[c] Natural infection (4,6,8).

(BC_1 and BC_2) were tested along with parental genotypes in 1983 and 1984 at the National School of Agriculture, Meknes, Morocco. The details on evaluation of the populations of each cross are given in Table 2.

Three isolates of *P. graminea* were used to test resistance. The isolates were obtained from Morocco and had been virulent on many barleys in previous tests. They also differentiated the parents. Populations of all crosses were inoculated with isolate 17. Populations of the cross Minnesota 23 × Atlas 68 were also inoculated with isolates 16 and 25.

Inoculum was prepared from stock cultures of the isolates of *P. graminea* that had been stored in infected dried barley leaves. Each stock culture was derived from a single conidium isolate of *P. graminea*. Inoculation was with vigorously growing cultures that were 10–12 days old, using methods of Mohammed and Mahmood (18) and Nilsson (19).

Kernels of parental genotypes and progenies from each cross were dipped into 5% sodium hypochlorite for 5 min, rinsed twice with sterile water, and air-dried at 30 C for 3 hr. The dry kernels (20–50 per petri dish) were evenly distributed in a sandwich of a culture of the pathogen growing on PDA and placed at 4 ± 1 C for 13 days. While exposed to the pathogen, kernels absorbed water and the embryos grew slightly, and infection was initiated if not completed. Kernels were removed from the cultures and immediately space-planted in rows in the field. Plots were irrigated as needed to maintain plant growth, and weeds were removed by hand.

Inoculation and planting were done twice during January and February 1983 and 1984 because of the large volume of material to be tested. At each inoculation and planting, samples were included from the parental populations and from as many of the filial populations as possible. A sample of a population contained at least 50 kernels except when only a few kernels were available. In these instances, all available kernels were tested once or groups of 20–30 kernels were tested twice. The F_1, BC_1, and BC_2 populations were tested in 1983 and the F_2 and F_3 populations, in both 1983 and 1984. Tests of the F_3 populations involved 100 plants per family; the number of families per cross varied from 72 to 147. Details on numbers of samples and plants tested are given in Table 2.

At heading, diseased and healthy plants in a row were counted and the incidence of disease was expressed as a percentage.

Data analysis. Because the data were expressed as percentages of plants diseased, they were transformed to arcsin $x^{1/2}$, where x was the percentage of diseased plants. Comparisons among the parents, the F_1, BC_1, BC_2, and F_2 generations were made on the transformed data (16,22). In the F_3 generation, the frequency distributions of F_3 families were used to help interpret F_2 segregation ratios, based on discontinuity points in the distribution as suggested by McKenzie and Rutgers (17) and Yoshimura et al (25). When the F_3 frequency distributions failed to suggest a segregation ratio, the F_3 families were classified as resistant, susceptible, or segregating on the basis of observed ranges of parental reactions as reported for genetics of wheat flag smut (1). The F_3 families with stripe disease incidence falling within the observed range of the resistant parent were considered to be resistant, whereas those

Table 2. Details on evaluations for resistance in barley populations to three isolates of *Pyrenophora graminea*[a]

Parents Filial generations	No. of samples tested	No. of plants tested	Disease mean (%)	Variance
Tests with isolate 17				
Minnesota 23 (P_1)	10	664	1.8	18.5
Atlas 68 (P_2)	10	653	53.0	290.0
F_1	...	...	...	...
BC_1 ($F_1 \times P_1$)	2	93	21.1	6.0
BC_2 ($F_1 \times P_2$)	1	47	23.0	...
F_2	12	998	9.3	18.4
F_3	118	11,800	...	...
Minnesota 23 (P_1)	8	521	1.1	1.6
Merzaga 077 (P_2)	8	520	50.0	218.0
F_1	2	93	6.5	11.0
BC_1	2	47	6.6	13.0
BC_2	2	43	38.2	280.0
F_2	12	1,036	6.6	16.2
F_3	100	10,000	...	...
Arig 8 (P_1)	4	278	15.0	430.0
Atlas 68 (P_2)	5	374	57.5	185.0
F_1	2	72	86.6	55.0
BC_1 ($F_1 \times BC_1$)	1	41	41.0	...
BC_2 ($F_1 \times BC_2$)	1	45	70.0	...
F_2	8	666	59.6	533.0
F_3	128	12,800	...	...
Arig 8 (P_1)	5	375	9.0	65.5
Merzaga 077 (P_2)	5	392	46.5	175.0
F_1	2	35	51.0	1,161.6
BC_1	1	36	62.0	...
BC_2	1	30	73.0	...
F_2	10	753	26.0	277.0
F_3	72	7,200	...	...
CI 9737 (P_1)	10	760	15.0	122.0
Atlas 68 (P_2)	7	491	69.0	414.0
F_1	2	83	88.5	3.9
BC_1	2	81	63.5	204.4
BC_2	2	89	88.5	19.2
F_2	10	853	36.0	32.0
F_3	99	9,900	...	...
CI 9737 (P_1)	10	717	11.0	49.6
Merzaga 077 (P_2)	10	750	54.0	236.0
F_1	...	...	...	...
BC_1	1	37	23.0	...
BC_2	1	25	72.0	...
F_2	10	808	12.0	44.0
F_3	100	10,000	...	...
Test with isolate 16				
Minnesota 23 (P_1)	9	257	0.3	0.9
Atlas 68 (P_2)	9	308	70.0	390.0
F_1	2	81	45.0	63.0
BC_1	1	63	7.5	...
BC_2	1	66	51.0	...
F_2	20	1,159	22.4	159.0
F_3	147	14,700	...	...
Test with isolate 25				
Minnesota 23 (P_1)	14	909	0.4	2.5
Atlas 68 (P_2)	14	1,062	89.0	175.2
F_1	...	...	...	...
BC_1	3	111	15.0	180.0
BC_2	1	33	24.0	...
F_2	7	610	56.6	81.5
F_3	109	10,900	...	...

[a] Populations consisted of parental samples and filial generations of crosses between resistant and susceptible barley cultivars. P_1 = resistant parent, P_2 = susceptible parent. F_3 = no lines evaluated.

falling within the observed range of the susceptible parent were considered to be susceptible. The F_3 families not showing reactions in either parental range were considered to be segregating. The number of F_3 families in each class was compared with the number expected in those classes. The chi-square test was used to check the goodness of fit of the proposed hypothesis to the observed distributions.

RESULTS

Resistance to isolate 17. *Minnesota 23 × Atlas 68.* Minnesota 23 was resistant (disease incidence ranged from 0 to 13.3%) and Atlas 68 was susceptible (disease incidence ranged from 37 to 100%; Fig. 1). The incidence of stripe in the parents did not overlap. The F_1 generation was not tested because of insufficient seed. The mean of the BC_1 generation (backcross of F_1 to resistant Minnesota 23) was similar to that of the resistant parent, indicating complete dominance of resistance of Minnesota 23 over susceptibility of Atlas 68. The BC_2 generation (backcross of F_1 to Atlas 68) had an intermediate reaction close to the midpoint of the two parents, approximating a 1:1 segregation ratio expected if resistance was due to a single dominant gene. In the F_2 generation, most plants were stripe-free; maximum incidence of stripe was about 20% in one sample. It is important to note that the F_2 distribution consisted of genetically alike samples inoculated under similar conditions. The mean stripe incidence in the F_2 generation was 17.5% that of the mean of Atlas 68, the susceptible parent. This mean incidence, which approaches 25% of the mean incidence of Atlas 68, and the reaction of the BC_1 and BC_2 generations suggest that resistance in Minnesota 23 may be governed by a single dominant gene.

The hypothesis that resistance was controlled by a dominant gene was tested with 118 F_3 families (Fig. 1). The distribution of the families was skewed toward resistance, thereby suggesting that resistance was dominant in Minnesota 23 and susceptibility was recessive in Atlas 68. The distribution of the F_3 families, however, did not indicate clear-cut classes, as would be expected if resistance were due to a single dominant gene; there was a high frequency of resistant F_3 families.

Because the F_3 distribution did not group families into distinct classes, the families were grouped into resistant, segregating, and susceptible classes on the basis of the range of stripe incidence of the parents (Table 3). The resistant, segregating, and susceptible F_3 families identified a 1:2:1 ratio, indicative of the segregation of a single gene.

On the basis of data from the backcross and F_2 populations, we concluded that resistance of Minnesota 23 is probably conditioned by a single dominant gene. This conclusion was

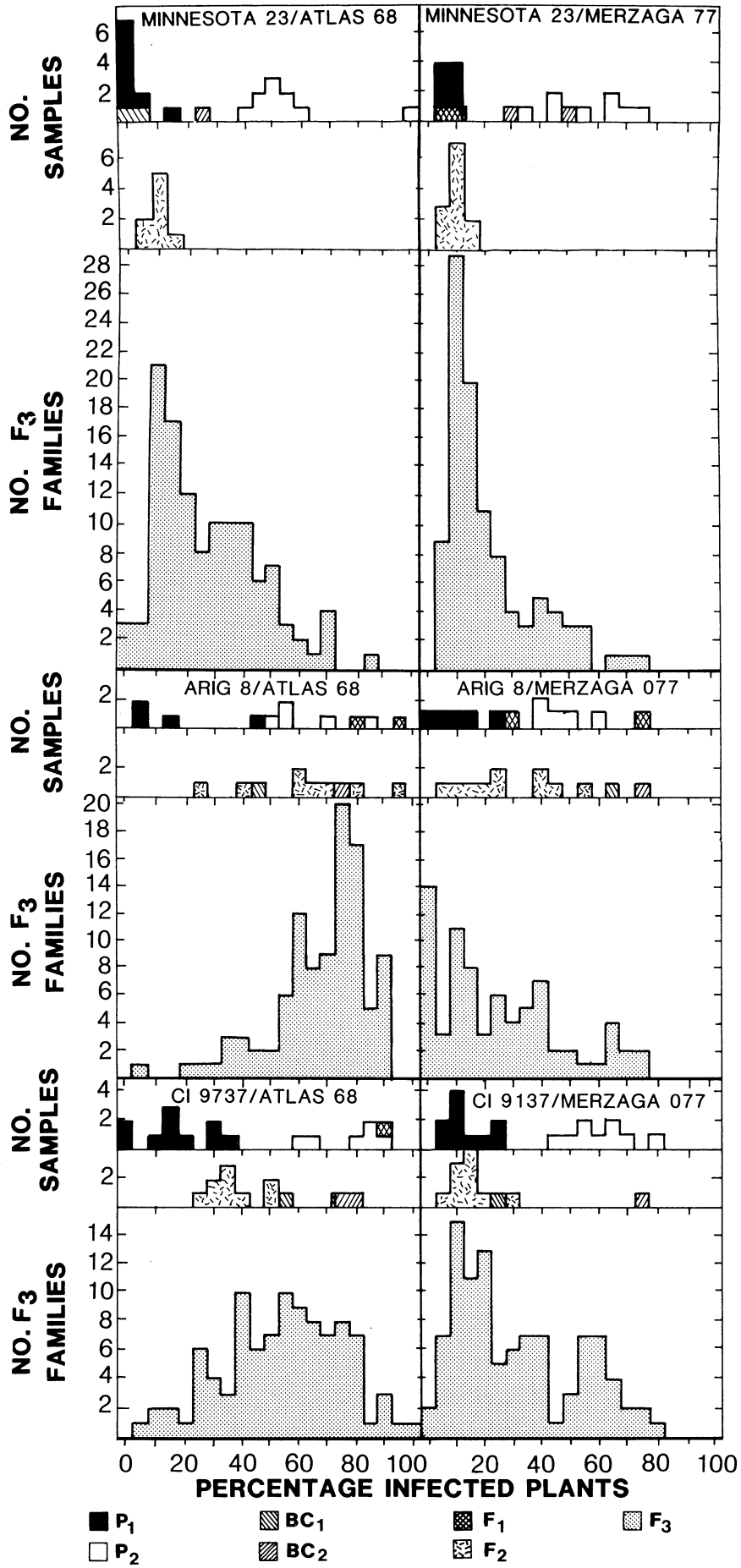

Fig. 1. Frequency distribution of infection classes (percentage infected plants) among seed samples and/or F_3 populations of parental and filial generations of six crosses of resistant and susceptible barley cultivars infected with isolate 17 of *Pyrenophora graminea*. Table 2 gives testing details.

supported by the segregation of the F_3 population into resistant, segregating, and susceptible classes.

Minnesota 23 × Merzaga 077. Minnesota 23 was resistant (disease incidence ranged from 5 to 10%), whereas Merzaga 077 was susceptible (disease incidence ranged from 35 to 75%; Fig. 1). The mean stripe incidence of the F_1 generation was similar to that of the resistant parent ($P = 0.05$), suggesting that resistance was completely dominant. Reactions of the F_2 and BC_1 generations confirmed that resistance was dominant and probably due to a single gene. The BC_2 generation showed a stripe incidence between that of the midpoint of the two parents and the mean incidence of the susceptible parent, which did not fully agree with the hypothesis that resistance was conditioned by a single dominant gene. The observed disease incidence, higher than expected in the BC_2 population, may have been due to the fact that most kernels of this generation were naked. Data from the F_2 generation suggested the dominance of resistance in Minnesota 23, since most plants in this generation were free from disease.

The hypothesis that resistance was controlled by a single dominant gene was tested with 100 F_3 families (Fig. 1). The distribution of the families was skewed toward resistance, thereby confirming the dominance of resistance in Minnesota 23. The F_3 families were grouped into resistant, segregating, and susceptible classes (Table 3), as with the cross Minnesota 23 × Atlas 68. The observed frequencies fit the 1:2:1 ratio, supporting the hypothesis of a single dominant gene controlling resistance in Minnesota 23.

The frequency of resistant F_3 families in the cross Minnesota 23 × Merzaga 077 was higher than in the cross Minnesota 23 × Atlas 68. This may have been because Merzaga 077 carries genetic factors that enhance the effect of the resistance gene in Minnesota 23. It is also possible that Atlas 68 carries factors that modify the action of the resistance gene from Minnesota 23.

Arig 8 × Merzaga 077. Arig 8 was moderately resistant (disease incidence ranged from 0 to 25%), whereas Merzaga 077 was susceptible (disease incidence ranged from 40 to 60%; Fig. 1). The F_1 generation showed a wide range of stripe disease incidence, but the mean of this generation suggested that resistance of Arig 8 was recessive.

Different samples of the F_2 generation showed an array of reactions from resistant to susceptible under the same experimental conditions, making it difficult to draw conclusions about gene action. The BC_1 and BC_2 generations both showed susceptible reactions, which also makes it difficult to draw conclusions about gene action.

The frequency distribution of infection classes among 72 F_3 families (Fig. 1) was skewed toward resistance, indicating a degree of dominance of resistance.

The classification of data of the F_3 families by the method described previously is presented in Table 3. The groupings of the F_3 families into infection classes approximated a 9:3:4 ratio, indicating that resistance in Arig 8 may be controlled by two dominant genes and that susceptibility in Merzaga 077 was governed by two recessive genes, with one showing epistasis. This conclusion contradicts that drawn on the basis of F_1, BC_1, and F_2 data, however. Thus it is difficult to explain the genetic control of resistance in this cross.

Arig 8 × Atlas 68. Arig 8 was moderately resistant (disease incidence ranged from 5 to 45%), whereas Atlas 68 was susceptible (incidence ranged from 45 to 80%; Fig. 1). While the mean values for the parents suggest they are distinct, the ranges indicate they overlap. Arig 8 was therefore considered to be only moderately resistant. The F_1 generation had a mean percentage infection of 86.8%, higher ($P = 0.01$) than that of the susceptible parent, Atlas 68, which may indicate that susceptibility of Atlas 68 was dominant over the resistance of Arig 8. The higher stripe incidence observed in the F_1 generation resulted from a higher exposure of the F_1 seed to inoculum because many of the kernels were naked or partially naked. Stripe incidence in the BC_1 generation was 36.25%, close to the midpoint of the two parents, suggesting a 1:1 segregation ratio, which would be expected if the resistance of Arig 8 were governed by one recessive gene. The reaction of BC_2 confirms that resistance in Arig 8 was recessive.

The F_2 generation samples showed a broad range in stripe infection that made it impossible to draw conclusions on the basis of F_2 data. The disease incidence mean of this generation, however, suggested that resistance of Arig 8 to isolate 17 of *P. graminea* may be recessive. The frequency distribution of infection classes among 100 F_3 families (Fig. 1) was skewed toward susceptibility, thereby confirming that resistance in Arig 8 to isolate 17 was recessive.

The distribution of the F_3 families into resistant, segregating, and susceptible classes did not identify a segregating class (Table 3). A number of families more susceptible than the susceptible parent and a low number of moderately resistant families were identified. The low number of resistant families suggested that resistance is a recessive trait. It was not possible to estimate the number of genes controlling resistance in this cross.

CI 9737 × Merzaga 077. CI 9737 was moderately resistant (disease incidence ranged from 5 to 25%) and Merzaga 077 was susceptible (disease incidence ranged from 45 to 80%; Fig. 1).

The F_1 generation was not tested because of insufficient seed. The mean stripe infection in the F_2 generation indicated dominance of resistance in CI 9737. If resistance was controlled by a

Table 3. Number of resistant, segregating, and susceptible F_3 families of barley crosses infected with different isolates of *Pyrenophora graminae*[a]

Number of F_3 families and disease incidence range per reaction class									
Resistant		Segregating		Susceptible					
No.	Range (%)	No.	Range (%)	No.	Range (%)	Hypothesis	χ^2	*P* value	Total families
Minnesota 23 × Atlas 68					**Tests with isolate 17**				
38	0.0–2.4	53	2.5–38.5	27	38.6–70.0	1:2:1	3.3	0.2–0.1	118
Minnesota 23 × Merzaga 077									
25	0.0–2.8	58	2.9–28.8	17	28.9–70.0	1:2:1	3.8	0.2–0.1	100
Arig 8 × Atlas 68									
9	1.2–41.5	0	41.6–41.9	119	42.0–100	...	...	...	128
Arig 8 × Merzaga 077									
39	0.0–20.5	17	20.6–36.7	16	26.8–65.0	9:3:4	7.27	0.5–0.3	72
CI 9737 × Atlas 68									
13	0.0–27.2	4	27.3–31.2	82	31.3–87.4	1:3	2.82	0.1–0.05	99
CI 9737 × Merzaga 077									
49	3.3–20.6	25	20.7–42.9	26	43.0–75.8	9:3:4	3.05	0.2–0.1	100
Minnesota 23 × Atlas 68					**Test with isolate 16**				
4	0.0–2.7	60	2.8–37.1	83	37.2–94.7	1:63	0.67	0.5–0.25	147
Minnesota 23 × Atlas 68					**Test with isolate 25**				
3	0.0–5.9	67	6.0–49.9	39	50.0–100	1:63	0.67	0.5–0.25	109

[a] Classification of families was based on observed ranges of stripe disease incidence of parents.

single dominant gene, the BC_1 population should show a reaction similar to that of the resistant parent and the BC_2 population should show a stripe incidence close to the midpoint of the two parents. The reaction for the BC_1 generation lies within the range of reactions of the resistant parent. The BC_2 generation did not agree with the hypothesis that a single dominant gene controls resistance in CI 9737. The lack of fit may be because a relatively small number of plants were observed in this generation or because resistance was not controlled by a single dominant gene.

The hypothesis that resistance was controlled by a single dominant gene was tested with 100 F_3 families (Fig. 1). The frequency distribution was skewed toward resistance, which supported the conclusion that resistance in CI 9737 is dominant. Three distinct modes were seen in the distribution: the first between 0 and 25%, the second between 25 and 45%, and the third between 45 and 80%. The large size of the first mode did not favor the 1:2:1 ratio expected if resistance of CI 9737 were governed by a single dominant gene. The test for the goodness of fit of the 9:3:4 ratio to the observed distribution gave a high *P* value (0.70–0.80), confirming that two genes govern stripe reaction in this cross. One gene probably shows a recessive epistatic effect on the other.

The classification of the F_3 families on the basis of observed ranges of parental reactions (Table 3) supported the 9:3:4 ratio expected if resistance to isolate 17 were controlled by two genes, one of which had a recessive epistatic effect on the other.

CI 9737 × Atlas 68. CI 9737 was moderately resistant (disease incidence ranged from 0 to 37%), whereas Atlas 68 was susceptible (disease incidence ranged from 30 to 90%; Fig. 1). The ranges of the parents overlapped somewhat.

The F_1 generation had a mean incidence of infection of 88.5%, higher than the mean of the susceptible parent, Atlas 68. However, the difference between the means of the F_1 and Atlas 68 was not significant, indicating recessiveness of resistance in CI 9737. The mean infection in the BC_2 also supported the hypothesis that resistance in CI 9737 to isolate 17 of *P. graminea* was recessive. The BC_1 generation had a mean infection closer to the mean of the susceptible parent than the midpoint of the parents, suggesting that resistance in CI 9737 may be governed by more than one recessive gene. The mean stripe disease incidence observed in the F_2 generation, however, was close to the midpoint of the parents, which does not fully support recessiveness of resistance in CI 9737.

The hypothesis that resistance was controlled by recessive genes was tested using the frequency distribution of infection classes among 99 F_3 families (Fig. 1). The distribution appeared to be continuous, so the families were grouped into resistant, segregating, and susceptible classes (Table 3) as with the cross Minnesota 23 × Atlas 68. As can be seen, the groupings of the F_3 families did not fit a ratio indicative of one or two genes.

We concluded that resistance in this cross was controlled by recessive genes. However, the number of genes involved could not be precisely estimated, although there probably were not more than two.

Resistance to isolates 16 and 25. Minnesota 23 × Atlas 68 was the only cross tested with these isolates. The frequency distribution of infection classes among seed lots of parental and filial generations inoculated with isolate 16 of *P. graminea* is shown in Figure 2. Minnesota 23 was resistant (disease incidence ranged from 0 to 5%), whereas Atlas 68 was susceptible (disease incidence ranged from 40 to 95%). The reaction of the F_1 generation was within the range of the susceptible parent Atlas 68, indicating that resistance in Minnesota 23 to isolate 16 is recessive. This result was also supported by the reaction of the BC_2 generation. This contrasts with data obtained with isolate 17 that indicated resistance in Minnesota 23 was dominant. If a single recessive gene conditioned resistance in Minnesota 23 to isolate 16, the reaction of the BC_1 would have been close to the midpoint of the parents, but it was similar to that of the resistant parent. The reaction of the F_2 was close to the midpoint of the parents, which suggests the action of more than one gene controlling stripe reaction to isolate 16.

The frequency distribution of infection classes among 147 F_3 families did not indicate that resistance was recessive (Fig. 2).

The classification of the F_3 families on the basis of the range of parental reactions (Table 3) indicated recessiveness of resistance in Minnesota 23 to isolate 16. The low frequency of families classified as resistant suggested that resistance of Minnesota 23 is probably governed by two genes with cumulative effects.

Progenies of the cross Minnesota 23 × Atlas 68 also were tested with isolate 25 of *P. graminea* (Fig. 2). Minnesota 23 was resistant (disease incidence ranged from 0 to 10%), whereas Atlas 69 was susceptible (disease incidence ranged from 45 to 100%). The F_1 generation was not tested because of insufficient seed. The BC_1 and BC_2 generations gave comparable stripe incidences, making it difficult to draw conclusions concerning the type of interaction between resistance factors in Minnesota 23 and susceptibility factors in Atlas 68. The F_2 generation, however, gave a reaction close to the midpoint of the parents, and the distribution of the F_3 families appeared to be continuous or semicontinuous. The

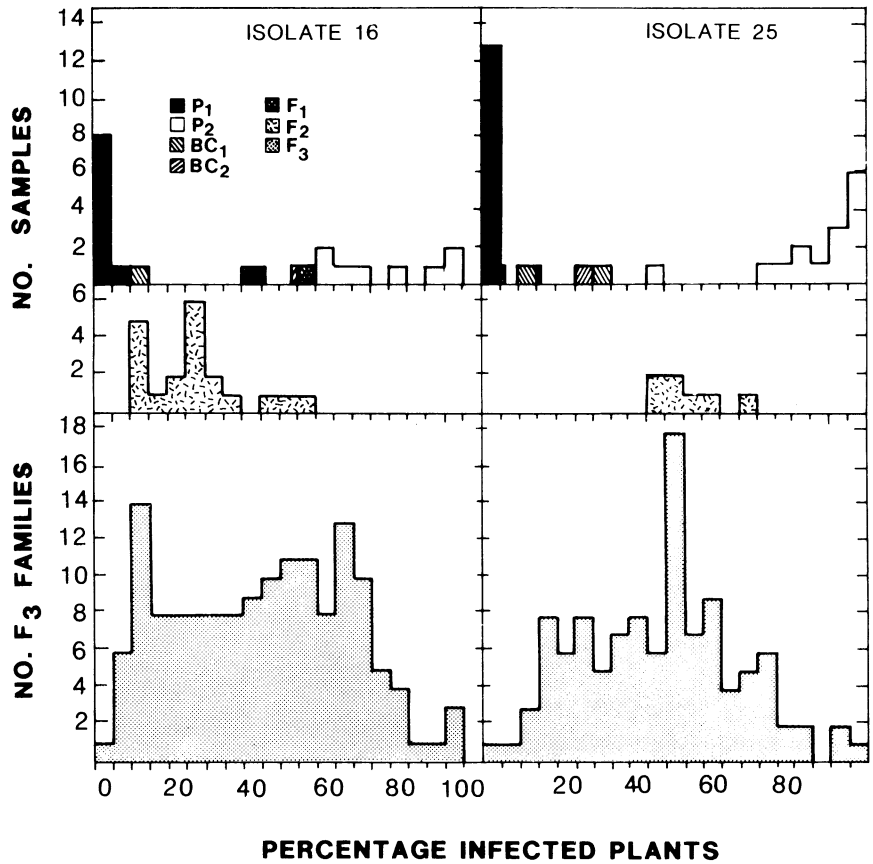

Fig. 2. Frequency distribution of infection classes (percentage infected plants) among seed samples and/or F_3 populations of parental and filial generations of barley cross Minnesota 23 × Atlas 68 infected with isolates 16 and 25 of *Pyrenophora graminea*. Table 2 gives testing details.

grouping of the F_3 families into resistant, segregating, and susceptible classes on the basis of parental reactions indicated that resistance in Minnesota 23 to isolate 25 may be governed by several genes (Table 3).

DISCUSSION

In this study, parental, F_1, F_2, BC_1, BC_2, and F_3 generations were tested under similar experimental conditions. The data obtained showed great variability, making it difficult to draw firm conclusions about the genetic control of resistance to *P. graminea* in certain barley crosses. These difficulties may be related to:

1. The reliability of the inoculation method. The method has been shown to be efficient in identifying highly resistant and highly susceptible genotypes but is less reliable with intermediate genotypes.

2. Possible variation among cultures used for inoculation. Although cultures used for inoculum traced back to a single conidium, variation may have occurred during subculturing.

3. Possible heterogeneity within parental populations. Populations were initiated from six to 10 single heads selected on the basis of phenotypic characteristics of the parents. The selected heads were bulked for each parent and increased.

4. Variability of stripe reaction. This may have been due to escape or to a small number of plants observed in certain samples.

5. The method of estimating the reaction classes of F_3 families. Because of the continuous gradation in the F_3, the method of categorizing F_3 families into resistant, segregating, and susceptible classes may pose a problem.

Backcross generations were used in this study to produce additional information to supplement data obtained from other generations. Data obtained from backcross generations were inconclusive in certain crosses, however. This difficulty may be removed by selfing the BC_1 and BC_2 plants and testing the BCF_2 families.

The high level of resistance in Minnesota 23 suggests that resistance is due to major gene(s). This may be the reason the data obtained from the testing of the crosses involving Minnesota 23 were more consistent than those obtained from other crosses.

Tests with isolates 16, 17, and 25 showed that resistance to *P. graminea* may be monogenic or oligogenic. Because of the small number of genes involved, resistance may be readily incorporated into breeding lines to minimize yield losses from barley stripe.

Results obtained also showed that resistance genes were influenced by the background into which they were incorporated. Atlas 68 tended to produce more susceptible progenies than Merzaga 077 when these genotypes were crossed to the different resistant parents.

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