Sources of Resistance in Barley to *Rhynchosporium secalis*

R. K. WEBSTER, Professor, Department of Plant Pathology, University of California, Davis 95616; L. F. JACKSON, Assistant Professor, University of Florida, Gainesville 32601; and C. W. SCHALLER, Professor, Department of Agronomy and Range Science, University of California, Davis 95616

ABSTRACT

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Nearly 18,000 entries from the USDA world barley collection were evaluated in the field for resistance to *Rhynchosporium secalis*. Maximum disease exposure was provided by repeated inoculations of a mixture of five California isolates with known combined abilities of pathogenicity on previously identified sources of resistance. No distinct pattern of origin of resistance was revealed. Nine entries from six countries (France, Japan, USA, Israel, Ethiopia, and Wales) showed no symptoms, but 273 entries with representatives of three species (*Hordeum distichum* L. emend Lam, *H.* vulgare L. emend Lam, and *H. irregulare* Åberg and Wiebe) showed high levels of resistance. All other entries were susceptible to the range of pathogenicity of the inoculum employed. Resistant entries included two-rowed and six-rowed spring, winter, and facultative types. Results nearly identical to those in the field were obtained when more than 2,000 of the entries were evaluated in the greenhouse. Use of newly identified sources of resistance should include consideration of the presence and frequency of specific races, because these differ throughout the world.

Leaf scald, caused by *Rhynchosporium secalis* (Oud.) Davis, is a serious foliage pathogen of barley (*Hordeum vulgare* L. emend. Lam) when environmental conditions favor disease development. Losses of up to 35% have been reported in California (21) and up to 70% in Australia (1). Resistance to barley scald has been described in many barley cultivars (8,10,13,15–18,22,23), but inheritance of resistance has been studied in relatively few (2–7,14,24,25). In these studies, 17 genes for resistance to various races of *R. secalis* were identified and their genetics demonstrated (Table 1).

In numerous cases one or two genes for resistance have been incorporated into agronomic cultivars only to succumb to new races or shifts in the pathogen populations. A most notable example is the short-term control obtained with the Atlas 46 cultivar in California. Atlas 46, containing Rh2 and Rh3 resistance genes (4), was released in 1947 as resistant to California races of *R. secalis*, but by 1956 it was considered completely susceptible. According to a recent survey of *R. secalis* in California, 39% of a sample of more than 250 isolates representing 75 races were capable of attacking Atlas 46 (9). However, Ali et al (1) described 35 distinct races of *R. secalis* from barley-growing areas of South Australia, and none were capable of attacking Atlas 46. Thus, even though known genes for resistance may not be effective for control against populations of a pathogen in one location, they need not necessarily be eliminated from use in another. There is ample justification to believe incorporation of known or newly defined resistant genes into agronomically acceptable cultivars is a worthy goal, especially in light of knowledge regarding intelligent gene-deployment based on potentials of stabilizing selection and monitoring presence and frequencies of specific races in pathogen populations. The objective of our study was to identify potentially new sources of resistant genes to control barley scald disease.

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MATERIALS AND METHODS

Seed of nearly 18,000 entries from the World Barley Collection maintained by the Beltsville Agricultural Research Center, USDA SEA/AR, was obtained and planted at Davis, CA, in late October 1976. Ten seeds of each entry were planted in a hill, 30 cm apart. The cultivar Numar, susceptible to all California races tested, was used as a control and was entered every 20 hills.

Plants were inoculated with conidia of a mixture of five California races representing the most virulent of the pathogenicity spectrum (9) and pathogenic to all previously identified sources of resistance (Table 1). Methods for growing and preparing inoculum have been described (9). Inoculum was sprayed onto plants with Hudson sprayers and calculated to deliver at least one million conidia per hill. Inoculations were timed to correspond with periods of free moisture when possible and were repeated three times. The first inoculation was applied when plants began to tiller and the last was applied in early February 1977 after most entries had grown to at least 30 cm in height.

Natural free moisture was supplemented by both sprinkler and furrow irrigation.

Observations for scald disease were made as plants headed. Because heading varied considerably among entries, the evaluation time could not be completely standardized, and a final rating for all entries was made during the last 2 wk of April 1977.

The rating system used (9) can be briefly described as follows: 0 = no visible symptoms, 1 = very small lesions confined to leaf margins, 2 = small lesions

Table 1. Genes for resistance to scald (*Rhynchosporium secalis*) in barley

Gene symbol designated by author	Cultivar in which first identified	Reference
Rh3	Turk CI 5611-2	4,15
Rh4	LaMesita CI 7565	4
$Rh^2 4$, Rh^2	Modoc CI 7566	4, 7
...	Trebi CI 936	14
Rh_a, Rh	Briar CI 7157	3
Rh2	Atlas CI 4118	4
Rh5	Turk CI 5611-2	4
rh8	Nigrinudum CI 2222	25
rh6	Jet CI 967	2
rh7, rh^5	Jet CI 967	2, 7
Rh9	Abyssinian CI 668	2
...	CI 3515	24
...	CI 8618	24
Rh^3	Wisconsin Winter × Glabron CI 8162	7
Rh^4	Osiris CI 1622	7
Rh10	Osiris CI 1622	7
rh11	CI 4364	7

not confined to leaf margins, 3 = large coalescing lesions involving most of the leaf area, and 4 = collapse of leaf with or without infection on glumes and awns. Categories 3 and 4 were considered susceptible, 1 and 2 resistant to moderately resistant, and 0 immune.

Methods used for evaluation in the greenhouse appear elsewhere (9).

RESULTS AND DISCUSSION

The experimental conditions resulted in a severe scald epidemic in the experimental area. All plantings of Numar (more than 900 hills) were severely infected and received a rating of 4. Numerous infections of the lemma, palea, and awns occurred, and in most cases the plants were nearly defoliated. Of the nearly 18,000 entries evaluated, nine developed no scald symptoms and were judged immune to the races used as inoculum and to those present naturally. The entries judged resistant (rated 1 or 2) are listed in Table 2. All other entries were susceptible (rated 3 or 4).

Results of screening more than 2,000 of the entries in the greenhouse under controlled conditions using a mixture of the same isolates and plants rated at the 1–4 leaf stage were essentially identical to those obtained in the field.

Unexploited sources of resistance to scald disease are readily available in the USDA world collection of barley, based on the fact that all sources studied previously (Table 1) are susceptible to the races employed as inoculum. It is not known how much repetition occurs within the newly identified sources, and no claim is made that each is distinct.

The origin of the resistant entries (Table 3) is in marked contrast to similar studies in which the same collection was surveyed for resistance to other barley diseases. Resistance to net blotch, *Pyrenophora teres* (Died.) Drechsl. was most frequent among barleys from Manchuria (20), whereas resistance to barley yellow dwarf virus was found almost exclusively in Ethiopian introductions (11,19). In a study of 650 Ethiopian barleys, Qualset and Moseman (12) found resistance to nine barley diseases, with frequencies ranging from 3.8 to 31.2% for different diseases. In contrast, only 22 entries from Ethiopia and one from China were resistant to the races of *R. secalis* used in this study. This is surprisingly low, considering the fact both countries are recognized as areas of genetic diversity for barley and collectively comprise approximately 20% of the collection.

The concentration of resistant types in countries with well-established breeding programs and where scald is severe (United States, Israel, Germany, and France) suggests that resistance to the complex races used in our study is the product of the accumulation of genes from several sources through artificial hybridization and subsequent man-directed selection. Consequently, finding resistance to these or similarly complex races conditioned by a single gene or from a single area is very remote. This is further emphasized by the fact that none of the previously identified genes (Table 1), individually or in naturally occurring combinations, provide protection against these particular races, although the reported parental material is of widely divergent origin.

On the other hand, knowledge of origin of collection may be useful in selecting entries adapted to different areas for

Table 2. Barley entries from the U.S. Department of Agriculture World Barley Collection that were resistant to scald disease in California in 1976

CI No.
00206, 00277,[a] 02036, 02746,[a] 03195,[a] 03384, 03824, 03825, 03888, 03938, 04294, 04376, 04377, 04677,[a] 05951, 06192, 06269, 06270, 06274,[a] 06377, 06493, 06499, 06561, 06563, 06565, 06567, 06628, 07032, 07033, 07071, 07072, 07283, 07284, 07319, 07330, 07331, 07508, 07526, 07528, 07531, 07588, 08063, 08064, 08071, 08073, 08074, 08075, 08076, 07571, 07572, 07573, 07574, 07575, 07577, 07580, 07582, 07585, 08266, 08269, 08271, 08272, 08275, 08278, 08281, 08292, 08446, 08476, 08618, 08795, 08796, 08797, 08798, 08801, 08802, 08803, 08804, 08979, 09018, 09055, 09518, 09521, 09522, 09544, 09565, 09567, 09573, 09702, 09796, 09863, 09864, 09866, 09867, 09889, 09890, 09891, 09946, 10011, 10012, 10032, 10045, 10046, 10048, 10049, 10051, 10052, 10053, 10054, 10055, 10056, 10057, 10058, 10097, 10430, 10431, 10536, 10543, 10665, 10848, 10878, 10894, 10895, 10896, 10900, 10901, 10902, 10903, 10904, 10905, 11150, 11161, 11162, 11164, 11165, 11178, 11180, 11220, 11278, 11285, 11286, 11358, 11371, 11372, 11579, 11580, 11632, 11641, 11642, 11643, 11644, 11668, 11669, 11838, 11910, 12080, 12251, 12491, 12502, 12688, 12783, 13036, 13169, 13192, 13201, 13202, 13618, 13689, 14930, 14945, 15143, 15186, 15233, 15375, 15397, 15398, 15424, 15425, 15426, 15427, 15428, 15430, 15431, 15538, 16224, 16264, 16268, 16269, 16270, 16446, 16447, 16448, 16487, 16488
PI No.
219796, 296795, 296865, 296881, 296904, 296912, 296921, 296942,[a] 328497,[a] 328981, 329037, 329047, 329110, 329121, 342425, 356299, 356459, 356562,[a] 356657, 356780, 362206, 369744, 371455, 382214, 382368, 382373, 382375, 382380, 382523, 382695, 382933, 382946, 383069, 383165, 383174, 383181, 383202, 386731, 386753, 386754, 386760, 386765, 386766, 386770, 386771, 386774, 386777, 386778, 386783, 386785, 386786, 386790, 386792, 386804, 386811, 386815, 386817, 386820, 386824, 386825, 386839, 386861, 386862, 386865, 386880, 386908, 386913, 386921, 386923, 386929, 386930, 386949, 386961, 386966, 386996, 387001, 387096, 387117, 387129,[a] 387167

[a]Developed no scald symptoms (immune to races tested).

Table 3. Distribution of scald resistance identified in the U.S. Department of Agriculture World Collection of barley

Origin of accession	**Resistant entries (no.)**	**Origin of accession**	**Resistant entries (no.)**
		Central Asia and	
Northwest Europe		Indian Subcontinent	
Britain	5	Afghanistan	...
Denmark	...	India	5
North Central Europe		Iran	...
Finland	...	Iraq	1
Germany	21	Pakistan	...
Netherlands	...	North Africa	
Norway	1	Algeria	...
Sweden	1	Egypt	...
Northeastern Europe		Morocco	...
Czechoslovakia	2	Tunisia	...
Poland	1	East Africa	
USSR	6	Ethiopia	22
Southwest Europe		Sudan	...
Belgium	2	South Africa	...
France	14	Far East	
Italy and Sicily	...	China	1
Portugal	1	Japan	2
Spain	...	Korea	...
Switzerland	1	Australia	...
Southeast Europe		North America	
Austria	8	Canada	5
Bulgaria	2	United States	111
Greece	1	Central and South America	
Hungary	...	Argentina	...
Romania	...	Colombia	...
Yugoslavia	4	Mexico	...
Middle East		Peru	...
Israel	14		
Jordan	...		
Syria	1		
Turkey	2		

potential use in breeding programs. Entries resistant to scald should be useful in many improvement programs, since they were obtained from 28 countries and included two-rowed and six-rowed spring, winter, and facultative types.

The success of these sources in controlling barley scald will obviously depend on the manner in which they are deployed and knowledge of the pathogen population in the area of intended use. We need extensive studies on prevailing races in the pathogen population as new sources of resistance are employed and also an awareness of the potential to recall "old genes" for use as populations adapt or where certain races do not occur.

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