Inheritance of Resistance to Kernel Discoloration of Barley

MONTE R. MILES, Former Research Assistant, and ROY D. WILCOXSON, Professor, Department of Plant Pathology, and DONALD C. RASMUSSON, Professor, Department of Agronomy and Plant Genetics, University of Minnesota, St. Paul 55108

ABSTRACT

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Progeny derived from six crosses of barleys resistant and susceptible to kernel discoloration were evaluated for resistance in the F_2 and F_3 generations. Plants were grown in the field under irrigation and inoculated with *Bipolaris sorokiniana*. Heritability estimates of 27–43% were obtained on an individual F_2 plant basis by parent-progeny correlation. Heritability estimates of 48–76% were obtained on a family basis from variance components from replicated F_3 nurseries. Distributions of the F_2 and F_3 populations were nearly continuous and tended to follow a bell shape. Data from both generations failed to confirm a fit to simple hypothesized segregation ratios. Transgressive segregation toward susceptibility was observed in F_3 populations of all crosses.

Kernel discoloration is an important disease of malting barley (*Hordeum vulgare* L. emend. Bowden) that reduces quality and market value in the upper midwestern United States. In addition, diseased kernels may contain fungal toxins harmful to livestock (19), and they may be involved in seedling blight and root rot diseases (5–7,12,14).

Kernel discoloration is a black to dark brown discoloration of the palea and lemma (16,19), associated primarily with *Bipolaris sorokiniana* (Sacc. ex Sorok.) Shoem., *Alternaria alternata* (Fr.) Keissler, and *Fusarium graminearum* Schwabe (1,2,4,9,10,13–15). Several sources of resistance have been reported (1,2,6,11), and the resistance possessed by Chevron and CI 9539 has been studied recently (2,3,18,22). Resistance to kernel discoloration in barley has been shown to be genetically controlled (1,3,6,11,22), and heritabilities of 20–50% in F_2 and F_3 generations have been reported (22). F_2 progeny appeared to segregate in a normal distribution, indicating quantitative gene action (22).

The objectives of this research were to obtain estimates of heritability for resistance to kernel discoloration in progenies of several crosses and to provide information and sources of germ plasm for breeding programs developing cultivars resistant to kernel discoloration.

MATERIALS AND METHODS

Six crosses were made between barleys that are resistant to kernel discoloration and those that are susceptible (Table 1). The resistant parents were derived from either Chevron or CI 9539. F_2 populations and parents were planted in the field in 1981 or 1982. Seeds were spaced 10 cm apart in rows 1 m long. A single spike was harvested from each F_2 plant for disease evaluation and to provide seed for the F_3 generation.

F_3 populations from crosses 38 and 39 (40–50 lines per cross) and parents were planted in the field in 1982, in 0.6-m rows at a rate of 20–24 seeds per row, in three replicated blocks. F_3 populations from crosses 49, 50, 56, and 57 (59–83 lines per cross) and parents were planted in 1984 in a similar manner. A single spike was harvested from each F_3 plant in each row. The severity of discoloration was evaluated on kernels from single spikes within each family in all six crosses (i.e., on a single-spike basis). Kernels were also evaluated for discoloration from bulk samples in crosses 49, 50, 56, and 57.

Plant populations were grown in the field at St. Paul, Minnesota. Inoculation with *B. sorokiniana* began when approximately 50% of the spikes had emerged from the boot and continued on alternate evenings for 3 wk (16). Conidia of *B. sorokiniana* were produced on a substrate of perlite-cornmeal-potato-dextrose agar (Difco) (17). A mixture of 40–50 single-spore cultures, obtained from throughout barley-growing regions in Minnesota, was used for producing the inoculum. Conidial suspensions ($5–10 \times 10^4$ conidia per milliliter) were applied at 2.8 kg/cm^2 (about 40 psi) from a 60-L tank mounted on a garden tractor. Tween 20 (25 ml per 60 L of inoculum) was used as a wetting agent. Approximately 0.15 L of inoculum was applied per row. Plots were watered with overhead sprinklers for 1 hr the morning following application of the inoculum and, when inoculation was discontinued, for 1 hr every evening until harvest.

The cultivars Chevron, CI 9539, Karl, Morex, and Robust were included in the nurseries at St. Paul at regular intervals to evaluate variability in the development of kernel discoloration and to provide controls with different degrees of resistance. In addition, these barleys and the parents of each cross (KD 72, KD 390, KD 58, KD 627, and M78-91) were evaluated in replicated nurseries in seven environments during 1982 and 1983. These environments consisted of natural conditions at Crookston, Morris, Rosemount, and St. Paul, Minnesota; irrigated plots at Rosemount and St. Paul; and plots that were irrigated and inoculated with *B. sorokiniana* at St. Paul.

Kernel discoloration was evaluated as a dark brown to black discoloration of the palea and lemma (16). Disease

Table 1. Crosses and pedigrees of resistant parents used in the study of inheritance of resistance to kernel discoloration in barley caused by *Bipolaris sorokiniana*

Cross	Parents (R/S)[x]	Pedigree of resistant parent
38	KD 627/Robust	Manker/CI 9539//Cree/3/Morex/4/M34[y]
39	KD 390/Morex	Chevron/M14//M18/3/Morex/4/Manker[z]
49	KD 72/Morex	Chevron/M14//M18/3/Manker/4/M34
50	KD 72/Robust	Chevron/M14//M18/3/Manker/4/M34
56	KD 58/M78-91	Chevron/M14//M18/3/Morex/4/Manker
57	KD 58/Robust	Chevron/M14//M18/3/Morex/4/Manker

[x] R = resistant or moderately resistant, with scores of 1–2. S = moderately susceptible or susceptible, with scores of 2–4, predominately 3 or 4.
[y] CI 9539 is the source of resistance to kernel discoloration.
[z] Chevron is the source of resistance to kernel discoloration.

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Table 2. Expected mean squares and components of variance from F_3 populations

Source of variation	Expected mean square[z]	Mean square
Replication	$\sigma^2_{e'} + s\sigma^2_E + sn\sigma^2_R$	
Among controls	$\sigma^2_{e'} + s\sigma^2_E + sn\sigma^2_R + rs\theta^2_T$	
Replication × controls	$\sigma^2_{e'} + s\sigma^2_E$	
Within controls (error 1)	$\sigma^2_{e'}$	M_4
Replication	$\sigma^2_e + \sigma^2_{wg} + s\sigma^2_E + sn\sigma^2_R$	
Among F_3 lines	$\sigma^2_e + \sigma^2_{wg} + s\sigma^2_E + rs\sigma^2_G$	M_3
Replication × lines	$\sigma^2_e + \sigma^2_{wg} + s\sigma^2_E$	M_2
Within lines	$\sigma^2_e + \sigma^2_{wg}$	M_1
Error 2	σ^2_e	

[z] r = Number of replications.
s = Number of samples per replication.
n = Number of lines.
σ^2_R = Variance attributed to replication.
θ^2_T = Variance among controls.
σ^2_E = Variance attributed to replication × genotype interaction.
$\sigma^2_{e'}$ = Environment variance = M_4.
σ^2_{wg} = Within-F_3-line variance
$= (\sigma^2_e + \sigma^2_{wg}) - (\sigma^2_{e'})$
$= (M_1 - M_4)$.
σ^2_G = Genotypic variance, F_3 lines
$= (\sigma^2_e + \sigma^2_{wg} + s\sigma^2_E + rs\sigma^2_G) - (\sigma^2_e + \sigma^2_{wg} + s\sigma^2_E)/rs$
$= M_3 - M_2/rs$.
H_B = Heritability on the plot basis
$= (M_3 - M_2)/M_3$.

Table 3. Mean scores for kernel discoloration of five barley cultivars included as checks in irrigated nurseries inoculated with *Bipolaris sorokiniana* at St. Paul, Minnesota

	Year[w]				Cultivar
Cultivar	1981	1982	1983	1984	mean[x]
Chevron	1.3	1.5	1.7	1.5	1.5 a
CI 9539	1.3	1.9	2.0	1.7	1.7 a
Robust	3.2	2.5	3.2	2.8	2.9 b
Morex	3.0	3.3	3.4	3.0	3.2 c
Karl[y]	—	4.3	4.7	3.7	4.2 d
Year mean[x]	2.6[z] b	2.7 b	3.0 c	2.5 b	

[w] Mean of 10 rows; spikes within each row were bulked for evaluation.
[x] Cultivar means or year means followed by the same letter are not significantly different; LSD = 0.2 (p = 0.05). The cultivar × year interaction is not significant. The coefficient of variation is 18.5.
[y] Karl was not included in 1981.
[z] Weighted to adjust for missing Karl data.

Table 4. Mean scores for kernel discoloration of 10 barleys grown in seven environments during 1982 and 1983

	Year[w,x]		Cultivar
Cultivar	1982	1983	mean[y]
Chevron	1.3 a	1.6 a	1.5
CI 9539	1.8 b	1.7 a	1.8
KD 72	2.0 c	2.4 b	2.2
KD 390	2.2 d	2.5 bc	2.4
KD 58	2.3 d	2.6 c	2.5
KD 627	2.3 d	2.6 c	2.5
Robust	2.3 d	2.9 d	2.6
Morex	3.1 e	2.8 d	2.9
M78-91	3.4 f	3.9 e	3.7
Karl	3.9 g	4.4 f	4.1
Year mean[z]	2.5	2.7	

[w] Mean from seven environments, with three replications per environment. The environments were untreated nurseries at Crookston, Morris, Rosemount, and St. Paul, Minnesota; irrigated nurseries at Rosemount and St. Paul; and an irrigated nursery inoculated with *Bipolaris sorokiniana* at St. Paul.
[x] The cultivar × year interaction is significant; LSD = 0.12 (p = 0.05). Cultivars were compared within years by Duncan's new multiple range test; means within the same column followed by the same letter are not significantly different.
[y] Differences between cultivars are significant (p = 0.05).
[z] The year means are not significantly different.

scoring was done after harvested spikes had been air-dried at 25–30 C for 2–4 wk and threshed. The severity of discoloration was estimated from a single spike or from a sample of kernels from a bulk of 10–20 spikes. The kernels were examined on white paper plates 15 cm in diameter under fluorescent lights. Discoloration severity in each sample was compared to that in control cultivars with a range of reactions. Severity was scored 1–5 as follows: 1 = less than 5% of the surface of the kernels in the sample discolored, with discoloration limited to the base of a few kernels; 2 = 6–15% of the surface discolored, primarily on the kernel base; 3 = 16–30% of the surface discolored, with discoloration not limited to the kernel base; 4 = 31–50% of the surface discolored, and almost all kernels with some discoloration; 5 = more than 50% of the surface stained black, and all kernels stained.

The distributions of mean scores for kernel discoloration of single spikes from F_3 populations were plotted, with class limits established by one-fourth of the mean standard deviation (8). Heritability in the F_2 generation, on a plant basis, was estimated by the correlation of F_2 plant scores with F_3 family means (on the single-spike basis). The correlation procedure was used because the ranges for F_3 families within each population tended to be less than those of samples of the F_2 plants.

Analysis of variance was done on the F_3 single-spike and bulk data from replicated nurseries. Heritability estimates on an F_3 family basis were obtained on both the F_3 bulk and the F_3 single-spike bases, by the variance component method (Table 2).

RESULTS

Parent and control cultivars. The severity of kernel discoloration each year of the study (1981–1984) was monitored with five cultivars that represent the range of disease reactions. Discoloration was more severe in 1983 than in the other years (Table 3). It was significantly less severe each year in Chevron and CI 9539 than in Robust, Morex, and Karl. These last three cultivars also differed from one another in discoloration. The year × cultivar interaction was not significant.

The kernel discoloration reactions of parent and control cultivars (Table 4) were evaluated in seven environments during 1982 and 1983 to provide a critical evaluation of the resistant parents. Cultivar and cultivar × year effects were significant (Table 4). CI 9539 and Morex were less discolored in 1983 than in 1982, unlike the remaining cultivars, which were more severely discolored in 1983. Although the cultivar × year effects were significant, the resistant parents ranked the same both years; KD 72 was the most resistant, followed by KD 390, KD 58, and KD 627. In both years, KD 58 and

KD 627 had significantly more severe discoloration than KD 72. Among the susceptible parents, Robust differed significantly from Morex in 1982 but not in 1983; M78-91 was the most susceptible parent both years.

Analysis of progeny distributions: F_2 plant data. The kernel discoloration scores of individual plants within the F_2 populations ranged from 1 to 5, with intermediate scores occurring most frequently. However, in crosses 38 and 39, there were no F_2 plants with scores of 1, and in cross 50 none were scored 5 (Table 5).

Analysis of progeny distributions: F_3 family data. F_3 families and parents were evaluated for kernel discoloration on both the single-spike and the bulk bases. Since the distributions obtained by both methods were similar, only single-spike data are presented (Table 6). The mean scores for kernel discoloration of resistant and susceptible parents in five of the six crosses were significantly different. In cross 38 the mean scores of the resistant parent, KD 627, and the susceptible parent, Robust, did not differ significantly.

The F_3 population of each cross formed a nearly continuous distribution (Table 6). Like the F_2 populations, the F_3 populations tended to form bell-shaped distributions, with families having intermediate kernel discoloration means being the most frequent. In all crosses one or more families were significantly more susceptible than the parent. All crosses produced families that were as resistant as the resistant parents. Crosses 50 and 57 produced families that were numerically more resistant than the resistant parents, but the difference was not significant.

Transgressive segregation toward susceptibility occurred in each cross (Table 6). This was most apparent in cross 38, in which 70% of the population was more susceptible than the susceptible parent. Increased susceptibility was also observed when F_3 population means were compared to midparent values (Table 6). In all six crosses the population mean was higher than the midparent value, indicating greater susceptibility than expected from the parental performance.

Segregation patterns. The frequency distributions of F_2 and F_3 populations were tested for fit to hypothesized genetic ratios (Table 7). Since the distributions were nearly continuous, an arbitrary division based on the kernel discoloration score of the resistant parent was used to decide on the number of lines within the resistant class. The remainder of the population was considered to be susceptible. This attempt to classify resistance was made difficult by overlapping parental ranges and the occurrence of lines more resistant than the parent in some crosses. Although some of the distributions did fit simple ratios, these ratios were not confirmed by data from both the F_2 and F_3 generations or when different methods of evaluation were used for F_3 populations. The continuous distribution and absence of confirmed ratios indicate that resistance to kernel discoloration in barley likely is not conditioned by the action of one or two genes.

Heritability. The individual plant heritability estimates obtained by F_2-F_3 parent-progeny correlation were lower than those obtained for F_3 families by the variance component method. Estimates of heritability on the individual F_2 plant basis ranged from 27% in cross 39 to 43% in cross 57 (Table 8). Heritability estimates on the F_3 family basis ranged from 48% in cross 50 to 76% in cross 56. Similar heritability estimates were obtained when F_3 families were evaluated on the single-spike and bulk bases.

The F_3 variance component method provided similar values for single-spike means and row bulks. The F_3 family estimates were obtained from one generation in a single year, whereas the F_2 parent-progeny estimates were from two generations planted in different years. Furthermore, the parent score is from a single evaluation for each F_2 plant, whereas the F_3 score is the mean of 30 single-spike evaluations or three row bulks.

Correlation of scoring methods. Kernel discoloration scores from individual F_2 plants were compared with mean scores of F_3 families obtained on both the bulk and the single-spike bases from crosses 49, 50, 56, and 57 (Table 9). Correlations were 40–50% when F_3 family means, on both the bulk and the single-spike bases, were compared with the F_2 plant score. Correlations were 80–85% when F_3 family bulk means were compared with family means on the single-spike basis. From these results, the two methods were considered to be similar, and hence either could be used for evaluating resistance.

DISCUSSION

Previous work (22), as well as the present study, suggested that resistance to kernel discoloration of barley is a quantitatively inherited trait. Both studies had bell-shaped F_2 distributions. The F_3 distributions of the present study also indicate that resistance to kernel discoloration is quantitatively inherited. The distributions were nearly continuous and tended to be bell-shaped. The quantitative nature of the trait is further supported by the failure of hypothesized one- and two-gene segregation ratios to consistently explain results when the F_2 and F_3 generations were compared along with methods for evaluating the resistance.

Transgressive segregation toward susceptibility was observed in F_2 plants as well as in F_3 families. This observation was based on the reaction of the susceptible parent. In addition, transgressive segregation is also indicated by the greater susceptibility of F_3 families than was expected from the midparent value. The increased susceptibility may

Table 5. Distributions of kernel discoloration scores of parents and F_2 plants of six barley crosses grown in an irrigated field at St. Paul, Minnesota, inoculated with *Bipolaris sorokiniana*

Cross and parents	No. of F_2 plants and parents evaluated	No. of F_2 plants and parent samples					Mean score
		5	4	3	2	1	
Cross 38	90	5	30	51	4	0	3.4
KD 627[y]	2				2		2.0
Robust[z]	2		2				4.0
Cross 39	119	6	21	74	18	0	3.1
KD 390[y]	2				1	1	1.5
Morex[z]	2		2				4.0
Cross 49	98	3	13	43	29	10	2.7
KD 72[y]	3				3		2.0
Morex[z]	3		1	2			3.3
Cross 50	59	0	6	23	25	5	2.5
KD 72[y]	3				3		2.0
Robust[z]	3			1	2		2.3
Cross 56	98	9	26	47	14	2	3.3
KD 58[y]	3			2	1		2.7
M78-91[z]	3	1	2				4.3
Cross 57	99	1	13	55	28	2	2.8
KD 58[y]	3			2	1		2.7
Robust[z]	3			3			3.0

[y] Resistant parent.
[z] Susceptible parent.

be explained by the breakup of epistatic gene combinations that condition resistance or by the inheritance of resistance in a recessive manner, conditioned by many loci, with the parents contributing different resistance alleles.

A quantitatively inherited trait that exhibited transgressive segregation and a skew toward susceptibility has been reported in studies of partial resistance of barley to *Puccinia hordei* (20,21). An evaluation of F_2 populations for latent

Table 6. Distribution of mean scores for kernel discoloration of F_3 families and parents of six barley crosses grown in an irrigated field at St. Paul, Minnesota, inoculated with *Bipolaris sorokiniana*

Cross and parents	No. of F_3 families and parents evaluated	No. of F_3 families and parents per kernel discoloration score[w]																					Mean score	LSD[x]	Mid-parent
		4.0	3.9	3.8	3.7	3.6	3.5	3.4	3.3	3.2	3.1	3.0	2.9	2.8	2.7	2.6	2.5	2.4	2.3	2.2	2.1	2.0	1.9	1.8			
Cross 38	44								2	1	4		2	9	4	6	6	3	3	4					2.67	0.38	2.28
KD 627[y]	3																			2	1				2.17		2.28
Robust[z]	3																	3							2.40		
Cross 39	46					2	2	3	3	4	6	4	5	6	4	3	4								3.01	0.34	
KD 390[y]	3															1	1		1						2.47		2.73
Morex[z]	3											3													3.00		
Cross 49	83					2				3	2	6	4	8	8	14	12	3	10	8	2	1			2.61	0.44	
KD 72[y]	6																			2		2	2		2.03		2.43
Morex[z]	9										1		2	1	5										2.80		
Cross 50	59							1		1			1	4	3	5	6	10	9	7	3	7		2	2.39	0.43	
KD 72[y]	6																			2		2	2		2.03		2.36
Robust[z]	8													1	4	2	1								2.66		
Cross 56	83	1	1	4		4	5	8	5	8	6	10	6	7	8	5	1	2	1	1					3.09	0.40	
KD 58[y]	6																		4		2				2.23		2.88
M78-91[z]	4					3		1																	3.55		
Cross 57	83							1	2	1	2	5	2	8	10	13	13	8	6	4	5		2	1	2.58	0.36	
KD 58[y]	6																		4		2				2.23		2.42
Robust[z]	8													1	4	2	1								2.66		

[w] The scores are the mean of 30 single-spike evaluations per family, with 10 spikes per replication. Resistance is indicated by the low means.
[x] Significant differences between F_3 families were measured by LSD ($p = 0.05$).
[y] Resistant parent.
[z] Susceptible parent.

Table 7. Frequency distributions of kernel discoloration scores of F_2 plants and mean scores of F_3 families grown in an irrigated field at St. Paul, Minnesota, inoculated with *Bipolaris sorokiniana*

Cross	Population	No. of F_2 plants and F_3 families	Evaluation method[w]	Distribution[x] S	Distribution[x] R	Ratio	Chi-square	*p*
38	F_2	90	Single spike	86	4	15:1	0.23	0.70
	F_3	44	Single spike	40	4	15:1	0.19	0.80
39	F_2	119	Single spike	101	18	15:1	14.60	0.01
	F_3	46	Single spike	39	7	15:1	4.77	0.05
49	F_2	98	Single spike	59	39	15:1	9.43	0.01
	F_3	83	Single spike	72	11	15:1	5.76	0.03
			Row bulk	67	16	13.3	0.01	0.99
50	F_2	59	Single spike	54	5[y]	15:1	0.07	0.80
	F_3	59	Single spike	40	19	3:1	1.09	0.20
			Single spike	57	2[z]	15:1	0.81	0.30
			Row bulk	45	14	3:1	0.02	0.90
56	F_2	98	Single spike	35	63	1:3	4.38	0.01
	F_3	83	Single spike	81	2	63:1	0.03	0.85
			Row bulk	78	5	15:1	0.02	0.90
57	F_2	99	Single spike	69	30	3:1	0.98	0.40
	F_3	83	Single spike	65	18	15:1	31.0	0.01
			Single spike	80	3[z]	63:1	1.12	0.25
			Row bulk	76	7	15:1	0.35	0.06
			Row bulk	82	1[y]	63:1	0.03	0.85

[w] F_3 families of crosses 38 and 39 were not evaluated on the row bulk basis.
[x] The resistant class (R) comprises F_2 plants and F_3 families that were similar to or more resistant than the resistant parent. The susceptible class (S) comprises F_2 plants and F_3 families that were less resistant than the resistant parent.
[y] When scores for the resistant and the susceptible parents overlapped, only plants more resistant than the susceptible parent were classed as resistant.
[z] In crosses in which families were more resistant than the resistant parent, these families were the resistant class.

periods showed that some crosses produced populations that were skewed toward a shorter latent period or susceptibility. This was attributed to the conditioning of a shorter latent period by four to seven loci, with a portion of these loci showing dominance (20).

There were differences in kernel discoloration scores among the resistant parents that may be attributed to different numbers of genes conditioning resistance in the parents or to the influence of the genetic background. The resistant parents had somewhat different pedigrees, though the original source of resistance was Chevron. CI 9539 was derived from a cross of Chevron with Manchuria (D. H. Smith, U.S. Department of Agriculture, Beltsville, MD, *personal communication*). None of the resistant parents were as resistant as Chevron, indicating a loss of resistance alleles or the breakup of epistatic gene combinations.

There were breaks in the F_3 distributions of most crosses. These occurred in the most resistant and most susceptible parts of the distributions. These breaks may be attributed to small populations that did not include all possible phenotypes.

The heritability of resistance to kernel discoloration in barley was low in F_2 and F_3 generations in an earlier study (22). In this earlier study, broad-sense heritability estimates ranged from 18 to 38% in the F_2 generation and from 35 to 53% in F_3. In the present study, the F_2 heritability estimates tended to be slightly higher, ranging from 27 to 43%. The differences in the heritability estimates of the two studies may be due to the removal of susceptible lines from the F_2 populations in the previous study. This selection reduced the genetic variance of the populations.

The heritabilities obtained on the family basis indicate that selection for resistance to kernel discoloration should be more effective in the F_3 generation than in the F_2 generation. Selection on the family basis should increase the chances of obtaining a barley with resistance to kernel discoloration and acceptable agronomic and quality traits.

On the basis of our results, the development of barley with resistance to kernel discoloration may begin in the F_2 generation to remove the most susceptible plants. More stringent selection for resistance could be done in the F_3 generation. To increase homozygosity, seeds of selected F_3 families could be advanced one generation in the greenhouse. In the F_5 generation, lines should be evaluated in replicated field trials.

Since the F_3 means obtained from both single-spike and bulk evaluations were highly correlated, F_3 and F_5 lines could be evaluated by a sample of kernels from bulked spikes of each row. This approach requires much less effort than scoring individual spikes.

Table 8. Individual and family heritability estimates obtained from kernel discoloration scores of F_2 and F_3 populations grown in an irrigated field at St. Paul, Minnesota, inoculated with *Bipolaris sorokiniana*

Cross	Parents (R/S)[w]	Heritability estimates (%)		
		Individual basis[x]	Family basis[y]	
			Single plant	Family bulk
38	KD 627/Robust	39	53	—[z]
39	KD 390/Morex	27	60	—[z]
49	KD 72/Morex	39	60	52
50	KD 72/Robust	39	48	64
56	KD 58/M78-91	38	76	69
57	KD 58/Robust	43	75	67

[w] R = resistant parent; S = susceptible parent.

[x] Heritability estimates on the individual plant basis were obtained by parent-progeny correlations of the kernel discoloration scores of F_2 plants with the mean scores of single spikes from F_3 families. $r = \text{Cov } F_2, F_3/(\text{Var } F_2 \times \text{Var } F_3)^{-2}$.

[y] Heritability estimates on the family basis were obtained from variance components from F_3 families evaluated as single spikes within families. H_B = (mean square among F_3 families − mean square of block × family)/mean square among F_3 families.

[z] Crosses 38 and 39 were not evaluated for kernel discoloration on the bulk basis.

Table 9. Correlation of respective kernel discoloration scores of F_2 spikes and F_3 family means from both bulk and single-spike evaluations of barley crosses grown in an irrigated field at St. Paul, Minnesota, inoculated with *Bipolaris sorokiniana*

Cross	Correlation[z]		
	F_2 single spikes with F_3 bulk	F_2 single spikes with F_3 single spikes	F_3 bulk with F_3 single spikes
49	0.47	0.43	0.80
50	0.42	0.44	0.82
56	0.44	0.40	0.85
57	0.49	0.44	0.83

[z] Pearson's correlation coefficient. All correlations are significant ($p = 0.05$).

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LITERATURE CITED

1. Anderson, W. H. 1974. Studies on the nature of resistance in barley to headblight incited by *Helminthosporium sativum*. Ph.D. thesis, University of Minnesota, St. Paul. 83 pp.
2. Anderson, W. H., and Banttari, E. E. 1976. The effect of *Bipolaris sorokiniana* on yield, kernel weight and kernel discoloration in six-row spring barleys. Plant Dis. Rep. 60:754-758.
3. Banttari, E. E., Anderson, W. H., and Rasmusson, D. C. 1975. Helminthosporium head blight resistance in six row spring barleys. Plant Dis. Rep. 59:274-277.
4. Christensen, J. J. 1963. Variability in the microflora in barley kernels. Plant Dis. Rep. 47:635-638.
5. Christensen, J. J., and Stakman, E. C. 1935. Relation of *Fusarium* and *Helminthosporium* in barley seed to seedling blight and yield. Phytopathology 25:309-327.
6. Clark, R. V. 1966. Reaction of barley lines to root rot, leaf spot and head blight. Can. J. Plant Sci. 46:603-609.
7. Clark, R. V., and Wallen, V. R. 1969. Seed infection of barley by *Cochliobolus sativus* and its influence on yield. Can. Plant Dis. Surv. 49:60-64.
8. Conner, W. J. 1971. Pages 363-367 in: Practical Nonparametric Statistics, 2nd ed. John Wiley & Sons, New York. 492 pp.
9. Crosier, W. F., and Waters, E. C. 1959. *Fusarium graminearum* and other fungi in seed stocks of small grains. Plant Dis. Rep. 43:1013-1015.
10. Greaney, F. J., and Machacek, J. E. 1942. Prevalence of seedborne fungi on cereals in certain seed inspection districts of Canada. Sci. Agric. 22:419-437.
11. Immer, F. R., and Christensen, J. J. 1943. Studies on susceptibility of varieties and strains of barley to Fusarium and Helminthosporium kernel blight when tested under muslin tents or in nurseries. J. Am. Soc. Agron. 35:515-522.
12. Loiselle, R. 1965. Inheritance of resistance to root rot and seedling blight of barley caused by *Helminthosporium sativum*. Can. J. Plant Sci. 45:238-242.
13. Machacek, J. E., and Greaney, F. S. 1938. The black-point or kernel smudge disease of cereal seed. Can. J. Res. Sect. C 16:84-113.
14. Mead, J. W. 1931. A study of seed trouble in relation to root-rot of cereals. Pages 84-90 in: Can. Dep. Agric. Rep. Dom. Bot. 1930.
15. Mead, J. W. 1942. Host-parasite relationships in a seedborne disease of barley caused by *Helminthosporium sativum* P. K. and B. Can. J. Res. Sect. C 20:501-523.
16. Miles, M. R. 1984. Kernel discoloration of barley: Effects of genotype, environment and microflora. M.S. thesis, University of Minnesota, St. Paul. 156 pp.
17. Miles, M. R., and Wilcoxson, R. D. 1984. Production of fungal inoculum using a substrate of perlite, cornmeal, and potato-dextrose agar. Plant Dis. 68:310.
18. Miles, M. R., Wilcoxson, R. D., Rasmusson, D. C., Wiersma, J., and Warnes, D. 1987. Influence of genotype and environment on kernel discoloration of midwestern malting barley. Plant Dis. 71:500-504.
19. Mirocha, C. J., Schauerhamer, B., and Pathre, S. V. 1974. Isolation, detection and quantitation of zearalenone in maize and barley. J. Assoc. Off. Anal. Chem. 57:1104-1110.
20. Parlevliet, J. E. 1978. Further evidence of polygenic inheritance of partial resistance in barley to leaf rust, *Puccinia hordei*. Euphytica 27:369-379.
21. Parlevliet, J. E., and Kuiper, H. J. 1985. Accumulating polygenes for partial resistance in barley to barley leaf rust, *Puccinia hordei*. I. Selection for increased latent periods. Euphytica 34:7-13.
22. Wilcoxson, R. D., Rasmusson, D. C., Banttari, E. E., and Johnson, D. A. 1980. Feasibility of selecting for resistance to kernel discoloration in barley. Plant Dis. 64:928-930.