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Title: EFFECT OF SCALD (RHYNCHOSPORIUM SECALIS) ON YIELD AND YIELD COMPONENTS OF TWELVE WINTER BARLEY (HORDEUM VULGARE)
GENOTYPES.

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Abstract approved:

Patrick M. Hayes

The effects of scald epidemics, induced by Rhynchosporium secalis
(Oud.) Davis, on the yield and quality of winter malting barley have not been reported. The principal objective of this investigation was to assess yield and quality losses in resistant and susceptible winter barley genotypes in diverse environments of the Pacific Northwest region of the United States. As consistent, uniform infection is required for resistance breeding, three inoculation methods were compared with a fungicide-protected check. Disease development was poor at one location (Pendleton) and excellent at the second (Corvallis). Yield reductions at Corvallis ranged from 27-40%. Resistant genotypes showed lower disease severities, and yield losses than susceptible genotypes. Natural infection was as effective in generating epidemics as infected straw or spore inoculation. Of the yield components, the number of spikes per unit area was the most affected by scald. There were no consistent effects of disease on grain weight, although kernel plumpness was reduced

under disease pressure. Genotype x environment interactions were pronounced. Resistant genotypes were higher yielding than susceptible genotypes in fungicide-protected plots under disease pressure. The opposite was true under minimal disease pressure.

EFFECT OF SCALD (RHYNCHOSPORIUM SECALIS) ON YIELD AND YIELD COMPONENTS OF TWELVE WINTER BARLEY (HORDEUM VULGARE) GENOTYPES

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Nusret Zencirci

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Professor of Plant Breeding and Genetics, in charge of major

Redacted for privacy

Head of Crop Science Department

Redacted for privacy

Dean of Graduate School

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Typed by Nusret Zencirci

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DEDICATED

TO

ELCIN and GIZEM

AND

TO

OUR PARENTS

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Chapter 1

EFFECT OF SCALD (RHYNCHOSPORIUM SECALIS) ON YIELD AND YIELD COMPONENTS OF TWELVE WINTER BARLEY (HORDEUM VULGARE) GENOTYPES

N. Zencirci and P. M. Hayes

ABSTRACT

The effects of scald epidemics, induced by Rhynchosporium secalis (Oud.) Davis, on the yield and quality of winter malting barley have not been reported. The principal objective of this investigation was to assess yield and quality losses in resistant and susceptible winter barley genotypes in diverse environments of the Pacific Northwest United States. As consistent, uniform infection is required for resistance breeding, three inoculation methods were compared with a fungicide-protected check. Disease development was poor at one location (Pendleton) and excellent at the second (Corvallis). Yield reductions at Corvallis ranged from 27-40%. Resistant genotypes showed lower disease severities, and yield losses than susceptible genotypes. Natural infection was as effective in generating epidemics as infected straw or spore inoculation. Of the yield components, the number of spikes per unit area was the most affected by scald. There were no consistent effects of disease on grain weight, although kernel plumpness was reduced under disease pressure. Genotype x environment interactions were pronounced. Resistant genotypes were higher yielding than susceptible genotypes in fungicide-protected plots under disease pressure. The opposite was true under minimal disease pressure.

Additional index words: Yield reduction, scald of barley, winter barley.

INTRODUCTION

Scald, caused by <u>Rhynchosporium secalis</u> (Oud.) Davis, is a barley disease of worldwide importance. Yield losses attributed to scald epidemics range from 5 to 45% (Shipton et al., 1974; Skoropad, 1966). Yield losses are ascribed to reductions in all yield components, particularly kernel weight and kernel number per spike (James et al., 1968; Schaller, 1951). Kernel weight is a trait of key importance for malting barley. Low kernel weight may lead to a malting barley crop being sold for feed at a lower price (Nutter et al., 1985).

Fungicides, such as Tilt (Propiconazole, 42% EC), effectively control scald (Johnston and MacLeod, 1987). Although scald control through genetic resistance is desirable on economic grounds, lasting resistance to pathogens with diverse arrays of virulence genes, such as <u>R. secalis</u> (Jackson and Webster, 1976), may be difficult to achieve. Multi-component mixtures, while effective in reducing scald severity, (McDonald et al., 1988), are not feasible in malting barley production, where the industry demands a homogeneous product meeting exacting quality standards.

Winter-habit malting barley is an attractive cereal crop alternative for the Pacific Northwest. However, there are limited data on yield and quality losses attributable to scald epidemics in the diverse environments characteristic of the region. The principal objective of this study was to relate scald disease severity to losses in yield and quality of elite winter barley lines grown in representative, but distinct, environments of Oregon. Supporting objectives were to compare (i) yield and quality losses in resistant and susceptible genotypes, and (ii) effectiveness of inoculation techniques.

MATERIALS AND METHODS

Twelve genotypes, three inoculation techniques (spore, straw and natural inoculum), and a fungicide-protected check were evaluated using a three-replicate, randomized block design, with a split-plot restriction, at two locations over a two-year period. Inoculation treatments and the fungicide protected check were main plot treatments and they were separated by 1.2 meter strips of wheat. Genotypes were subplots. Each subplot was 2.4 meter-long. Experiments were drill-planted at a seeding rate of 125 kg ha⁻¹ with a 17.5 cm row spacing.

Experiments were grown at Corvallis and Pendleton, Oregon in 1986-87 and 1987-88. The Corvallis environment, with moderate temperature and abundant precipitation, provides an optimum environment for disease development. Total rainfall in the growing season was 93.5 cm in 1986-87 and 95.0 cm in 1987-88. Pendleton, a potentially important winter barley production area, is an environment characterized by a lower rainfall. Total annual rainfall was 40.4 cm in 1986-1987 and 32.6 cm in 1987-1988.

Tilt (Propiconazole, 42% EC) was applied to check plots at a rate of 345 ml ha⁻¹ beginning at the 10.1 growth stage (Feekes scale, after Large,1954) at Corvallis and at the 10.3 growth stage at Pendleton. All inoculation treatments reflected current spectra of virulence genes. Natural inoculation plots were exposed to native inoculum. Infected straw inoculation was as described by Khan et al. (1984). Spore suspensions were applied at a concentration of 3 X 10⁶ ml⁻¹ as described by Piening (1979).

Twelve winter barley lines, both six- and two-row, were sub-plot treatments. The 12 lines, representing elite materials in the Oregon State University barley breeding program, were previously selected based on multi-environment, replicated yield performance. Six lines had long-term mean plot scald ratings of \leq 5 on a 1-9 scale (1=resistant and 9=susceptible) and were considered resistant. Six lines had long-term ratings \geq 7 and were considered susceptible.

Grain yield was measured on a plot basis. The number of spikes was counted on one linear meter of row in each plot. Kernel number per spike was based on 20 spikes per plot selected at random. Kernel weight was obtained from a 100 gram sample. Percent plump kernels was determined by sorting a 100-gram sample of seed on a Niagra sample grader for 30 seconds. Grains remaining on a slotted sieve, 0.238 by 1.91 cm, were weighed and recorded as percent plump grains. In 1986-87, scald severity based on the percentage of leaf area showing typical infection symptoms was estimated at the 10.3 growth stage (Feekes scale, after Large, 1954) in Pendleton and at the 10.2 and 10.5 growth stages in Corvallis. In 1987-88, scald severity was estimated at the 10.5 growth stage in Pendleton and the 10.2, 10.5, and 11.2 growth stages in Corvallis. The Rhynchosporium leaf blotch scale of James (1971) was used to determine severity on the three uppermost leaves of 10 tillers per plot selected at random. Severities on first (flag), second, and third leaves were averaged and used to compare resistant and susceptible genotypes. Data were analyzed using analysis of variance and regression procedures of the Statistical Analysis System (SAS, 1982).

RESULTS AND DISCUSSION

The complexity of host parasite interactions in the barley/scald system was evident in the combined analysis of variance of agronomic traits and disease severity readings. In most cases, second and higher order interactions were significant. Error variances from individual location analyses of variance were heterogenous for all traits according to the Hartley's Maximum F-ratio test (Hartley, 1955). Because of significant interactions and heterogeneity of error, results from Corvallis and Pendleton are considered separately.

PENDLETON EXPERIMENTS

Environmental conditions did not favor disease development at Pendleton during the two years of this study. Overall mean severity readings were consistently low: the maximum was 0.8% in 1986-87 and 1.2% in 1987-88. Scald epidemics are favored by long periods of cool, low temperature (Skoropad, 1966; Mayfield and Clare, 1985). In greenhouse studies, it has been shown that scald requires a relative humidity of 90-100% at 15-21 °C for 24-48 hours to develop lesions (Jackson and Webster, 1976; Zhang et al., 1987). These conditions are rarely met in Pendleton and similar environments of the Columbia Basin. It is, therefore, unlikely that regular losses due to scald infection will occur. In these production environments resistance would serve as an insurance against irregular epiphytotics.

Genotypes considered susceptible were significantly higher yielding than putatively resistant genotypes under all inoculation treatments, including fungicide-protection. The higher yield of susceptible genotypes is not attributed to a cost associated with resistance alleles, but rather to the selection criteria

applied during elite line development. The 12 genotypes evaluated in this study were selected during six years of multiple-environment evaluation. Two-row genotypes, although generally more susceptible to scald than six-row genotypes, are frequently selected because of their adaptation and quality characteristics in more arid environments.

Even under the light disease pressure of the Pendleton environments, susceptible genotypes gave higher yields in fungicide-protected than in inoculated plots. This may be due to a yield-increasing effect of Tilt, as reported by Johnston and MacLeod (1987). Scald lesions were observed on third and lower leaves of resistant and susceptible genotypes in inoculated plots.

Resistant and susceptible lines did not differ for disease severity in 1986-87, but in 1987-88 scald severities on third leaves were significantly greater in the susceptible lines. However, the low severities on first and second leaves resulted in low mean severity values.

CORVALLIS EXPERIMENTS

Corvallis was a highly favorable environment for scald development during the two years of the study. Under all inoculation treatments, epidemic development followed a similar pattern for both susceptible and resistant genotypes. In contrast to James et al. (1968), who recorded the highest scald severity on first (flag) leaves at G.S 11.2 (Feekes scale, after Large 1954), the highest scald severity in our experiments was observed on third leaves. Progressively less severity was seen on second and flag leaves. A similar pattern of scald development recorded at G.S. 11.1 (Feekes scale, after Large, 1954) was reported by Khan and D'Antuono (1985).

In all inoculation treatments, disease severities for any given leaf were consistently higher in susceptible than in resistant genotypes (Table 1). In general, disease progression on resistant genotypes was slower than on susceptible genotypes (data are not shown). In 1987-88, the highest percent scald incidences on third leaves at G.S. 10.2 were 36.8% and 23.3% in susceptible and resistant lines, respectively (Table 1). These severities are substantially lower than those reported by Schaller (1951), but comparable to those reported by James et al.(1968).

INOCULATION treatments. Tilt effectively controlled scald in both years. In 1986-87 both susceptible and resistant genotypes treated with Tilt had a low disease incidence, with an average severity for the three uppermost leaves of 2.36% and 1.18%, respectively. Susceptible genotypes had higher severities on second and third leaves, and higher mean severities (Table 1). In the second-year, mean severity readings for susceptible and resistant genotypes were 3.03 and 0.62%, respectively. The second and third leaf severities of susceptible lines were higher than those of resistant lines as well (Table 1).

Although the mean difference in disease severity between resistant and susceptible genotypes was not significant in fungicide-protected plots, resistant genotypes did have a lower incidence of scald on lower leaves. This potential for greater photosynthetic activity may, in part, account for the superior yield performance of resistant genotypes in environments favoring scald development.

In both years, resistant genotypes had significantly lower disease severity readings than susceptible genotypes for each inoculation method. Although

natural infection was less effective than straw and spore inoculations for susceptible genotypes, all three inoculation techniques - natural infection, spore inoculation, and straw inoculation - were equally effective in generating disease in resistant genotypes in 1986-87. Natural infection, spore inoculation, and straw inoculation were equally effective for both resistant and susceptible genotypes in 1987-88 (Table 1).

YIELD losses. Disease severity had a significant effect on yield at Corvallis during both years of the experiment. In the first year, mean yields of both susceptible and resistant lines in protected, natural inoculum, straw inoculated and spore inoculated plots were 5.39, 2.99, and 2.93 t ha⁻¹, respectively. The maximum yield reduction, expressed as a percentage of protected plot yield, was 46% in spore-inoculated plots, with an average disease severity of 14.7%. In the second season, mean grain yields were 7.00, 5.11, 5.30, and 5.30 t ha-1, respectively. A maximum yield reduction, expressed as a percentage of protected plot yield, of 27% was observed in natural infection treatments. The lower yield loss in the second year may have resulted from environmental conditions favoring yield potential expression over scald development. Lower average temperatures were recorded during the disease development season (March through May) in 1987-88 than in 1986-87. In both years, the average precipitation during the same period was comparable. Genetic resistance was effective in reducing scald severity and presumably increasing yield. Yields of resistant and susceptible lines in protected plots were statistically different in 1986-87 and 1987-88 (Tables 2 and 3). Susceptible lines, in 1986-87, showed a maximum loss of 51% in straw inoculated plots, at a

disease severity of 19.8%. The maximum yield loss observed in resistant genotypes was 42% at a scald severity of 10.4%. In the second year, the highest yield loss in susceptible genotypes was 33%, again in straw inoculated plots, with an average severity of 16.4%. A maximum yield reduction of 25% was observed in resistant genotypes, at a 5.3% disease severity in spore-inoculated plots.

Scald severity increases in inoculated plots, expressed as a percentage of protected plots, resulted in lower yield reductions in resistant lines than in susceptible ones. In the first year, a 100% increase in disease severity in straw inoculated plots relative to protected plots reduced yield by 4% with resistant genotypes and reduced yield by 7% with susceptible genotypes (Tables 1, 2 and 3). The regression of scald severity on grain yield was highly significant for both susceptible and resistant genotypes, with R² values of 0.62 and 0.50. respectively. In the second year, a 100% increase in disease severity in straw inoculated plots relative to protected plots gave a 2% reduction in yield for resistant genotypes and a 6% yield-reduction for susceptible genotypes (Tables 1, 2 and 3). R² values were 0.53 and 0.33 for susceptible and resistant genotypes, respectively.

Four susceptible lines were two-row and two susceptible lines were six-row. Five of the six-row were resistant and one was susceptible. Unequal distribution of tolerance in six-row and two-row genotypes complicated the identification of principal yield components affected by scald. In general, two-row genotypes have higher kernel weight, more spikes per unit area and fewer kernels per spike than six-row genotypes of comparable yield potential. In the

orthogonal contrast of two- versus six-row genotypes, differences in number of spikes per unit area, number of kernels per spike, hundred kernel weight, and percent of plump and thin kernels were highly significant (P=0.01) in both years. The yield difference between two- and six-row genotypes were not significant (P=0.09) in 1986-87. It was significant (P=0.05) in 1987-88.

James et al. (1968) reported that scald infections significantly reduced hundred kernel weight. While hundred kernel weight of both resistant and susceptible genotypes in treated plots was lower than in protected plots in 1986-87, the difference was significant only for susceptible lines. Under straw inoculation, susceptible lines had higher hundred kernel weight than resistant lines (Table 2). In 1987-88 hundred kernel weights of resistant and susceptible lines within protected plots were statistically different, as expected given spike morphology. However, within inoculation treatments resistant and susceptible lines were not statistically different (P=0.05).

In 1986-87, with susceptible lines, the number of kernels per spike was not significantly different when fungicide protected and inoculation treatments were compared. Resistant genotypes had the lowest number of kernels per spike in fungicide-protected plots (Table 2). In 1987-88, kernels per spike was comparable in all mainplots (Table 3). With susceptible genotypes, the highest number of kernels per spike was recorded in spore-inoculated plots. Inoculation treatments had no effect on the hundred kernel weight of resistant genotypes. Fungicide protection led to significantly higher hundred kernel weights in susceptible genotypes in 1987-88.

Of the yield components - hundred kernel weight, number of kernels per spike, and number of spikes per unit area the latter trait was most affected by scald (Tables 2 and 3). In 1986-87, the number of spikes per unit area for both resistant and susceptible genotypes was significantly lower in inoculated than in protected plots. Within inoculation treatments, the number of spikes per unit area was significantly lower in susceptible lines than in resistant ones. Higher number of spikes per unit area values were expected with two row genotypes. Reduced tillering may account, in large part, for yield reductions in these genotypes.

In both years, susceptible genotypes, predominantly two-row, had higher percent plump kernels than resistant genotypes, and fungicide protection consistently allowed for maximum grain filling (Tables 2 and 3). In 1986-87, even with fungicide protection, the percent plump kernels was lower than minimum malting requirements for both resistant and susceptible genotypes (Table 2). In the second year, percent plump differences were significant when fungicide-protected and inoculation treatments were compared for both resistant and susceptible genotypes. Even with infection, percent plump values for both resistant and susceptible genotypes met minimum standards (Table 3).

CONCLUSIONS

Yield losses attributable to scald varied depending upon environment, year, and the level of genetic resistance of test genotypes. Disease severity was minimal in both years at Pendleton. Protected plots gave higher yields, perhaps due to the control of disease in the lower canopy and/or a yield enhancing effect of Tilt. Putatively susceptible genotypes, predominantly two-row, were higher yielding than resistant genotypes in protected and inoculated plots at Pendleton.

At Corvallis, in general, putative genetic resistance was confirmed.

However, resistant (six-row) genotypes were higher yielding than susceptible (two-row) genotypes in fungicide protected plots, complicating the separation of resistance and adaptation effects.

Severity readings were comparable in both years, but yield losses were lower in the second year. The maximum yield loss, expressed as a percentage of protected plot yield, was 46% in the first year and 27% in the second year. Schaller (1951) observed that the number of spikes per plant was reduced under severe disease pressure, but ascribed yield losses primarily due to reductions in number of kernels per spike. On the other hand, James et al. (1968) reported that 100 kernel weight was most affected by scald severity. In these experiments, the number of kernels per spike was consistent or even increased with scald pressure. Reductions in hundred kernel weight values were not statistically significant. Scald infection had a significant effect on grain filling, as measured by the percentage of plump kernels. The number of spikes per unit area was most affected by scald both in susceptible and resistant

genotypes. Resistant and susceptible genotypes responded differentially to environments, underscoring the importance of genotype x environment interaction in selection for broad adaptation to very diverse environments.

Table 1. Percentage of the area affected by scald on first (flag), second, third leaves, and average severity at Feekes 10.2 growth stage for resistant and susceptible winter barley lines grown under three inoculation treatments and fungicide-protection at Corvallis in 1986-87 and 1987-88.

			IN	OCULATION T	REATMENTS				
<u>YEAR</u>	LEAFNATUR		TURAL STRAW		SF	SPORE		ED	
<u>1986-87</u>		Res	Sus	Res	Sus	Res	Sus	Res	Sus
	First	0.60†	1.46	1.48	3.98	1.40	1.93	0.05	0.46
	Second	8.30	11.80	13.30	18.50	12.66	14.50	1.31	2.11
	Third	22.00	31.16	23.33	36.83	22.21	35.67	2.18	4.53
	Average	10.37	14.80	12.85	19.83	12.02	17.33	1.18	2.36
<u> 1987 - 88</u>									
	First	0.05	3.33	0.28	3.83	0.05	2.21	0.05	0.16
	Second	1.61	12.73	2.50	14.10	1.11	12.26	0.00	0.56
	Third	14.05	40.00	18.71	31.33	14.83	36.00	1.83	8.38
Standard	Average errors : 0.72	5.23	18.69	7.16	16.42	5.33	16.82	0.62	3.03

rd errors: 0.72, 1.86, 2.038, 1.44 for resistant genotypes within the same treatment, 1.02, 1.42, 2.26, 1.62 for susceptible genotypes within same treatment, and 0.36, 0.77, 1.05, 0.77 between resistant and susceptible genotypes for first leaf, second leaf, and third leaf, respectively in 1986-87. In 1987-88, standard errors: 0.40, 1.95, 1.70, 0.99 between resistant genotypes within same treatment, and 1.27, 2.23, 2.85, 1.76 between susceptible genotypes within the same treatment, and 0.37, 0.69, 1.03, 0.71 between resistant and susceptible genotypes. † Percent leaf area affected by scald.

Table 2. Grain yield, hundred kernel weight, number of kernels per spike, number of spikes per meter, and percent plump kernels in a comparison of scald inoculation treatments in resistant and susceptible winter barley genotypes grown at Corvallis in 1986-87.

	Grain yid (t ha -1		6† Hundred kernel%† weight (a)		,	Kernels/ %†		%† Plump kernels 9		s %†
			. —		RESISTA	NT LINE	<u>meter</u> S			
NATURAL	3.30	58	2.78	86	54.17	111	112.83	82	34.33	69
STRAW	3.45	61	2.77	88	54.91	112	117.17	85	34.16	68
SPORE	3.29	58	2.75	88	53.26	109	122.33	89	35.83	72
PROTECTED	5.66	100	3.14	100	48.78	100	137.33	100	50.00	100
LSD (0.05)	0.04	*	0.74		2.56		4.95		4.33	
	· · · · · · · · · · · · · · · · · · ·				SUSCEPT	IBLĘ LIN	IES			· · · · ·
NATURAL	2.68	52	3.50	86	36.81	103	108.88	76	55.00	78
STRAW	2.53	49	3.60	88	36.80	103	123.00	86	51.83	73
SPORE	2.56	50	3.48	86	34.81	97	116.83	82	52.83	75
PROTECTED	5,12	100	4.06	100	35.68	100	143.33	100	70.83	100
LSD (0.05)	0.04		0.60	data o d	4.47	 	6.10		4.68	

LSD (0.05): resistant vs susceptible; grain yield, 0.02; hundred kernel weight, 0.44; kernels/spike, 1.77; spikes/meter, 2.71; percent plump kernels, 2.24.

[†] percent of protected plots.

Table 3. Grain yield, hundred kernel weight, number of kernels per spike, number of spikes per meter, and percent of plump kernels in a comparison of scald inoculation treatments in resistant and susceptible winter barley genotypes grown at Corvallis in 1987-88.

	Grain yi (t ha -1		Hundred kernel weigh		Kernels/ spike	%† 	Spikes/ meter	%† ———	Percent plump kerr	%† <u>nel</u>
	-				RESISTANT I	LINES				
NATURAL	6.70	88	3.88	97	52.61	102	102.16	96	79.33	93
STRAW	5.90	77	3.77	94	52.51	102	112.00	105	73.66	86
SPORE	5.73	75	3.69	92	52.36	102	99.50	93	73.66	86
PROTECTED	7,59	100	3.97	100	51.58	100	106.67	100	85.16	10
LSD (0.05)	0.04	· ·	0.62		2.08	_	8.07		3.29	
					SUSCEPT	IBLE LI	NES			
NATURAL	5.02	78	4.36	96	36.00	103	113.67	92	87.66	95
STRAW	4.32	67	4.54	100	35.58	102	116.17	94	86.16	93
SPORE	4.88	76	4.17	92	39.95	114	109.16	88	85.83	93
PROTECTED	6.42	100	4.54	100	34.90	100	123.17	100	92.33	100
LSD (0.05)	0.04		0.77		3.86		5.78	-	3.17	

LSD (0.05): resistant vs susceptible; grain yield, 0.02; hundred kernel weight, 0.15; kernels/spike, 1.51; spikes/meter, 2.29; percent plump kernels, 2.28.

[†] percent of protected plots.

REFERENCES

- Hartley, H. O. 1955. Some recent developments in analysis of variance. Comm. Pure and Appl. Math. 8: 47-72.
- Jackson, L. F., and R. K. Webster. 1976. The dynamics of a controlled population of <u>Rhynchosporium secalis</u>, changes in race composition and frequencies. Phytopathology 66: 726-728.
- James, C. 1971. A manual of assessment keys for plant diseases. Canada Dept. of Agric. Public. No : 1458.
- James, W. C., J. E. E. Jenkins, and J. L. Jemmet. 1968. The relationship between leaf blotch caused by <u>Rhynchosporium secalis</u> and losses in grain yield of spring barley. Ann. Appl. Biol. 62: 273-88.
- Johnston, H. W., and J. A. MacLeod. 1987. Response of spring barley to fungicides, plant growth regulators, and supplemental nitrogen. Can. J. Plant Pathol. 9: 255-59.
- Khan, T.N. and M.F. D'Antuano. 1985. Relationship between scald (Rhynchosporium secalis) and losses in grain yield of barley in Western Australia.

 Austr. J. Agric. Res. 36: 655-61.
- Khan, T. N., P. A. Portman, and R. McLean. 1984. Evaluating resistance of barley breeding lines to scald in field plots. Euphytica. 33: 897-901.
- Mayfield, A. H., and B. G. Clare. 1985. Effect of high temperatures on scald lesion development and sporulation in barley infected with Rhynchosporium secalis. Austr. J. Agric. Res. 36:197-200.

- McDonald, B. A., R. W. Allard, and R. K. Webster. 1988. Responses of two-,three-, and four-component barley mixtures to a variable pathogen population. Crop Sci. 28: 447-52.
- Nutter, F. W., V. D. Pederson, and A. E. Foster. 1985. Effects of inoculations with <u>Cochliobolus sativus</u> at specific growth stages on grain yield and quality of malting barley. Crop Sci. 25: 933-38.
- Piening, L. J. 1979. A simple method of inoculating barley in the field with Rhynchosporium secalis. Barley News., 23 : 13.
- SAS Institute, Inc. 1982. SAS User's Guide: Statistics. SAS Institute, Inc., Cary, NC.
- Schaller, C. W. 1951. The effect of mildew and scald infection on yield and quality of barley. Agron. J. 43: 183-88.
- Shipton, W. A., W. J. R. Boyd and S. M. Ali. 1974. Scald of barley. Rev. Plant Path. 53: 839-61.
- Skoropad, W. P. 1966. Sporulating potential of <u>Rhynchosporium secalis</u> on naturally infected leaves of barley. Can. J. Plant Sci. 46: 243-47.
- Zadoks, J. C. and R. D. Schein. 1979. Epidemiology and plant disease management. pp. 264-267. Oxford University Press, Inc. USA.
- Zhang, Q., R. K. Webster, R. W. Allard. 1987. Geographical distribution and associations between resistance to four races of Rhynchosporium secalis. Phytopathology 77: 352-357.

Chapter 2

LITERATURE REVIEW

INTRODUCTION

Scald, incited by <u>Rhynchosporium secalis</u> (Oud.) Davis, is a major foliar disease of barley (<u>Hordeum vulgare L.</u>). While scald has been recognized as an economically important disease for the past century, changes in agricultural practices - closer rotations, reduced tillage, and increased fertilizer inputs - are thought to have favored disease development in recent years.

Yield losses attributable to scald epidemics have been quantified in a number of studies. The impact of scald epidemics on yield and malting quality of winter habit barley in the Pacific Northwest, however, is not known.

This research was designed to answer questions regarding the importance of scald in Pacific Northwest malting and feed barley production. The specific objectives of this investigation were to:

- (i) assess yield and quality losses attributable to scald in diverse environments of the Pacific Northwest,
- (ii) test effectiveness of resistance in putatively tolerant elite winter barley lines, and
 - (iii) identify an efficient inoculation technique.

Plant disease is the consequence of complex interactions involving pathogen, host, and environment. Components of the disease triangle are addressed in turn, as they relate to barley scald incited by <u>Rhynchosporium</u> secalis.

THE PATHOGEN

In 1897, Oudemans first described the pathogen inciting barley scald, naming it Marsonia secalis (Oudem). The fungus was subsequently isolated in Germany and placed in a new genus, Rhyhncosporium, on account of the beaked one-septate spores. Formal descriptions were provided later, and the fungus was reclassified by Davis as R. secalis (Oudem) J. J. Davis. Further generic description was provided by Caldwell (1937).

Caldwell (1937) described and classified the fungus as follows: "parasitic, producing spots on leaves; sterile mycelium sparse in mesophile of host; mycelium subcuticular at first, later developing into a superficial fertile stroma more or less covering the leaf spot; conidiophore absent, hyaline, sessile on cells of fertile stroma. The genus, Rhynchosporium takes place in the classification, Moniliaceae Hyalodymae, Micronemeae of the imperfect fungi. The conidia of several isolates from host and culture appear to be relatively uniform in shape and size. The average conidia length and diameter are 15.8 and 3.3 microns respectively. One-celled conidia without septum formation have

only one nucleus while two celled spores have a nucleus in cell. Heterocaryotic phenomena might not apply to <u>R</u>. <u>secalis</u> because it is uninucleate".

Rhynchosporium secalis incited barley disease is commonly referred to as Rhynchosporium scald or simply scald (Shipton et al.,1974). Scald affects leaf lamina, sheaths, and auricles (Ali and Boyd, 1974; Brooks, 1928). Bluishgrey initial lesions with a water-soaked appearance change to light grey-grayish white with a dark brown edge as infection progresses. Eventually, centers of lesions dry out. Lesions can develop separately, coalesce, or develop progressively along the leaf (Ali, 1972). Symptoms under greenhouse and field conditions differ. No definite lesions occur after inoculation under greenhouse conditions, except for a light gray-green discoloration along the leaf margin. Shortly afterward tissue collapses and dries (Harrabi, 1982).

Principle sources of primary inoculum of <u>R</u>. <u>secalis</u> are infected plant debris (Caldwell,1937; Ayesu-offei and Carter,1971), mycelium in the pericarp and/or hull of infected seeds (Reed, 1957), and infected seed itself (Skoropad,1959). The relative importance of sources of primary inoculum is a matter of some debate. Rain splash is the main source for secondary inoculum dispersal of scald (Ayesu-offei and Clare, 1970).

Under cool, moist conditions conidia are produced on the superficial stromata present on the infected debris. The fungus retains sporulating ability for at least one year (Skoropad, 1966). Conidia germinate from one or both cells within 4-12 hours in available water at 15-20 °C. More than one germ tube

may arise from either cell and branch simply (Ayesu-offei, 1971). Germ tubes may continue to elongate but some of them develop appressoria. Appressoria may be either at the tips of germ tubes or sessile on the conidia.

Penetration can occur either directly or through stomata (Shipton et al.,1974) in the absence of appressorial formation and ends within 24 hours (Ayesu-offei and Clare, 1970). Infection hyphae development follows the initial cuticle penetration caused by possible enzymatic degradation of the cuticle. Subcuticular mycelium then grows between epidermal cells and branches profusely (Shipton et al.,1974). Infection increases as stomata open in the light and hyphae penetrate the epidermal cell layer. The collapse of the epidermis is preceded by marked thickening of the cell wall.

Cell disruption may be caused by a toxin, Rhynchosporoside, one of the 1-0-alpha cellobiosides of 1,2 proanediol (Beltran and Strobel, 1980). The fungus may increase the permeability of host cells and use the nutrients concentrated in this free space (Jones and Ayres, 1972). The production of toxic metabolites by R. secalis has been reported (Ayesu-offei and Clare, 1970; and Beltran and Strobel, 1980).

Either mutation or a parasexual cycle may be sources of genetic variation in the fungus. Jackson and Webster (1976) reported that final single spore isolates represented a greater degree of pathogenic variability than had existed in five original inoculum mixtures. Ali (1972) speculated on the presence of a sexual cycle because of the presence of microconidia.

Many authors have demonstrated the presence of races in <u>R</u>. <u>secalis</u> (Shipton et al.,1974; Jackson and Webster,1976). Sarasola and Campi (1947) were to first to identify scald races. Later, these authors demonstrated that <u>Rhynchosporium</u> isolates from barley also attack four grass species in four genera. Isolates specialized in their ability to attack barley varieties as well. Riddle and Suneson (1948), however, could not detect physiological races.

Schein (1960) designated seven races as U.S.1 to U.S.7 while working with eight isolates from different parts of the U.S.A. Two additional races, U.S.8 and U.S.9, were identified by Dyck and Schaller (1961), using two more differentials in addition Schein's original set. Ten races (J1 to J10) in Japan (Kajiwara and Iwata,1963) have been identified. While Reed (1959) reported marked variation in pathogenicity and distinct pathogenic biotypes, Skoropad (1960) found no clear evidence of pathogenic races in Canada. Distinct race spectra have been reported in different countries using the same differentials (Owen,1963; Ceoloni,1980; and Jackson and Webster,1976). Perhaps a limited range of host genotypes and isolates, along with a failure to distinguish between resistance to penetration and resistance to colonization have complicated analysis of the race picture (Shipton et al.,1974).

Host genotype, pathogen genotype, and environment as well as substrate composition and culture age can influence scald virulence (Shipton et al., 1974). The optimum temperature for disease development is 18-21 °C

(Bartels, 1928). Infection does not require direct moisture, but the atmosphere must be humid enough for tissue to absorb moisture (Skoropad, 1966).

Conidia germinate at relative humidities above 92%, but often fail to germinate under continuous optimal conditions. Conidial germination failed in lesions due to the presence of self inhibitors at low spore concentrations but it did not when spore concentration was high (120,000 spores/cm³). At high concentrations, there is apparently feedback inhibition of inhibitor (Ayres and Owen,1970).

Lesion development is fast when inoculated plants are held at 15-18 °C before placing them at 24 °C. Slow development and sporulation of the fungus at low temperatures may allow for initial infection and lesion development during early and late spring when plant growth is restricted (Caldwell,1937). Ali (1972) has shown that temperature affects symptom expression depending on the combination of isolate and host genotype. High (18-30 °C) and low (12-20 °C) diurnal temperature regimes altered the virulence of certain isolates. Genotype resistance reactions varied depending on temperature.

The age of host tissue affects reaction. The leaves of susceptible genotypes become more susceptible with age, although host age affected symptom expression only in certain genotypes. Certain genotypes remained resistant throughout their life cycle. Greater symptom expression occurred when the host permitted restricted lesion development at earlier growth stages

(Ozoe,1956). Spore concentration X genotype interactions have been observed as well (Ali,1972).

Fertility can affect resistance expression. Excess mineral nitrogen results in higher and/or enhanced levels of susceptibility with certain genotypes (Gambit, 1967, reported by Shipton et al., 1974).

SOURCES OF HOST RESISTANCE

Resistant cultivars may reduce yield losses incited by <u>R</u>. <u>secalis</u>. Breeding for scald resistance requires an understanding of the genetic control of resistance.

Early studies of the inheritance of scald resistance showed that resistance was controlled by a single recessive gene (Shipton et al., 1974). Riddle and Brigs (1950) identified a single dominant gene for resistance in cvs. La Mesita, Trebi, and Modoc (California No.1311) and two genes in Turk (C.I.5611-2), one of which was allelic to that common in La Mesita, Trebi, and Modoc. Bryner (1957) reported that resistance in cv. Brier (C.V.7157) was conditioned by a single dominant gene.

The earliest extensive work on scald genetics showed that there are five resistance genes, Rh2, Rh3, Rh4, Rh4² (an allele of Rh4), and Rh5. A single dominant gene, RH2 was also found in Atlas, whereas Atlas46, a derivative of Atlas and Turk, has a second gene (Dyck and Schaller, 1961). Cv. Turk has

been reported to have either two dominant genes, Rh3 and Rh5, (Riddle and Suneson, 1948) or one gene (Starling et al.,1971). Ali (1975) reported the single gene in cv. Turk is allelic or closely linked to that of cv. La Mesita. In other genotypes, recessive resistance genes rh6, rh7, rh8, and rh9 were reported by Habgood and Hayes (1970).

Shipton et al. (1974) speculated that few genes govern resistance to scald. Variability in penetrance and expressivity due to environment and genetic background of material, variations in the pathogenic characteristics of fungal isolates and in their aggressiveness, suggest that resolution of the genetic basis for resistance <u>R. secalis</u> is far from complete.

YIELD LOSSES

Yield loss estimates due to disease may assist in allocation of resources to research. Yield losses due to disease can be estimated in field experiments (James et al.,1968) or using non-plot methods (single tillers or half-field) (Richardson, 1981). Simple or multiple regression analyses are carried out to examine the relationship between disease and grain yield.

Two approaches to measuring disease-related yield losses in field experiments are 1) isogeneic lines 2) fungicide-protection. An example of the former approach is the work of Schaller (1951), who compared yield differences between isogeneic lines differing only for scald resistance. By this method,

scald related yield loss was estimated as 22.3%. Reductions were due primarily to lower kernel number and reduced kernel weight. Yield losses were not related to specific levels of disease, but scald affected 75% of the leaf area in the susceptible isogeneic line.

The work of James et al. (1968) exemplifies the fungicide-protected method of disease loss assessment. Yields of resistant and susceptible genotypes were compared in fungicide-protected and inoculated plots. A consistent linear relationship between disease on the upper two leaves and yield loss at growth stage 11.1 (Feekes scale, after Large,1954) was found. In the first year of this experiment, a susceptible cultivar, Cambrinus, suffered a reduction of 62% in yield when the flag-leaf lamina was 100% infected by scald. In the second year, the estimated yield loss was 46% and 40% when 100% infection occurred on the flag and second leaf, respectively. The principal yield component affected was hundred kernel weight.

ARTIFICIAL INOCULATION

Stable and adequate amounts of inoculum, representing the full spectrum of virulence, are necessary to study the resistance to any given disease.

Practical inoculation techniques for field studies are 1) infected straw (Khan et al.,1984), 2) spore inoculation, using spores obtained from fresh leaves of volunteer plants in the spring (Piening,1979), and 3) spores obtained through aseptic culture of the fungus.

Some commonly used synthetic media for fungus culture are: 1) lima bean agar to isolate and increase inoculum (Harrabi, 1982), and 2) water agar to isolate and potato dextrose agar to increase inoculum (Jackson and Webster, 1976).

Schein (1960) studied the growth of scald on 16 different media and found optimum growth occurred on rice polish media. Growth on potato dextrose agar was almost comparable. However, optimum sporulation was found on lima bean agar, where growth was poor. Subsequently, he concluded that potato dextrose is the best medium for general work, where both good growth and sporulation are required.

Different amounts of spores per volume of inoculating solution (50,000 to 6X10⁶ ml⁻¹) have been used (Zhang et al.,1987; and Jackson and Webster, 1976; Harrabi, 1982), apparently with equal effectiveness.

DISEASE CONTROL

Methods of disease control are provision of barriers to colonization (horizontal and vertical resistance), sanitation, and fungicide-protection (Shipton et al., 1974).

Durable resistance to a pathogen with diverse arrays of virulence genes, such as scald, may be difficult to achieve (McDonald et al.,1988). Cultivar mixtures, multilines (Frey et al.,1977; Marshall, 1979), composite crosses and/or

male-sterile-facilitated recurrent selection schemes (Barnes et al., 1971) may be useful in breeding for resistance to scald.

McDonald et al., (1988) reported that the largest reductions in scald incidence were obtained with mixtures of susceptible lines. Genes for resistance deployed singly in mixtures and resistance genes pyramided into single lines did not differ in scald incidence reduction. Cultivar mixtures may offer an effective strategy for scald control, particularly in feed barley production, where cultivar identity is not critical.

Recurrent selection is an effective breeding method for accumulating genes and developing multigenic resistance (Barnes et al., 1971). The use of recurrent selection is restricted in self-pollinated crops because of difficulties in recombination and evaluation. In barley, genetic male sterility can facilitate recombination. A number of male sterile genes have been identified in barley (Hockett and Eslick, 1968). Harrabi (1982) hypothesized that multigenic resistance incorporated by recurrent selection could provide durable resistance to scald. However, after four cycles of recurrent selection for resistance to three scald isolates he did not detect a significant increase in resistance and concluded that it may be difficult to incorporate multiple resistance alleles in a single cultivar via male sterile facilitated recurrent selection. Ramage (1981) speculated that selection for agronomic characters should be practiced before selection for disease resistance in a modified recurrent selection scheme.

Cultural practices - rotations with other crops, burning stubble, deep plowing, late planting, and destruction of volunteer barley plants and grasses - may reduce sources of primary inoculum (Shipton et al., 1974). Wide spacing between rows (Bartels,1928) and balanced fertilizers without excess of nitrogen (Ozoe,1956) may reduce scald incidence by developing a less favorable microclimate for disease development as well. Fungicides, Tilt (Propiconazole, 42% EC); Bayleton (Triadimfon, 50%, WP) (Mayfield and Clare,1985); and Dithane A40 (Nabam) (Skoropad, 1960) effectively control scald. Widespread use in field production may not be economically justified (James et al., 1968). Tilt (Propiconazole, 42% EC) has a differential yield enhancement effect, depending on genotype and may lead to higher protein (Johnston and MacLeod, 1987).

BIBLIOGRAPHY

- Ali, S. M. 1972. Studies in the breeding of scald (Rhynchosporium secalis) resistance in barley (Hordeum vulgare). Ph. D. Thesis, Uni. Western Australia.
- Ali, S. M. 1975. Inheritance of scald resistance in barley. Aust. J. Agr. Res. 26: 251-257.
- Ayesu-offei, E. N. 1971. Leaf scald of barley. Ph.D. Thesis, Uni. of Adelaide.
- Ayesu-offei, E. N. and B. G. Clare. 1970. Processes in the infection of barley leaves by <u>Rhynchosporium secalis</u>. Aust. J. Bio. Sci. 23: 299-307.
- Ayesu-offei, E.N. and M.V. Carter. 1971. Epidemiology of leaf scald of barley.

 Aust. J. Agr. Res., 22: 383-90.
- Ayres, P. G. and H. Owen. 1970. Factors influencing spore germination in Rhynchosporium secalis. Trans. Brit. Myco. Soc. 54: 389-394.
- Barnes, D. K., C. H. Hanson, F. I. Frosheiser and L. J. Elling. 1971. Recurrent selection for bacterial wilt resistance in alfalfa. Crop Sci. 11: 545-546.
- Bartels, F. 1928. Studies on Marsonia graminocola. Plant Dis. (Germany) 5:73.
- Beltran, J. P. and G. A. Strobel. 1980. Rhynchosporoside binding patterns of barley. FEDS letters. 96: 34-35.

- Brooks, F. T. 1928. Observations on <u>Rhynchosporium secalis</u> (Oud.) Davis, leaf blotch of barley and rye. New Phyt. 27: 215-218.
- Bryner, C. S. 1957. Inheritance of scald resistance in barley. Ph. D. Thesis (Diss. Abs. 17: 2752).
- Burleigh, J. R., M. Tajani, and M. Seck. 1988. Effects of <u>Pyrenophora teres</u> and weeds on barley yield and yield components. Phytopathology 78: 295-99.
- Caldwell, R. M. 1937. Rhynchosporium scald of barley, rye and other grasses. J. Agr. Res. 55: 175-198.
- Ceoloni, C. 1980. Race differentiation and search for sources of resistance to Rhynchosporium secalis in barley in Italy. Euphytica. 29: 547-553.
- Dyck, P. L. and C. W. Schaller. 1961. Inheritance of resistance in barley to several physiological races of the scald fungus. Can. J. Gen. Cytol. 3: 153-164.
- Frey, K. J., J. A. Browning, and M. D. Simons. 1977. Management systems for host genes to control disease loss. Ann. N. Y. Acad. Sci. 287: 255-274.
- Habgood, R. M. and J. D. Hayes. 1970. The inheritance of resistance to Rhynchosporium secalis in barley. Welsh Plant Breeding Station. Wales.
- Harrabi, M. M. 1982. The inheritance of resistance of barley (<u>Hordeum vulgare</u>) to <u>Rhynchosporium secalis</u>. Ph. D. Thesis, Montana State Uni., Bozeman, Montana.

- Hartley, H.O. 1955. Some recent developments in analysis of variance. Comm.

 Pure and Appl. Math. 8:47-72.
- Hocket, E. A. and R. F. Eslick. 1968. Genetic male sterility in barley II. Available spring and winter stocks. Crop Sci. 8: 754-755.
- Jackson, L. F., and R. K. Webster. 1976. The dynamics of a controlled population of <u>Rhynchosporium secalis</u>, changes in race composition and frequencies. Phytopathology 66: 726-728.
- James, C. 1971. A manual of assessment keys for plant diseases. Canada Dept. of Agric. Pulbic. No : 1458.
- James, W. C., J. E. E. Jenkins, and J. L. Jemmet. 1968. The relationship between leaf blotch caused by <u>Rhynchosporium secalis</u> and losses in grain yield of spring barley. Ann. Appl. Biol. 62: 273-88.
- James, W. C. and P. S. Teng. 1979. The quantification of production constraints associated with plant disease. In Applied Biology. Ed. T. Cooker. Vol. 4, pp. 201-67. Academy Press: London.
- Johnston, H. W., and J. A. MacLeod. 1987. Response of spring barley to fungicides, plant growth regulators, and supplemental nitrogen. Can. J. Plant Pathol. 9: 255-59.
- Jones, P. and P. G. Ayres. 1972. The nutrition of subcuticular mycelium of Rhynchosporium secalis (barley leaf blotch): Permeability changes induced in the host. Phys. Plant Path. 2: 383-392.

- Kajiwara, J. and Y. Iwata. 1963. Studies on the strains of the barley scald fungus

 Rhynchosporium secalis (Oud.) Davis. Bull. Nat. Inst. Agr. Sci. Tokyo. 15

 : 1-73.
- Khan, T.N. and M.F. D'Antuano. 1985. Relationship between scald

 (Rhynchosporium secalis) and losses in grain yield of barley in Western

 Australia. Austr. J. Agric. Res. 36: 655-61.
- Khan, T.N., P.A. Portmann and R. Mclean. 1984. Evaluating resistance of barley breeding lines to scald in field plots. Euphytica 33: 897-901.
- Large, E. C. 1954. Growth stages in cereals: Illustration of the Feekes scale.

 Plant Pathology. 3: 128-29.
- Marshall, D.R. 1979. Multiline varieties and disease control. II. The dirty crop approach with components carrying two or more genes for resistance. Euphytica 28: 145-159.
- Mayfield, A.H. and B.G. Clare. 1985. Effects of high temperatures on scald lesion development and sporulation in barley infected with Rhynchosporium-secalis. Aust. J. Agr. Res. 36: 197-200.
- McDonald, B. A., R. W. Allard, and R. K. Webster. 1988. Responses of two-, three-, and four-component barley mixtures to a variable pathogen population. Crop Sci. 28: 447-52.
- Nutter, F.W., V.D. Pederson, and A.E. Foster. 1985. Effects of inoculations with <u>Cochliobolus sativus</u> at specific growth stages on grain yield and quality of malting barley. Crop. Sci. 25: 933-38.

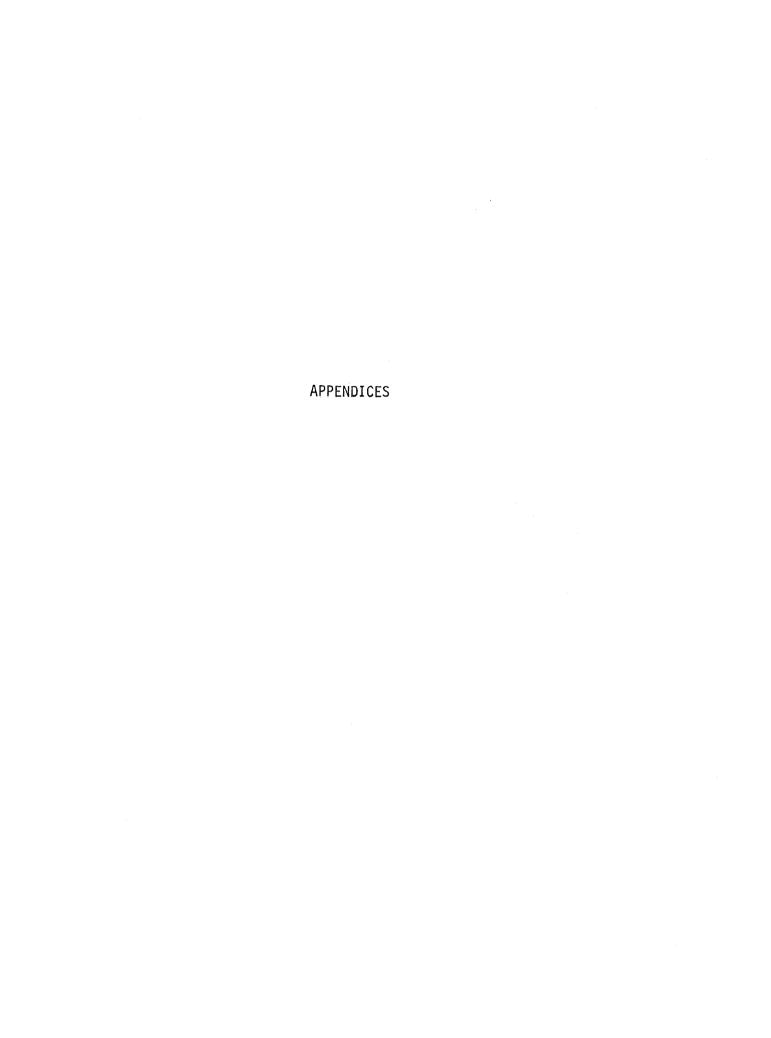
- Owen, H. 1963. Physiologic races of <u>Rhynchosporium secalis</u> on cultivated barley. Trans. Brit. Myco. Society. 46: 604-608.
- Ozoe, S. 1956. Studies on the Rhynchosporium scald of barley and its control.

 Bull. Shimane Agri. Coll. 1: 1-122.
- Piening, L. J. 1979. A simple method of inoculating barley in the field with Rhynchosporium_secalis. Barley News., 23, p.13.
- Ramage, R. T. 1981. Comments about the use of male sterile facilitated recurrent selection. Barley News. 24: 52-53.
- Reed, H. R. 1957. Studies on barley scald. Uni. of Tennessee Agr. Exp. Sta. Bull. 2, p. 43.
- Reed, H. R. 1959. Studies on barley scald. Uni. of Tenn. Agr. Exp. Stn. Bull. 268.
- Richardson, M. J. 1981. The determination of yield relationship by non-plot methods. In Crop Loss Assessment Methods. Ed. L. Chiarappa. Suppl. 3, FAO/Common. Agr. Bur., Slough, U.K., p.p. 59-63.
- Riddle, O.C. and F.N. Briggs. 1950. Inheritance of resistance to scald in barley.

 Hilgardia. 20: 18-27.
- Riddle, O. C. and C. A. Suneson. 1948. Sources and use of scald resistance in barley. Agron. J. 40: 926-928.
- Sarasola, J. A. and M. D. Campi. 1947. Reactions of some barley to

 <u>Rhynchosporium secalis</u> in Argentina. Rev. Agr. Res. 1: 243-260.
- SAS Institute, Inc. 1982. SAS User's Guide: Statistics. SAS Inst., Inc. Cary, NC.

- Schaller, C. W. 1951. The effect of mildew and scald infection on yield and quality of barley. Agr. J. 43: 183-88.
- Schein, R. D. 1960. Physiologic and pathogenic Specialization of Rhynchosporium-secalis. Penn. Agr. Exp. Sta. Bull. 664.
- Shipton, W.A., W.J.R. Boyd and S.M. Ali. 1974. Scald of barley. Rev. Plant Pathology 53: 839-861.
- Skoropad, W. P. 1966. Sporulating potential of <u>Rhynchosporium secalis</u> on naturally infected leaves of barley. Can. J. Plant Sci. 46: 243-47.
- Skoropad, W. P. 1960. Barley scald in the prairie provinces of Canada. Comm. Phytopathology News. 6: 25-27.
- Skoropad, W. P. 1959. Seed and seedling infection of barley by <u>Rhynchosporium secalis</u>. Phytopathology 49: 623-626.
- Starling, T. M., C. W. Roane, and Kuo-Ruey Chi. 1971. Inheritance of reaction to Rhynchosporium secalis in winter barley. Int. Barley Gent. Symp. Pullman, Washington. pp. 513-519.
- Teng, P. S., and R. E. Gaunt. 1980. Modelling systems of disease and yield loss in cereals. Agr. Sys. 6: 131-54.
- Zadoks, J. C., and Schein R. D. 1979. Epidemiology and plant disease management. pp.264-267. Oxford University Press, Inc. USA.
- Zhang, Q., Webster, R. K. and Allard, R. W. 1987. Geographical distribution and associations between resistance to four races of <u>Rhynchosporium</u> secalis. Phytopathology 77: 352-357.



APPENDIX 1

SCALD DEVELOPMENT ON RESISTANT AND SUSCEPTIBLE GENOTYPES.

In 1986-87, scald severity on first (flag), second, and third leaves was estimated at the 10.3 growth stage (Feekes' scale, after Large, 1954) in Pendleton and at the 10.2 and 10.5 growth stages in Corvallis. In 1987-88, scald severity was estimated at the 10.5 growth stage in Pendleton and the 10.2, 10.5, and 11.2 growth stages in Corvallis.

In both years, at G.S.10.5, resistant and susceptible genotypes in protected plots had the lowest scald incidence on all three leaves. Susceptible genotypes had higher scald severities than resistant genotypes in all inoculated plots including the fungicide-protected plots. The average scald severities at G. S. 10.5 were 17.12-20.66% in resistant genotypes and 25.03-30.00% in susceptible genotypes in 1986-87. In general, inoculation techniques were equally effective in generating epidemics in both susceptible and resistant genotypes (Appendix 1, table 1).

In 1987-88, average scald severities at G. S. 10.5 were 9.65-12.00% in resistant genotypes and 26.67-28.66% in susceptible genotypes. Again, inoculation treatments were in general, equally effective (Appendix 1, table 3). In Corvallis 1987-88, average scald severity at G. S. 11.2 was 17.83-25.27% in resistant genotypes and 30.67-33.67% in susceptible genotypes. Straw

inoculation gave the highest scald severities in both resistant and susceptible genotypes (Appendix 1, table 2).

Scald epidemic development continued until senescence at Corvallis in both years. However, infection was slower in fungicide-protected plots and on resistant genotypes in all main plots. Third leaves had the highest scald incidence and those were followed by second and first (flag) leaves at both years (Appendix 1, tables 1 and 2).

Appendix 1, table 1. Percentage of leaf area destroyed by scald on first (flag), second, and third leaves, and average severities at Feekes 10.5. Values for resistant and susceptible winter barley lines grown under three inoculation treatments and in a fungicide-protected check at Corvallis in 1986-87 and 1987-88.

		INOCULATION TREATMENT							
<u>YEARS</u>	<u>LEAF</u>	NATURAL		STRAW		SPORE		PROTECTED	
					% SCALD SEVERITY				
<u>1986-87</u>		Res	Sus	Res	Sus	Res	Sus	Res	Sus
	First	8.82 †	11.60	8.33	14.48	5.72	14.40	0.47	2.06
	Second	19.00	19.16	19.63	26.00	14.17	27.16	1.60	4.05
	Third	34.00	44.33	32.33	47.83	31.47	48.45	3.00	6.23
	Average	20.66	25.03	20.09	29.44	<u>17.12</u>	30.00	1.67	4.11
<u>1987-88</u>									
	First	2.67	9.85	1.88	8.73	3.00	8.76	0.16	1.08
	Second	7.52	21.05	8.35	25.83	10.83	22.03	0.68	2.35
	Third	18.83	49.33	23.67	52.16	22.16	48.50	2.21	14.66
	Average	9.65	26.67	11.37	28.66	12.00	26.67	1.02	6.03

Standard errors: 1.87, 2.55, 2.19, 1.97 for resistant genotypes within the same treatment, 1.94, 2.14, 2.04, 1.88 for susceptible genotypes within same treatment, and 0.72, 0.83, 0.84, 0.75 between resistant and susceptible genotypes for first leaf, second leaf, and third leaf, respectively in 1986-87. In 1987-88, standard errors 0.95, 1.97, 2.14, 1.69 between resistant genotypes within same treatment, and 1.47, 2.56, 2.88, 2.30 between susceptible genotypes within the same treatment, and 0.53, 0.93, 1.14, 0.88 between resistant and susceptible genotypes. † Percent leaf area covered by scald.

Appendix 1, table 2. Percentage of leaf area destroyed by scald on first (flag), second, and third leaves and average severities at Feekes 11.2. Values for resistant and susceptible winter barley lines grown under three inoculation and in a fungicide protected check at Corvallis in 1987-88.

	INOCULATION TREATMENTS								
YEARS	<u>NATURAL</u>		STRAW		SPORE		PROTECTED		
	% SCALD SEVERITY								
<u>1987-88</u>	Res	Sus	Res	Sus	Res	Sus	Res	Sus	
First	12.78 †	21.83	18.98	25.33	13.72	21.50	1.68	2.85	
Second	17.33	25.66	21.50	31.83	16.77	28.50	1.76	3.73	
Third	23.00	44.50	34.83	44.00	26.17	47.00	2.83	14.45	
Average	17.83	30.67	25.17	33.66	18.95	32.33	2.10	6.98	

Standard errors: 2.22, 2.50, 2.47, 2.25 for resistant genotypes within the same treatment, 2.29, 2.28, 2.12, 2.23 for susceptible genotypes within same treatment, and 0.87, 0.90, 0.88, 0.87 between resistant and susceptible genotypes for first leaf, second leaf, and third leaf, respectively in 1987-88.

† Percent leaf area covered by scald.

APPENDIX 2

EFFECT of SCALD SEVERITY on BIOLOGICAL YIELD, HARVEST INDEX, HEADING DATE, and PLANT HEIGHT.

In 1986-87, inoculation of both resistant and susceptible genotypes resulted in lower harvest index and biological yield when compared with fungicide-protected plots (Appendix 2, table 1). Within susceptible and resistant genotypes, heading date did not differ. However, resistant genotypes were significantly later in days to heading than susceptible genotypes. Plant height in inoculation treatments, in general, was not different from that in fungicide-protected plots; but it decreased in inoculation treatments in susceptible genotypes. Overall, susceptible and resistant genotypes were of comparable height.

In 1987-88, inoculation of both resistant and susceptible genotypes did not give lower harvest index, but did reduce biological yield when compared with fungicide-protected plots (Appendix 2, table 2). Within susceptible and resistant genotypes, heading date did not differ. However, resistant genotypes were significantly later in heading than susceptible genotypes. Expression of plant height was comparable to the first year. However, within susceptible genotypes, fungicide-protected plots were not consistently taller than inoculated plots. Overall, resistant genotypes were taller.

Appendix 2, table 1. Harvest index, biological yield, days to heading (April 1 = 1), and plant height in a comparison of scald inoculation treatments in resistant and susceptible winter barley genotypes grown at Corvallis in 1986-87.

	Harvest Index	% †	Biological <u>yield</u>	%† RESISTA	Days to <u>heading</u> ANT LINES	% †	Plant <u>height</u>	% †
NATURAL	0.19	79	253.17	75	35.00	103	109.33	101
STRAW	0.18	75	292.50	86	35.83	106	105.66	98
SPORE	0.20	83	241.83	71	34.50	102	108.67	101
PROTECTED	0.24	100	339.00	100	33.83	100	107.67	100
LSD (0.05)	0.03		11.24		2.82		3.84	
	SUSCEPTIBLE LINES							
NATURAL	0.19	76	181.83	55	27.17	112	104.00	92
STRAW	0.16	64	180.17	54	26.50	109	103.67	92
SPORE	0.17	68	179.33	54	26.66	110	107.50	95
PROTECTED	0.25	100	329.83	100	24.33	100	113.00	100
LSD (0.05)	0.03		8.22		3.70	_	3.31	

LSD (0.05): resistant vs susceptible; harvest index 0.12; biological yield, 4.43; days to heading, 1.12; plant height, 1.25.

[†] percent of protected plots.

Appendix 2, table 2. Harvest index, biological yield, days to heading (April 1 = 1), and plant height in a comparison of scald inoculation treatments in resistant and susceptible winter barley genotypes grown at Corvallis in 1987-88.

	Harvest <u>Index</u>	% †	Biological <u>yield</u>	%† <u>RESIST</u>	Days to heading ANT LINES	% †	Plant <u>height</u>	% †
NATURAL	0.36	97	323.00	88	38.00	100	117.33	101
STRAW	0.36	97	299.33	82	38.00	100	119.00	102
SPORE	0.37	100	312.33	86	37.67	99	118.67	102
PROTECTED	0.37	100	365.00	100	38.00_	100	116.67	100
LSD (0.05)	0.03		11.74		2.89	•	3.82	
		SUSCEPTIBLE LINES						
NATURAL	0.32	84	289.66	93	33.00	101	106.33	106
STRAW	0.36	95	246.17	79	32.83	100	111.33	101
SPORE	0.37	97	249.67	80	33.33	102	109.17	99
PROTECTED	0.38	100	311.17	100	32.83	100	110.33	100
LSD (0.05)	0.03		11.22		3.10		5.26	

LSD (0.05): resistant vs susceptible; harvest index 0.11; biological yield, 4.19; days to heading, 1.00; plant height, 2.00.

[†] percent of protected plots.

Appendix 3, table 1. Genotypes in the study and their pedigrees, selection numbers, reactions to scald and row-type in the experiments conducted at both Corvallis and Pendleton in 1986-87 and 1987-88

ENTRY NO.	PEDIGREE	SELECTION NUMBER	REACTION TO SCALD	SPIKE MORPHOLOGY
1	ROBUR/WA2196-68	OWB773160	RESISTANT	SIX-ROW
2	WA2116-67/BELTS67-1623	OWB71035	SUSCEPTIBLE	SIX-ROW
3	PERGA/S.W//WA1094-67	OWB71072	RESISTANT	SIX-ROW
4	1285/ASTRIX	FB7360702	RESISTANT	SIX-ROW
5	WA1245-68/72A8265	OWB773174	RESISTANT	SIX-ROW
6	NY6005-18/OWB70173	OWB783169	SUSCEPTIBLE	SIX-ROW
7	BOYER		RESISTANT	SIX-ROW
8	F5HJ556/125-83	OWB71035	RESISTANT	TWO-ROW
9	COSS/DWB71080-4H-1H	OWB773032	SUSCEPTIBLE	TWO-ROW
10	SONATE/OL	A76AS005	SUSCEPTIBLE	TWO-ROW
11	AR/RM1508,F1//COSS	SWB763150	SUSCEPTIBLE	TWO-ROW
12	SONJA/MST,F1//PULL72222	OWB7732628	SUSCEPTIBLE	TWO-ROW