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D. F. Garvin · A. H. D. Brown · J. J. Burdon

Inheritance and chromosome locations of scald-resistance genes derived from Iranian and Turkish wild barleys

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Abstract A set of advanced backcross barley lines derived from crosses between cv Clipper and different Iranian and Turkish wild barleys, which are homozygous for particular isozyme-marked donor intervals, was screened for resistance to barley scald. Eight lines that consistently exhibited scald resistance were identified, and genetic analysis indicated that single dominant genes encoded resistance in five of the lines, single recessive genes were present in two lines, and a pair of unlinked, dominant genes encoded the resistance in the last line. Linkage between the scald-resistance gene and the isozyme marking the introgressed donor chromosome interval was detected in four lines, allowing the chromosome locations of these resistance genes to be determined. One such resistance gene resides on barley chromosome 5, to which no other scald-resistance genes have been mapped; this gene has been designated *Rrs14*. A survey of the effectiveness of the eight resistance genes against a set of virulent pathotypes of the scald pathogen revealed that four of the lines were completely resistant to all of them. In two instances, the recovery of more than one scald-resistance gene from a single original donor parent could be demonstrated. These scald-resistance genes should provide additional opportunities for breeding programs that aim to develop scald-resistant barley cultivars.

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D. F. Garvin[1]
CRC For Plant Science, GPO Box 475, Canberra ACT 2601, Australia

A. H. D. Brown (✉) · J. J. Burdon
CSIRO Division of Plant Industry, GPO Box 1600, Canberra ACT 2601, Australia

Present address:
[1] U.S. Plant, Soil, and Nutrition Laboratory, Tower Road, Ithaca, NY 14853, USA

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Introduction

Scald, or leaf blotch, is a serious foliar disease that affects barley (*Hordeum vulgare* L.) in many of the major production regions of the crop around the world, including Canada, the USA, Europe, and Australia (Shipton et al. 1974). The primary loss from scald is reduced yield, which can reach or exceed 25% (Schaller 1951; James et al. 1968; Khan 1986; Vivar et al. 1987; Abbott et al. 1991). In addition, scald damage is manifested as a lowering of quality characteristics such as 1000-kernel weight and kernel plumpness (Schaller 1951; James et al. 1968; Khan and Crosbie 1988). This reduction in quality characteristics is particularly detrimental for malting barleys, since the grain is size-selected to eliminate the unsuitably small fraction prior to malting.

The causal agent of scald is the fungus *Rhynchosporium secalis* (Oud.) J. J. Davis. The pathogen is spread from plant to plant primarily by water-splash dispersion of spores, and can persist from season to season in crop residue (Shipton et al. 1974). Perhaps the most significant feature of *R. secalis* is the high level of pathogenic variability encountered in natural populations, which has repeatedly been demonstrated in different regions of the world where the disease is a problem (Jackson and Webster 1976; Brown 1990; Tekauz 1991; Jørgensen and Smedegaard-Petersen 1995). Somatic recombination (Burdon et al. 1994) and high mutation rates (Goodwin et al. 1994) may contribute to this variation, and alternative wild host plants of the pathogen may exert sustained selection pressure for greater pathogen virulence (Ali and Boyd 1974; Brown 1990).

One approach to reducing the severity of scald in the field is through the use of fungicides. This enables a measure of control in some situations, but the effectiveness of fungicides in controlling scald can vary from location to location (Kendall et al. 1993). Furthermore, pathotypes of *R. secalis* that are resistant to commonly used fungicides now have been identified in field populations, reducing fungicide effectiveness in some instances (Kendall et al. 1993; Locke and Phillips 1995). These detrimental aspects of fungicide use, coupled with their cost and environmental concerns regarding pesticide use, may lead to a gradual curtailment of their use for scald control.

An alternative approach to scald control is through the use of naturally occurring scald-resistance genes, of which 13 have so far been named (Jørgensen 1992; Wettstein-Knowles 1993). The incorporation of these genes into barley cultivars has been useful in combating scald in many instances. However, the different resistance genes work with varying degrees of effectiveness against pathotypes of *R. secalis* from different continents as well as from different geographic areas within the same country (Ali and Boyd 1974; Tekauz 1991). Furthermore, even if resistance genes are initially found to be effective under field conditions, the highly variable nature of *R. secalis* may result in the selection of new pathotypes that can overcome them over time. This is best illustrated by the failure of the cultivar Atlas 46 to exhibit long-term scald resistance in California (Houston and Ashworth 1957). New scald-resistance genes would therefore be beneficial for controlling scald since they can provide breeding alternatives in situations where known resistance genes have failed to confer adequate protection to the disease, or where they may lose effectiveness over time. Also, with greater numbers of scald-resistance genes, more options become available for pyramiding the genes within cultivars to improve their overall effectiveness, a breeding strategy that is likely to gain momentum as molecular markers for different scald-resistance genes continue to become available (Barua et al. 1993; Abbott et al. 1995; Schweizer et al. 1995).

The present study reports the genetic basis of scald resistance derived from different wild barley (*Hordeum vulgare* ssp. *spontaneum*) accessions collected in Iran and Turkey. Linkage between the scald-resistance genes and segregating isozyme markers is estimated, and the effectiveness of these genes against Australian pathotypes of *R. secalis* that can overcome some previously available resistance genes is examined.

Materials and methods

Plant materials

Backcrossing between different Iranian and Turkish wild barleys and the scald-susceptible Australian cultivar Clipper was conducted, with Clipper serving as the recurrent parent. Individual backcross lines were established in which the wild barley allele for one of 19 different isozyme loci was selectively maintained in the heterozygous state until the BC_3F_1, resulting in an average reversion of the genome to approximately 90% of the recurrent parent. Of the remaining portion of the genome derived from the donor, approximately 50% is expected to be located on the segment harboring the maintained donor isozyme gene (Brown et al. 1989). Individual BC_3F_1 plants were selfed to generate segregating BC_3F_2 populations (denoted by specific numbers). From each such population, a single individual homozygous for the introgressed donor isozyme allele was isolated and selfed to produce a BC_3F_3 line homozygous for the isozyme-marked donor segment (referred to by the same numerical designation as the BC_3F_2 population from which it was derived). For more details on the general method of line development, see Brown et al. (1988).

Disease inoculation procedures

The procedures used for plant culture and subsequent inoculation with *R. secalis* are described elsewhere (Abbott et al. 1991). Clipper plants were included (one plant per each ten experimental plants) as a positive control in inoculations.

Survey of BC_3F_3 lines for evidence of scald resistance

Twentythree BC_3F_3 lines, either deriving from different wild barley parents or from the same parent but selected to maintain different donor isozyme loci, were initially surveyed for evidence of scald resistance with an equimolar mix of spores from four different Australian pathotypes of *R. secalis* (R109, R112, R126, and R144). Eight plants from each line were simultaneously inoculated, grown in a shadehouse for approximately 3 weeks, and subsequently compared to Clipper for evidence of reduced necrotic lesions indicative of the presence of scald-resistance genes. The survey was repeated twice, during different times of the year, to confirm the consistency of the results. Lines uniformly exhibiting less scald damage than Clipper were chosen for further in-depth evaluation, as described below.

Genetic analysis of scald resistance

The inheritance of scald resistance in BC_3F_3 lines that were true-breeding for scald resistance was examined by scoring the BC_3F_2 population from which the BC_3F_3 line was derived for scald-resistance segregation, and using this information to infer the underlying genetic control of resistance in the BC_3F_3 line. Approximately 100 BC_3F_2 plants were inoculated with *R. secalis* pathotype R109 which was used in the initial BC_3F_3 survey, and grown under controlled environmental conditions described previously (Abbott et al. 1991), except that the temperature at which the plants were maintained during disease development was lowered to 12°C. Plants were rated for disease damage after approximately 18 days. Resistance ratings were based upon the numerical systems of Ali and Boyd (1974) and Jackson and Webster (1976), in which a score of 0 represents an absence of disease symptoms, 1 = the presence of small lesions confined to the leaf margins, 2 = small lesions mostly confined to the leaf margins, 3 = obvious lesions on all parts of the leaf surface, and 4 = large coalescing lesions leading to leaf collapse. Ratings between 0 and 2 were considered to represent resistant responses. The ratio of resistant to susceptible plants in each segregating population was tested for goodness-of-fit to different genetic models using the χ^2 statistic.

Linkage between scald-resistance genes and isozyme loci

To test whether the scald-resistance genes resided on the selectively introgressed donor chromosomal segments in any of the lines, linkage between the isozymes marking the introgressed segments and the scald-resistance genes was examined. Prior to inoculation with *R. secalis* and subsequent collection of disease-resistance segregation data, the BC_3F_2 plants were scored for isozyme segregation, using methods previously described (Brown 1983). Linkage between isozyme loci and scald-resistance genes was first assessed by contingency χ^2 analysis. Where significant deviations from independent segregation were detected, maximum-likelihood estimates of recombination rates and map distances were calculated using the Linkage-1 computer program (Suiter et al. 1983).

Performance of resistance genes against virulent pathotypes of *R. secalis*

To test the broad potential effectiveness of the resistance genes identified in this study, resistant BC_3F_3 lines were inoculated with a set of four different *R. secalis* pathotypes (H1.1, H2.1, WA3076, K8) collected from different regions in Australia that are known to be virulent on different barley cultivars. For each pathotype, eight plants of each line were simultaneously inoculated, grown under the same environmental conditions used for the genetic analysis, and subsequently numerically rated for disease damage.

Table 1 List of scald-resistant BC_3F_3 lines identified in this study, the wild barley donor parents used in their construction, and the introgressed donor isozyme gene in each line

Line number	Donor parent	Geographic origin of donor[a]	Introgressed donor isozyme gene
208	CPI 109829[b]	Mehran, Iran	*Gpi1*
219	CPI 109832	Dezful, Iran	*Acp2*
234	CPI 109846	Gaziantep, Turkey (3 km east)	*Est2*
239[c]	CPI 109851	Bitlis, Turkey (60 km south)	*Enp1*
240	CPI 109853	Diyarbakir, Turkey (43 km north)	*Adh2*
242	CPI 109860	Siverek, Turkey (20 km east)	*Acp2*
245	CPI 109861	Siverek, Turkey (9 km east)	*Enp1*
246[d]	CPI 109853	Diyarbakir, Turkey	*Est5*
249[e]	CPI 109846	Gaziantep, Turkey	*Idh1*

[a] For further information on original collection sites, see Nevo et al. (1986 a, b)
[b] Australian Commonwealth Plant Introduction number
[c] Resistance not confirmed during genetic analysis
[d] Derived from same donor plant as line 240
[e] Derived from same donor plant as line 234

Results

Scald resistance in backcross lines

Of the 23 BC_3F_3 lines twice screened for resistance to the mixture of four different *R. secalis* pathotypes, nine uniformly exhibited lower levels of scald damage than Clipper in both evaluations. Four of these lines appeared to be disease-free (lines 208, 240, 245, 249), while the other five (lines 219, 234, 239, 242, 246) exhibited varying reductions in scald damage relative to Clipper. These lines were therefore presumed to possess one or more scald-resistance genes. Information on the pedigrees of resistant BC_3F_3 lines can be found in Table 1.

Genetic control of scald resistance in backcross lines

The nine BC_3F_2 populations from which the resistant BC_3F_3 lines were derived were screened for scald-resistance segregation under controlled conditions that have been shown to permit rapid development of scald lesions on the plants (Abbott et al. 1992). The success of the inoculations conducted for these genetic studies is reflected by the consistently high disease ratings on the Clipper control plants; across all inoculations, 98% were rated as 3 or greater. This also indicates that the pathogen inoculum was applied consistently in the experiments.

Eight of the BC_3F_2 populations segregated for scald resistance (Table 2) while the ninth (population 239) exhibited no apparent resistance to the pathogen, and was not studied further. The scald-resistance segregation ratios in five of the BC_3F_2 populations (208, 219, 240, 242, and 245) conformed with a dominant monogenic model, whereas segregation for scald resistance in populations 234 and 246 suggested that the resistance was encoded by a single, recessive gene (Table 2). Resistant plants in BC_3F_2 populations 208, 240, and 245 were free of disease symptoms (rating of 0), while the resistant plants in populations 219, 234, 242, and 246 expressed some symptoms of the disease, with most being rated between 1 and 2.

Table 2 Segregation for scald resistance in BC_3F_2 populations, and goodness-of-fit of data to specific genetic models

BC_3F_2 population	Scald-resistance segregation		χ^2	*P*-value
	Resistant	Susceptible		
208	78	26	0.00[a]	1.00
219	71	33	2.51[a]	>0.10
234	27	77	0.05[b]	>0.75
240	73	27	0.21[a]	>0.50
242	80	24	0.15[a]	>0.50
245	76	28	0.15[a]	>0.50
246	21	83	1.28[b]	>0.25
249	95	9	1.03[c]	>0.25

[a] Calculated for monogenic, dominant resistance
[b] Calculated for monogenic, recessive resistance
[c] Calculated for digenic, dominant resistance

In contrast to the other seven BC_3F_2 populations, scald-resistance segregation in population 249 corresponded closely with the 15:1 ratio expected from the segregation for two unlinked, dominant resistance genes (Table 2). Further analysis of these data after decomposition into the three obvious classes of individuals in the segregating population – those with no disease symptoms, those with moderate resistance, and those that were fully susceptible – produced numbers (76, 19, and 9 respectively) that corresponded very closely to the 12:3:1 ratio expected for segregation at two unlinked genes, one of which confers a high level of scald resistance, and the other a moderate level of resistance ($\chi^2 = 1.03$, $P > 0.50$). The individuals scored as moderately resistant were unlikely to be inoculation escapes, since the Clipper control plants were uniformly susceptible. Since BC_3F_3 line 249 is true breeding for complete resistance to the *R. secalis* pathotypes used in the original BC_3F_3 screening (including R109 used in the genetic analysis), it is homozygous for the stronger of the two resistance genes detected in the BC_3F_2 population. However, the presence or absence of the more moderate resistance gene cannot be established since the stronger gene effectively masks its phenotype.

Linkage between scald-resistance genes and isozyme markers

In each BC_3F_2 population, segregation ratios for the isozyme gene maintained in a heterozygous state during backcrossing fit a co-dominant, monogenic model (Table 3). Unbiased segregation for both the scald-resistance and isozyme genes in all populations allowed maximum-likelihood estimation of recombination rates in those instances where non-independent segregation between the loci was revealed by contingency χ^2 analysis. In three of the seven populations possessing monogenic scald resistance (208, 242, 246), linkage between the scald-resistance gene and the isozyme gene marking the introgressed donor interval was detected. This proportion conforms to theoretical expectations (Brown et al. 1989). The resistance gene in BC_3F_2 population 208 was linked to *Gpi1*, which places it on barley chromosome 5. Similarly, the resistance genes present in lines 242 and 246 can be positioned to barley chromosomes 4 and 1, by virtue of their respective linkages with *Acp2* and *Est5* (Table 3).

Despite the fact that the scald resistance in BC_3F_2 population 249 was apparently controlled by two genes, linkage between *Idh1* and the resistance gene conferring a high level of resistance was still examined by classifying BC_3F_2 individuals strictly upon the presence or absence of any disease symptoms. This allowed linkage analysis between *Idh1* and the gene conferring complete scald resistance to be examined independently of the effects of the other resistance gene. This analysis revealed that the two genes are tightly linked (Table 3). The chromosomal location of *Idh1* in barley is currently undetermined, and so the chromosome location of this resistance gene was not established.

Effectiveness of resistance genes against virulent pathotypes of *R. secalis*

A range of effectiveness against the virulent *R. secalis* pathotypes was consistently observed among the different resistance genes. Complete resistance (rating of 0) to all four pathotypes was conferred by the resistance genes in four of the lines (lines 208, 240, 245, 249) and three other lines (219, 234, and 246) were effective to varying degrees against one or more of the pathotypes. In contrast, the resistance gene in line 242 did not condition resistance to any of the virulent pathotypes (Table 4). These results closely parallel those of the initial BC_3F_3 line evaluations; the four lines exhibiting an absence of disease symptoms when co-inoculated with the mixture of *R. secalis* pathotypes in the original BC_3F_3 survey were the same lines that were found to be completely resistant to all the virulent pathotypes.

Table 3 Goodness-of-fit of isozyme segregation in BC_3F_2 populations to a co-dominant monogenic model, and linkage estimates between isozyme loci and scald-resistance genes

BC_3F_2 Family	Segregating isozyme	Isozyme segregation[a]			χ^2	*P*-value	Linkage between isozyme and scald-resistance genes		
		CC	CW	WW			Contingency χ^2 (*P*-value)	Map distance $\pm$ error (cM)	Barley chromosome location
208	*Gpi1*	33	49	22	2.67	>0.25	45.3 (<0.000)	14.7 $\pm$ 3.7	5
219	*Acp2*	23	53	28	0.52	>0.75	0.6 n.s.		
234	*Est2*	27	50	27	0.15	>0.90	0.3 n.s.		
240	*Adh2*	29	46	28	1.19	>0.50	2.4 n.s.		
242	*Acp2*	23	46	35	4.15	>0.10	44.2 (<0.000)	15.6 $\pm$ 3.8	4
245	*Enp1*	20	56	28	1.85	>0.25	3.1 n.s.		
246	*Est5*	28	54	22	0.85	>0.50	20.8 (<0.000)	25.5 $\pm$ 4.7	1
249	*Idh1*	28	52	24	0.31	>0.75	84.8 (<0.000)	3.8 $\pm$ 1.9	Unknown

[a] CC, homozygous for the Clipper allele; CW, heterozygous; WW, homozygous for the wild barley donor allele

Table 4 Response of scald-resistant BC_3F_3 lines to a set of virulent pathotypes of *R. secalis*

Line number	Response to pathotype			
	H1.1	H2.1	K8	WA3076
208	R[a]	R	R	R
219	M[b]	S[c]	S	S
234	R	M	M	S
240	R	R	R	R
242	S	S	S	S
245	R	R	R	R
246	M	S	S	S
249	R	R	R	R
Clipper	S	S	S	S

[a] Complete resistance (rating = 0)
[b] Moderately resistant (rating = 1–2)
[c] Susceptible (rating = 3–4)

Discussion

Progress toward characterizing new resistance genes to barley scald has not proceeded as rapidly as might be expected, particularly given the seriousness of the disease in many parts of the world (Shipton et al. 1974). This may be due to the fact that some currently available resistance genes are sufficiently effective for controlling the disease, and it may also reflect the general difficulty of conducting accurate genetic studies for scald resistance, particularly under field conditions. Nonetheless, it is clear that existing scald-resistance genes are under constant threat of being overcome by new virulent pathotypes of the scald pathogen (Brown 1990). Thus, novel scald-resistance genes may serve two useful purposes. First, they may be of value in situations where existing scald-resistance genes do not confer adequate protection against the disease. Second, they can provide a measure of security to breeding programs should resistance genes that are currently in use fail over time.

Genetic interpretations of scald-resistance can vary, depending on the pathotypes of the pathogen being used, the genetic background in which a resistance gene resides, and the conditions under which the screening is conducted. The plant material and experimental techniques used in this study are ideal for circumventing some of these problems. First, Clipper is uniformly susceptible to all tested *R. secalis* pathotypes (Ali et al. 1976; Brown 1990); it therefore serves as an ideal genetic background for studies of scald resistance. Furthermore, the use of BC_3 generation material for genetic studies means that little of the donor genome remains, thus eliminating most unlinked modifiers of disease-resistance gene expression that might be present in the donor genome and which could confound genetic analysis. Lastly, the use of seedling-resistance screening and single plant scoring, which has previously been shown to yield accurate genetic data (Abbott et al. 1992), has permitted the genetic characterization of these new scald-resistance genes far more efficiently than if the study had been attempted under field conditions.

The eight resistance genes characterized in this study display a high level of distinctness when compared amongst themselves. They can be divided into seven classes, based upon a combination of inheritance, linkage, pathotypic effectiveness, and preliminary field performance data. This result tends to support the belief that wild barley is a particularly rich source of genetic variation that may be useful for barley improvement (Brown 1983).

Among the scald-resistance genes identified here, the gene carried by line 208 is of particular note. This gene is approximately 15 cM from *Gpi1* on barley chromosome 5. No other scald-resistance genes have been mapped to this chromosome, and thus we have designated it *Rrs14*. Barley chromosomes 1 and 4, which the resistances genes in lines 242 and 246 reside on, are known to harbor either major scald-resistance genes or QTLs for resistance to the disease (Wettstein-Knowles 1993; Backes et al. 1995). The determination of allelic relationships among these new wild barley-derived scald-resistance genes and those that have been previously described awaits the identification of *R. secalis* pathotypes that discriminate between the various genes, the development of intercross populations from the different gene sources, and the extensive disease resistance screening of such populations using the discriminating pathotypes.

Previous studies have demonstrated that seedling scald-resistance correlates well with scald-resistance in the field (Abbott et al. 1991; Xue et al. 1995). Indeed, a series of scald-resistance genes derived from Israeli wild barleys that were characterized in this fashion (Abbott et al. 1991, 1992) are effective in the field, and at least one of these genes provides a foundation for scald resistance in the new barley cultivar "Tantangara". Similarly, results of preliminary field trials of several of the scald-resistant BC_3F_3 lines characterized here indicate that the resistance genes they possess are at least as effective in the field as the genes derived from Israeli wild barleys. Additionally, the new set of genes described here exhibit a greater average effectiveness against the virulent *R. secalis* pathotypes used this study when compared with those derived from the Israeli wild barleys. Both findings suggest that the scald-resistance genes reported here will be useful for developing scald-resistant barley cultivars.

If new scald-resistance genes are to be incorporated into scald-resistance breeding programs, molecular markers for them would be of great benefit. The tight linkage between the scald-resistance gene in line 249 and *Idh1*, coupled with the low level of polymorphism at *Idh1* in barley and the rarity of the *Idh1* allele tagging the gene, will make *Idh1* extremely useful for introducing the line 249 resistance gene into new germ plasm. And, although the other mapped resistance genes in

lines 208 (*Rrs14*), 242, and 246 are not as tightly linked to their respective isozyme loci, indirect selection may still be effective for them. Furthermore, this linkage information will prove useful for future development of more closely linked markers.

In two instances, more than one resistance gene was recovered from a particular donor individual. Lines 240 and 246 both trace back to the same wild barley plant, but were selected to maintain different segments of that donor during backcrossing (Table 1). While line 240 possesses a single dominant gene that confers a high level of scald resistance, line 246 possesses a single recessive gene that imparts moderate resistance. Even more interesting is the case of lines 234 and 249 which, like lines 240 and 246, are derived from the same donor plant but were selected to contain different donor chromosome segments (Table 1). Line 234 possesses a single recessive resistance gene, while BC_3F_2 population 249 was segregating for two unlinked, dominant resistance genes. This indicates the presence of a minimum of three different scald-resistance genes in the original wild barley donor plant. Such a finding is in agreement with studies of other putative "gene-for-gene" systems in which multiple resistance genes have been detected (Burdon 1987).

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References

Abbott DC, Burdon JJ, Jarosz AM, Brown AHD, Müller WJ, Read BJ (1991) The relationship between seedling infection types and field reactions to leaf scald in Clipper barley backcross lines. Aust J Agric Res 42 : 801–809

Abbott DC, Brown AHD, Burdon JJ (1992) Genes for scald resistance from wild barley (*Hordeum vulgare* ssp *spontaneum*) and their linkage to isozyme markers. Euphytica 61 : 225–231

Abbott DC, Lagudah ES, Brown AHD (1995) Identification of RFLPs flanking a scald-resistance gene on barley chromosome 6. J Hered 86 : 152–154

Ali SM, Boyd WJR (1974) Host range and physiologic specialization in *Rhynchosporium secalis*. Aust J Agric Res 25 : 21–31

Ali SM, Mayfield AH, Clare BG (1976) Pathogenicity of 203 isolates of *Rhynchosporium secalis* on 21 barley cultivars. Physiol Plant Pathol 9 : 135–143

Backes G, Graner A, Foroughi-Wehr B, Fischbeck G, Wenzel G, Jahoor A (1995) Localization of quantitative trait loci (QTLs) for agronomic important characters by the use of a RFLP map in barley (*Hordeum vulgare* L.) Theor Appl Genet 90 : 294–302

Barua UM, Chalmers KJ, Hackett CA, Thomas WTB, Powell W, Waugh R (1993) Identification of RAPD markers linked to a *Rhynchosporium secalis* resistance locus in barley using near-isogenic lines and bulked segregant analysis. Heredity 71 : 177–184

Brown AHD (1983) Barley. In Tanksley SD, Orton TJ (eds) Isozymes in plant genetics and breeding, part B. Elsevier Science Publishers, Amsterdam, pp 57–77

Brown AHD, Munday J, Oram RN (1988) Use of isozyme-marked segments from wild barley (*Hordeum spontaneum*) in barley breeding. Plant Breed 100 : 280–288

Brown AHD, Lawrence GJ, Jenkin M, Douglass J, Gregory E (1989) Linkage drag in backcross breeding in barley. J Hered 80 : 234–239

Brown JS (1990) Pathogenic variation among isolates of *Rhynchosporium secalis* from barley grass growing in south eastern Australia. Euphytica 50 : 81–89

Burdon JJ (1987) Phenotypic and genetic patterns of resistance to the pathogen *Phakopsora pachyrhizi* in populations of *Glycine canescens*. Oecologia 73 : 257–267

Burdon JJ, Abbott DC, Brown AHD, Brown JS (1994) Genetic structure of the scald pathogen (*Rhynchosporium secalis*) in south east Australia: implications for control strategies. Aust J Agric Res 45 : 1445–1454

Goodwin SB, Webster RK, Allard RW (1994) Evidence for mutation and migration as sources of genetic variation in populations of *Rhynchosporium secalis*. Phytopathology 84 : 1047–1053

Houston BR, Ashworth LJ Jr (1957) Newly determined races of the barley scald fungus in California. Phytopathology 47 : 525

Jackson LF, Webster RK (1976) Race differentiation, distribution, and frequency of *Rhynchosporium secalis* in California. Phytopathology 66 : 719–725

James WC, Jenkins JEE, Jemmett JL (1968) The relationship between leaf blotch caused by *Rhynchosporium secalis* and losses in grain yield of spring barley. Ann Appl Biol 62 : 273–288

Jørgensen JH (1992) Coordinator's report: disease and pest-resistant genes. Barley Genet Newslett 22 : 110–134

Jørgensen HJL, Smedegaard-Petersen V (1995) Pathogenic variation of *Rhynchosporium secalis* in Denmark and sources of resistance in barley. Plant Dis 79 : 297–301

Kendall SJ, Hollomon DW, Cooke LR, Jones DR (1993) Changes in sensitivity to DMI fungicides in *Rhynchosporium secalis*. Crop Prot 12 : 357–362

Khan TN (1986) Effects of fungicide treatments on scald [*Rhynchosporium secalis* (Oud.) J. Davis] infection and yield of barley in Western Australia. Aust J Expt Agric 26 : 231–235

Khan TN, Crosbie GB (1988) Effect of scald [*Rhynchosporium secalis* (Oud.) J. Davis] infection on some quality characteristics of barley. Aust J Expt Agric 28 : 783–785

Locke T, Phillips AN (1995) The occurrence of carbendazim resistance in *Rhynchosporium secalis* on winter barley in England and Wales in 1992 and 1993. Plant Pathol 44 : 294–300

Nevo E, Zohary D, Beiles A, Kaplan D, Storch N (1986 a) Genetic diversity and environmental associations of wild barley, *Hordeum spontaneum*, in Turkey. Genetica 68 : 203–213

Nevo E, Beiles A, Kaplan D, Storch N, Zohary D (1986 b) Genetic diversity and environmental associations of wild barley *Hordeum spontaneum* (*Poaceae*), in Iran. Plant Syst Evol 153 : 141–164

Schaller CW (1951) The effect of mildew and scald infection on yield and quality of barley. Agron J 43 : 183–188

Schweizer GF, Baumer M, Daniel G, Rugel H, Röder MS (1995) RFLP markers linked to scald (*Rhynchosporium secalis*)-resistance gene *Rh2* in barley. Theor Appl Genet 90 : 920–924

Shipton WA, Boyd WJR, Ali SM (1974) Scald of barley. Rev Plant Pathol 53 : 839–861

Suiter KA, Wendel JF, Case JS (1983) LINKAGE-1: a PASCAL computer program for the detection and analysis of genetic linkage. J Hered 74 : 203–204

Tekauz A (1991) Pathogenic variation in *Rhynchosporium secalis* on barley in Canada. Can J Plant Pathol 13 : 298–304

Vivar HE, Burnett PA, Bowman JE (1987) Barley breeding for multiple disease resistance. Barley Genet V : 615–623

Wettstein-Knowles P von (1993) Barley (Hordeum vulgare) 2n = 14. In : O'Brien SJ (ed) Genetic maps. Locus maps of complex genomes, 6th edn. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, pp 6.110–6.129

Xue AG, Burnett PA, Helm J, Rossnagel BG (1995) Variation in seedling and adult-plant resistance to *Rhynchosporium secalis* in barley. Can J Plant Pathol 17 : 46–48