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Heritability of common root rot and spot blotch resistance in barley

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Heritability of resistance to common root rot and spot blotch [*Cochliobolus sativus*] was investigated in two resistant-by-susceptible barley crosses. Parental cultivars, F_1s and progeny lines were evaluated for common root rot reaction in a field disease nursery in four experiments and for spot blotch reaction in two growth chamber experiments. The heritability of common root rot resistance of cross Fr926-77 × Deuce ranged from 56 to 85%. Heritability for spot blotch resistance in this cross was 43 and 61%. For the cross Virden × Ellice, heritability for common root rot resistance ranged from 53 to 78%, and heritability for spot blotch resistance was 73 and 78%. Common root rot resistance appeared to be conditioned by dominant genes in Virden × Ellice, but the genetic basis of the resistance could not be determined for Fr926-77 × Deuce. The continuous nature of the distributions of mean disease reactions of progeny lines and estimation of the number of genes conditioning resistance indicated that inheritance of resistance for both diseases was quantitative in nature. No association between common root rot reaction and spot blotch reaction was detected.

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On a étudié l'héritabilité de la résistance à la pourriture sèche et à la tache helminthosporienne [*Cochliobolus sativus*] chez deux croisements entre une orge résistante et une orge sensible. On a évalué le comportement des cultivars parentaux, de la F_1 et des lignées de descendants à l'égard de la pourriture sèche inoculée dans le cadre de quatre expériences dans une pépinière temporaire, de même que leur comportement à l'égard de la tache helminthosporienne dans deux expériences en chambre de croissance. Pour le croisement Fr926-77 × Deuce, l'héritabilité de la résistance variait de 56 à 85 % dans le cas de la pourriture sèche et de 43 à 61 % dans celui de la tache helminthosporienne. Pour le croisement Virden × Ellice, l'héritabilité de la résistance variait de 53 à 78 % dans le premier cas et de 73 à 78 % dans le second. La résistance à la pourriture sèche semblait être conditionnée par des gènes dominants dans le croisement Virden × Ellice, mais il a été impossible d'identifier la source génétique de la résistance dans le croisement Fr926-77 × Deuce. La distribution uniforme des comportements moyens des lignées de descendants à l'égard des maladies et l'estimation du nombre de gènes conférant la résistance laissent entendre que l'héritabilité de la résistance aux deux maladies était quantitative. On n'a décelé aucune associaion entre la réaction à la pourriture sèche et celle à la tache helminthosporienne.

Common root rot and spot blotch [*Cochliobolus sativus* (Ito & Kurib.) Drechsl. ex Dastur, anamorph *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem., syn. *Helminthosporium sativum* Pamm., King & Bakke] are barley diseases that are present in most cereal growing areas of the world. Common root rot is a problem in the drier temperate regions (Mathre 1982) and spot blotch is economically important in humid temperate and tropical areas (Clark 1979, Hetzler et al. 1991). Annual yield reductions due to common root rot were estimated to average 10% on the Canadian prairies (Piening et al. 1976). In Ontario and Quebec, spot blotch has been found to cause yield reductions of 20 to 30% in commonly grown barley cultivars (Clark 1979, Dostaler et al. 1987).

Variation in resistance exists in barley for both common root rot and spot blotch (Duczek 1984, Wilcoxson et al. 1990). The use of genetic resistance to control common root rot and spot blotch is desirable because additional grower inputs are not required. Heritability determines the contribution of the hereditary and environmental components of the phenotype of an individual (Falconer 1981); it is a measure of the proportion of the total variance of a trait attributable to the average effect of the genes conditioning the trait. Prediction of the response to selection is assisted by knowledge of the heritability and the genetics of resistance.

Relatively few studies have been conducted on the heritability and genetics of resistance to common root rot of barley. Common root rot resistance has been attributed to many genes and quantitative inheritance (Cohen et al. 1969). However, one report suggested resistance was conditioned by two dominant genes (Loiselle 1965). In wheat, resistance to common root rot was reported to be due to one major recessive gene and one or two minor genes in one cross (McKenzie & Atkinson 1968), and by at least three genes in two other crosses (Bailey et al. 1988). Sallans and Tinline (1965) suggested resistance in wheat was heritable, and quantitative in nature, but heritability values were not determined.

A number of researchers have reported qualitative inheritance for spot blotch resistance, with one to a few genes conditioning resistance (Arny 1951, Brandt 1955, Hayes & Stakman 1921, Luthra & Rao

1973, Wilcoxson et al. 1990). Jorgensen (1990) listed four genes, each present in various crosses, conditioning resistance to spot blotch in barley; three were located on chromosomes II, V, and VII, respectively. The objective of this study was to determine the heritability of common root rot and spot blotch resistance in two barley crosses.

Materials and methods

Germplasm. Parents, F_1s, and progeny from two hybrid combinations were evaluated for resistance to common root rot and spot blotch. The cross Fr926-77 × Deuce (F × D) was chosen because the progeny were doubled haploid lines (200) derived from another culture of F_1 plants (courtesy of K.N. Kao, Plant Biotechnology Institute, Saskatoon). Fr926-77 has the pedigree: Traill*2/3/*H. depressum/H. compressum//H. bulbosum* 55/*H. vulgare**4/4/Conquest/5/ ND1381/Larker//Beacon; it is a 6-row breeding line of feed quality (personal communication, J. Franckowiak, North Dakota State University, Fargo, N.D.). Preliminary data indicated it was more susceptible to common root rot (disease rating 61 ± 4.3%) than Deuce (42 ± 6.4%) and more resistant to spot blotch (47 ± 2.0%) than Deuce (62 ± 2.3%). Deuce has the pedigree Summit/Norbert and is a 2-row feed barley (Agriculture Canada Licence 2650).

For the cross Virden × Ellice (V × E), 259 F_5 recombinant inbred lines produced by single seed descent were evaluated. An additional benefit of this cross was that Virden was highly resistant and Ellice very susceptible to both diseases. The F_7 generation was evaluated for common root rot reaction in 1991 and both the F_7 and F_8 generations in 1992. Spot blotch was assessed on the F_8 generation in two experiments. Virden is a 6-row feed barley (Agriculture Canada Licence 2834). It has the pedigree: WA6415-66/4/Bonanza/3/Dickson//Trail/-NDB112*2/5/Glenn//Bonanza/Dickson, according to R. Wolfe (personal communication), where WA6415-66 was a sister selection of Steptoe. Field evaluations over at least 8 station-years at Saskatoon and Scott indicated that Virden was highly resistant to common root rot (16 ± 4.0%) whereas Ellice, a 2-row malting type, was more susceptible (55 ± 7.0%). Ellice (Agriculture Canada Licence number 2715) has the pedigree: CI5791/Parkland//Betzes/3/ Betzes/Piroline/4/Akka/5/Centennial/6/ Klages/7/ Cambrinus/Tern. Preliminary data collected for this study indicated a more resistant spot blotch reaction for Virden (24 ± 2.3%) than Ellice (43 ± 4.5%).

Common root rot. Common root rot was evaluated in naturally infested field nurseries in replicated experiments consisting of 2-m-long single row plots planted 6–8 cm deep at two locations in each of two years. Twenty-five to thirty plants were evaluated per plot at the heading to ripening growth stage (Feekes scale 10.1 to 11). Disease severity (%) was determined as the number of severely infected plants divided by the total number of plants evaluated multiplied by 100. Severely infected plants were those with dark brown discoloration and lesions covering 50% or more of the subcrown internode.

In 1991, the F × D cross was evaluated in a 4-replicate experiment at Saskatoon and a 2-replicate experiment at Scott, and cross V × E was evaluated in 2 replicates at Saskatoon. In 1992, cross F × D was evaluated at two locations: 4 replicates at Saskatoon and 2 replicates at the Goodale Crop Research Farm. The F_7 and F_8 generations of cross V × E were evaluated at Saskatoon in 1992, using 3 replicates, and in 2 replicates for the F_8 generation at the Goodale Crop Research Farm. The number of progeny lines from each cross varied with year and location (experiment) due to availability of seed. The large number of progeny lines required the grouping of lines in each experiment into 2 to 6 tests depending on the site and year. Each test included a number of progeny lines, the parent cultivars, F_1 if available, and the susceptible check Brier.

Spot blotch. Two isolates of *C. sativus* (SB and 2715) were used as inoculum in spot blotch evaluations. Each isolate was grown separately in 9 cm petri dishes on minimal media agar at 20°C under 12 hours of natural light. After 7–10 days conidia were collected by flooding the plates with approximately 10 mL of sterile distilled water and scraping the agar surface with a metal spatula to dislodge the conidia. Equal volumes of conidial suspension from each isolate were combined and filtered through four layers of cheesecloth. Conidial concentration was adjusted to 5600 conidia per mL using a hemacytometer.

Spot blotch experiments were conducted in growth chambers with a daily cycle of 14 h florescent lighting supplemented with incandescent bulbs (275 $\mu E\ cm^{-2}\ sec^{-1}$) at 22°C and 10 h dark at 16°C. Plants were grown in 10-cm square pots using soil-less mix (Stringam 1971). Eight seeds per genotype were planted in a pot and seedlings thinned to six after two weeks.

Plants at the 3-leaf stage were inoculated with a hand held spray bottle (two weeks after seeding). An initial misting with 200 mL of 0.1% Tween 20 solution was applied over 48 pots, followed immediately by inoculation with 500 mL of conidial suspension containing 0.2% Tween 20. Plants were placed in humidity chambers (polyethylene-wrapped wooden frames) which accommodated 48 pots. In the first evaluation of both crosses (Experiment 1), mist from an ultrasonic humidifier (model SH8DH, Super Electric Co. (Canada) Ltd.) was directed into an open end of the chamber continuously for 24 h after inoculation, then for alternating periods of 1 h for the fol-

lowing 24 h. Chambers were sealed and plants rated 5 days later (i.e. 1 week after inoculation). In the second evaluation of the crosses (Experiment 2) chambers were completely sealed after inoculation and mist from the humidifier was applied through a tube attached to one end of the chamber. Mist was applied continuously for the first 24 h and in alternating 30 minute intervals for the following 24 h. The mist tube was then sealed and plants were rated 5 days later.

The Horsfall-Barratt (1945) rating scale was used to evaluate spot blotch on three leaves of each of six plants per genotype, using 11 categories based on the percentage leaf area infected. Lines were evaluated in groups of 12 including the check cultivar, Bonanza. Each line was replicated four times within the chamber. These 48 pots (12 lines replicated 4 times) were considered a test. An experiment consisted of sufficient tests to evaluate all lines from a cross. Variation among tests within experiments was accounted for by partitioning the sums of squares for tests. Two experiments were conducted for each cross.

Analysis. Analysis of variance and parent-offspring regression procedures (Statistical Analysis System, 1989, version 6.07, SAS Institute Inc., Cary, North Carolina) were used to calculate variation among experiments and heritability. The doubled haploid progeny of cross F × D were homozygous and therefore without genetic variance within progeny lines. The error mean square from the analysis of doubled haploid progeny supplied an estimate of environmental variance (Table 1). The progeny of cross V × E were not assumed to be completely homozygous since they were derived from a single seed descent procedure, F_2 through to F_5, and then each line was bulked to the F_7. An estimate of environmental variance was obtained from the nonsegregating parental and F_1 generations. However, in all cases this estimate was not significantly different from the estimate of environmental variance supplied by the error mean square from the analysis of the progeny lines as determined by an *F* test. Therefore the latter estimate of environmental variance was used in the calculation of heritability.

Heritability was calculated from mean squares of the analysis of variance from each year and location on a mean plot basis as follows:

$$h^2 = \sigma^2_g/(\sigma^2_g + \sigma^2_e)$$
$$= Vg_a/[Vg_a + Ve/r], \text{ where:}$$
$$Vg_a = (MS3 - MS4)/r,$$
$$Ve = MS4,$$
$$r = \text{replicates}$$

Standard error (S.E.) of heritability was calculated from the formula of Becker (1984), which used variances of the variance components in the heritability estimates:

$$\text{S.E. } (h^2) = \sqrt{\{[y^2 * Var(x) + x^2 * Var(y) - 2xy * Covar(x,y)]/y^4\}}, \text{ where:}$$

$$x = Vg_a = (MS3 - MS4)/r,$$
$$y = Vg_a + Ve = (MS3 - MS4)/r + MS4/r,$$
$$Var(x) = \{[2(MS3)^2/(df+2)] + [2(MS4)^2/(df+2)]\},$$
$$Var(y) = [(2(MS3)^2/(df+2)], \text{ and}$$
$$Covar(x,y) = [(2(MS3)^2/(df+2)]$$

The minimum number of loci conditioning the difference in reaction of two parents to common root rot and spot blotch was calculated using the Castle-Wright method of moments as modified by Mulitze and Baker (1985).

The parent-offspring regression method of calculating heritability (Falconer 1981) was used for cross V × E at Saskatoon in 1992 where F_7 and F_8 progeny were grown within the same experiment. The F_7 and F_8 progeny lines were not paired; each received separate randomization to prevent confusion of environmental and genetic effects. The regression coefficient of F_8 on F_7 lines represented the heritability of common root rot reaction. The results of the parent-offspring regression method were compared with results from the analysis of variance method for this experiment.

The genetics of resistance was examined for each cross for common root rot by comparing disease reaction means for the parent cultivars and the F_1 obtained within the same test of each experiment (the F_1 was present only in one test of each experiment). Spearman's coefficient of rank correlation (Little & Hills 1978) was used to evaluate relationships among common root rot and spot blotch reactions for various experiments.

Table 1. Analysis of variance for double haploid (F × D) and recombinant inbred (V × E) progeny, mean squares, and genetic and environmental variance components used to calculate heritability for reaction to common root rot and spot blotch from each experiment

Source of variation	df†	Mean squares	Variance components‡
Test	t - 1	MS1	
Replicate (Test)	t(r - 1)	MS2	
Genotype (Test)	$\sum_{i=1}^{t} (g_i - 1)$	MS3	$Ve + rVg_a$
Error	$\sum_{i=1}^{t} [(r - 1)(g_i - 1)]$	MS4	Ve
Total			

† Degrees of freedom where t = number of tests in an experiment, r = number of replications, and g = number of genotypes.
‡ Variance components where Vg_a = genetic variance among lines, and Ve = environmental variance among and within lines.

Results

Common root rot. Distributions of mean common root rot reactions of progeny lines of cross F × D did not show any clearly defined groups related to resis-

Table 2. Distributions of common root rot and spot blotch reactions in 5% intervals and the mean reaction (%) ± standard error (S.E.) of progeny lines of the crosses Fr926-77 × Deuce (F × D) and Virden × Ellice (V × E) from each experiment

Cross and experiment		Mean (%)	S.E.	No. of progeny	Upper limit of common root rot class (%)																				
					5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100
Common root rot																								
F×D	Saskatoon 1991	50.4	1.1	163				1	4	9	13	13	22	18	24	12	23	13	9	1	1			
	Scott 1991	60.9	1.6	143				3	4	3	8	8	10	8	10	9	11	23	15	10	9	6	4	2
	Saskatoon 1992	57.6	1.3	200				3	5	5	11	15	14	20	17	20	15	18	14	19	16	4	3	1
	Goodale 1992	46.8	1.2	198			6	7	7	12	26	13	22	29	18	17	13	11	10	4	1	2		
V×E	Saskatoon 1991	67.4	1.0	256				1	1	0	5	8	10	22	19	26	21	28	22	30	27	19	13	4
	Goodale 1992	42.4	1.2	236		3	1	8	13	20	23	20	26	28	19	22	16	10	12	8	1	3	3	
	Saskatoon F_7 1992	17.5	0.8	259		35	56	43	35	29	20	15	8	5	6	0	6	1						
	Saskatoon F_8 1992	18.5	0.8	259		26	62	40	39	21	27	12	11	6	10	2	1	0	1	0	1			
Spot blotch																								
F×D	Experiment 1	35.6	0.9	192			5	19	20	24	30	24	21	23	16	4	4	1	1					
	Experiment 2	39.1	1.0	199			2	17	22	21	18	25	28	22	15	13	7	5	4					
V×E	Experiment 1	35.6	0.8	229			4	17	22	37	40	28	30	22	12	9	6	2						
	Experiment 2	47.0	0.8	263			2	3	10	7	26	32	36	42	36	22	25	11	7	3	1			

tant or susceptible reactions (Table 2). Differences between the F × D parents were obtained only at Saskatoon in 1992 where Fr926-77 was lower than Deuce (Table 3). The F_1 of cross F × D had a lower disease severity rating (9.1 ± 4.8) than either parent at Saskatoon in 1991 ($P = 0.01$). In 1992 at Saskatoon the disease reaction of the F_1 (25.3 ± 5.2) was similar to Fr926-77 and significantly less than Deuce ($P = 0.01$). At Goodale, differences in disease reaction were not detected among the parents or the F_1 ($P = 0.05$). The length of the subcrown internodes of Deuce were observed to be very short, usually less than 1 cm for most plants. This observation was noted on at least 10 of the 2-row progeny lines.

The heritability estimates for common root rot reaction for cross F × D, calculated from mean squares from the analysis of variance, ranged from 56 to 85% over the four experiments (Table 4). The minimum number of loci conditioning common root rot reaction in each experiment was greater than three and therefore quantitative in nature. The coefficients of variation were between 23 and 30% for these experiments.

The distribution of V × E progeny lines in 1991 indicated a higher level of disease than in 1992 (Table 2). The distribution of F_7 and F_8 lines at Saskatoon in 1992 showed a skewed distribution of genotype means toward resistance. No clearly defined groups related to resistant or susceptible reactions were distinguished. Significant differences between the mean common root rot reactions of Virden and Ellice were detected in all experiments with Virden lower than Ellice (Table 3). In 1991, the mean common root rot reaction of the F_1 (67.9 ± 9.4) was near the mid-parent value and significantly different from both parents ($P \leq 0.05$). In 1992, at Saskatoon the F_1 was similar in common root rot reaction (5.6 ± 2.9) to Virden and significantly lower than Ellice ($P = 0.01$). At Goodale the F_1 was again similar (25.9 ± 12.6) to Virden and less than Ellice ($P = 0.12$).

Heritability estimates of common root rot reaction in the V × E cross using the analysis of variance method ranged from 59 to 78% (Table 4). Heritability of common root rot reaction as determined by the parent-offspring regression method, using the F_7 and F_8 progeny lines at Saskatoon in 1992, was 53% with a standard error of 3% ($R^2 = 0.27$). Quantitative inheritance of common root rot reaction was suggested since the minimum number of loci conditioning common root rot reaction was estimated at greater than 3 in all experiments. Coefficients of variation were 20% at Saskatoon 1991, 34% at Goodale 1992, 59% for the F_7, and 61% for the F_8 generation at Saskatoon 1992.

Spot blotch. The reactions of most of the progeny lines of cross F × D were between class limits

Table 3. Number of observations (n), mean percentage common root rot (CRR) reaction ± standard error (S.E.) of the susceptible check Brier and the parent cultivars of the crosses Fr926-77 × Deuce and Virden × Ellice

	Saskatoon 1991		Scott 1991		Saskatoon 1992		Goodale 1992	
Cultivar	n	CRR ± S.E.	n	CRR ± S.E.	n	CRR ± S.E.	n	CRR ± S.E.
Fr926-77 × Deuce								
Fr926-77	8	65.5 ± 5.9	4	55.0 ± 10.2	64	30.7 ± 2.0	32	30.6 ± 2.1
Deuce	8	49.0 ± 5.1	4	53.8 ± 10.3	64	68.7 ± 2.0	32	35.6 ± 2.3
Brier	48	76.5 ± 2.0	16	81.6 ± 5.2	64	89.2 ± 1.0	28	68.6 ± 2.7
Virden × Ellice								
Virden	6	32.2 ± 7.6			72	3.1 ± 0.4	24	19.4 ± 2.7
Ellice	6	93.9 ± 2.9			72	51.3 ± 1.8	24	69.0 ± 3.9
Brier	6	80.5 ± 4.5			72	74.6 ± 1.8	24	67.4 ± 2.9
t-test comparison								
Fr926-77 vs. Deuce		ns		ns		**		ns
Virden vs. Ellice		**				**		**

ns not significant at $P = 0.05$: ** significant at $P = 0.01$.

Table 4. Source of variation, mean squares, and heritability (h^2) ± standard error (S.E.) from analysis of variance of progeny of cross Fr926-77 × Deuce and Virden × Ellice for common root rot reaction in 1991 and 1992

Source of variation	Mean squares	df	MS	df	MS	df	MS	df	MS
Fr926-77 × Deuce		1991				1992			
		Saskatoon		Scott		Saskatoon		Goodale	
Genotype (Test)	MS3	161	747	141	476	196	1288	194	445
Error	MS4	483	203	141	209	587	189	194	191
h^2 ± S.E. (%)		73 ± 13.9		56 ± 14.7		85 ± 6.8		57 ± 12.3	
Virden × Ellice		1991		1992					
		Saskatoon		Goodale		Saskatoon F_7		Saskatoon F_8	
Genotype (Test)	MS3	253	515	233	515	253	486	253	478
Error	MS4	253	192	233	210	506	108	506	127
h^2 ± S.E. (%)		63 ± 9.3		59 ± 10.6		78 ± 7.2		74 ± 8.7	

20–55% in both experiments (Table 2). However, the spot blotch class in which the maximum number of progeny lines occurred and the overall mean, were higher in Experiment 2 than in Experiment 1. Fr926-77 was more resistant to spot blotch than was Deuce in both experiments (Table 5).

Heritability estimates of 43 and 61% were obtained for spot blotch in the cross F × D (Table 6). Calculations of the minimum number of loci conditioning spot blotch reaction indicated quantitative inheritance in this cross. Coefficients of variation for progeny lines were similar in both experiments (27 and 31%).

Distributions of mean spot blotch reactions of progeny lines from cross V × E were similar in both experiments, although the mean reaction of all lines was greater in Experiment 2 than in Experiment 1 (Table 2). Significant differences between the parents in spot blotch reaction were obtained in both experiments with Virden consistently lower than Ellice (Table 5). Coefficients of variation were the same (21%) in the analyses of variance for progeny lines in each experiment. Heritability estimates of spot blotch reaction in the V × E cross were 73 and 78% (Table 6). Quantitative inheritance was suggested from the calculation of the minimum number of loci controlling spot blotch reaction in this cross.

Table 5. Number of observations (n), mean percentage spot blotch (SB) reaction ± standard error (S.E.) of the susceptible check Bonanza and the parent cultivars of the crosses Fr926-77 × Deuce, and Virden × Ellice

	Experiment 1		Experiment 2	
Cultivar	n	SB ± S.E.	n	SB ± S.E.
Fr926-77 × Deuce				
Fr926-77	20	35.9 ± 3.4	20	30.6 ± 3.4
Deuce	20	49.2 ± 3.5	20	49.0 ± 5.3
Bonanza	84	42.8 ± 1.4	80	39.6 ± 1.5
Virden × Ellice				
Virden	28	21.5 ± 1.5	32	30.9 ± 2.7
Ellice	28	48.6 ± 2.3	32	60.3 ± 3.0
Bonanza	92	38.3 ± 1.3	104	48.8 ± 1.6
t-test comparison				
Fr926-77 vs. Deuce		**		**
Virden vs. Ellice		**		**

** significant at $P = 0.01$.

Table 6. Sources of variation and mean squares, and heritability (h^2) ± standard error (S.E.) from analysis of variance of progeny of crosses Fr926-77 × Deuce and Virden × Ellice, for spot blotch reaction in each experiment

Source of variation	Mean squares	Fr926-77 × Deuce				Virden × Ellice			
		Experiment 1		Experiment 2		Experiment 1		Experiment 2	
		df	MS	df	MS	df	MS	df	MS
Genotype (Test)	MS3	184	248	180	267	206	247	237	380
Error	MS4	552	96	540	152	618	55	710	101
h^2 ± S.E. (%)		61 ± 18.5		43 ± 27.6		78 ± 10.1		73 ± 11.3	

Relationship between common root rot and spot blotch. Spearman correlation coefficients on progeny mean disease reactions between common root rot and spot blotch experiments were significant for only cross F × D between common root rot reaction at Scott, 1991 and spot blotch reaction in Experiment 2. In this case, there was a weak inverse correlation (-0.28).

Correlations among common root rot experiments were significant for all experiments for both crosses and ranged from 0.28 to 0.66. Spot blotch experiments of cross F × D were not correlated, although a significant correlation of 0.45 was obtained between spot blotch experiments of cross V × E.

Discussion

Heritability estimates of common root rot resistance were moderate to high in this study. This is similar to a heritability estimate of 56.7% in a barley cross reported by Bailey and Wolfe (1994). In wheat, Bailey et al. (1988) reported estimates of greater than 70% for early generations in one cross and 32–78% in another. Estimates of the minimum number of loci conditioning common root rot reaction (based on analysis of progeny lines) were greater than three in all experiments for both crosses, indicative of quantitative inheritance. Distributions of mean common root rot reactions of progeny lines indicated that simple gene ratios were not present. Quantitative inheritance of common root rot reaction is in agreement with the conclusions of Cohen et al. (1969), who obtained similar results in barley. In wheat, Sallans and Tinline (1965) suggested reaction to common root rot was a heritable trait based on consistent reactions among highly resistant and highly susceptible progeny lines of crosses at four or five locations over two years. They found no simple ratios of resistant and susceptible classes in the segregating progenies they examined and concluded, as in the present study, that resistance was quantitative.

Observations on F_1 means for cross V × E indicated the inheritance of resistance was not dominant in 1991 but was in 1992. However, the estimates obtained on the F_1 in 1991 were not as precise as in 1992 due to poor germination of F_1 seed in 1991. On average only 11 plants were evaluated (range of 7 to 16) for each F_1 plot in 1991. In 1992 30 plants were evaluated in each plot and similar results were obtained at the two locations. Therefore it was concluded that resistance to common root rot in cross V × E was conditioned by dominant genes.

Heritability estimates for common root rot reaction among experiments were more variable for cross F × D than for cross V × E. This may have been due to a problem of disease evaluation on short subcrown internodes in F × D. Disease intensity ratings may have increased unintentionally due to the subjective nature of disease assessment. For example, a small lesion may have resulted in a more severe rating due to the small relative size of the internode. Differences in soil conditions among experiments may have influenced seeding depths and in turn the length of the subcrown internodes which affected disease assessment. Common root rot severity has been found to increase with depth of seeding (Duczek & Piening 1982). These factors, coupled with a possible genetic predisposition of Deuce to short subcrown internodes, may also have contributed to variability in estimates of disease reactions among experiments. Short subcrown internodes, as compared to normal subcrown internodes, might be expected to have a lower probability of contact with inoculum and therefore reduced infection; however, in a disease nursery the chance of disease escape was limited.

Deuce had been tested from 1983 to 1990 and consistently rated moderately resistant relative to other cultivars, with a disease rating of 42% ± a standard error of 6.4%. Preliminary evaluation of parent lines had also suggested that Deuce was the more resistant parent in this F × D cross. These observations suggest that the disease evaluation of Deuce in this experiment was not a true measure of its reaction to common root rot since a resistant reaction was expected but was not obtained. This reaction prevented us from making conclusions concerning the genetics of resistance.

Although the disease reaction of Deuce was inconsistent with the preliminary evaluations, the estimates of heritability in the cross F × D were calculated from the homozygous doubled haploid progeny lines only. Therefore the common root rot reactions of the parents were not critical to these calculations. In addition, Fr926-77 and 95% of the progeny lines did not have

short subcrown internodes, so disease ratings for them should not have been affected. This is supported by the distributions of progeny lines that were consistent over years and locations (Tables 2 and 3).

Moderate to high heritability estimates for spot blotch reaction and quantitative inheritance were determined in this study. These results differ from those of other researchers who found spot blotch resistance in barley to be qualitative in nature (Hayes & Stakman 1921, Arny 1951, Brandt 1955, Luthra & Rao 1973, Wilcoxson et al. 1990). Those studies were based on lesion type and used various rating scales to quantify the data. Wilcoxson et al. (1990) suggested that resistance to spot blotch was probably conditioned by one or two genes in germplasm derived from a well known source of resistance to spot blotch, NDB112. The spot blotch resistant parents in this study (Fr926-77 and Virden) may also carry resistance genes from NDB112. Fr926-77 was derived from very diverse germplasm, including a number of wild species and the cultivars Beacon and Traill, both of which have NDB112 in their background (J. Franckowiak, personal communication; Wilcoxson et al. 1990). The pedigree of Virden contained the cultivar Dickson, which also has NDB112 resistance (Wilcoxson et al. 1990). Continuous distributions of mean spot blotch reactions from resistant to susceptible obtained for both crosses in this study did not lend themselves to classification into distinct groups, supporting the conclusion of quantitative inheritance, which differed from the results of Wilcoxson et al. (1990).

The analysis of variance and parent-offspring regression methods of heritability estimation were used for cross V × E at Saskatoon, 1992. The estimate was lower for the parent-offspring regression method than for the analysis of variance method. This agrees with Frey and Horner (1955), who found the parent-offspring regression method tended to underestimate true heritability values, whereas the analysis of variance method gave values which closely approximated the results obtained in selection experiments. Parent-offspring regression is a straightforward method of calculating heritability and gives a more reliable measure of the standard error (Falconer 1981). However, the coefficient of determination (R^2 = 0.27) in this study indicated that the proportion of the total variation of the F_8 generation explained by regression on the F_7 was small. This suggested little genetic variation relative to environmental variation between generations. By the F_7 generation, lines were essentially homozygous and therefore differed little from the F_8 generation.

Little relationship was found between common root rot and spot blotch resistance, suggesting that genes controlling common root rot reaction in these crosses are not the same as those controlling spot blotch reaction. Similar results were reported by Clark (1966), who found that all progeny from an interspecific cross of barley were susceptible to spot blotch but had a wide range of reactions to common root rot. In a study of 16 wheat lines, Ahmed (1989) found that some lines were resistant to both common root rot and spot blotch, while other lines showed differential reaction to the two diseases. Conner (1990) examined 18 wheat lines for reaction to common root rot, spot blotch, and black point and detected no strong correlations among reactions to the diseases.

In conclusion, common root rot and spot blotch reactions in both crosses in this study were heritable. Estimates of the minimum number of loci conditioning resistance, plus the continuous nature of the distributions of progeny line means indicated quantitative inheritance for both diseases. The resistance to common root rot appeared to be conditioned by dominant genes for cross Virden × Ellice. No association was found between reaction to common root rot and reaction to spot blotch. The heritability estimates obtained in this study indicate that selection for common root rot and spot blotch resistance in barley should be successful, but that selection and evaluation should be delayed until later generations unless doubled haploids are used.

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