Race Differentiation, Distribution, and Frequency of Rhynchosporium secalis in California

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ABSTRACT

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The pathogenic variability of the barley scald fungus, *Rhynchosporium secalis,* in California was examined. One hundred seventy-five single-spore isolates of the fungus, obtained from naturally infected barley in 23 counties, were differentiated into 75 pathogenic races on 14 barley cultivars representing many of the currently known genes for specific resistance to scald. The 75 races encompassed a wide spectrum of pathogenicity and included a race pathogenic to none of the differentials as well as a race pathogenic to all of them. Four races, which accounted for 37% of the isolates, included two races from each end of the race spectrum; that is, two races pathogenic to relatively few of the differentials and two races pathogenic to most of the differentials. The group of races pathogenic to most of the differentials was concentrated in the southern San Joaquin Valley, the major barley-producing area of the state. The differential reactions of the cultivars revealed the relative effectiveness of the various sources of resistance to the California population of the fungus. The most effective cultivars, C.I. 5831, Hudson, and Turk, were susceptible to 26, 34, and 36%, respectively, of the population. The disease reactions of the cultivars in the host range revealed differences in the resistance of cultivars previously thought to carry identical resistance genes. The cultivars involved either had additional genes for resistance and susceptibility or the genes previously thought to be identical were different.

Additional key words: barley scald, *Hordeum vulgare* L., disease resistance.

Leaf scald, caused by the fungus *Rhynchosporium secalis* (Oud.) Davis, is a serious foliage disease of barley *(Hordeum vulgare* L.) in California, where yield losses of up to 35% have been reported (19). Atlas 46 (C.I. 7323), with genes for desirable agronomic characters from Atlas (C.I. 4118), and genes for race-specific scald resistance from Turk (C.I. 5611-2), was released in California in 1947, as a scald-resistant cultivar. By 1953 the scald disease had been found on Atlas 46 at several locations, and by 1956 Atlas 46 was considered extremely susceptible (8). Consequently, the only attempt thus far to control the disease through the conventional vertical resistance method in California resulted in short-term control. In retrospect, the development of a cultivar having lasting resistance to scald was unlikely, for although the pathogenic variability of the causal organism had been well-documented (3, 5, 7, 8, 9, 10, 11, 14, 15, 16, 18, 20, 21, 25), the occurrence, frequency, and distribution of specific pathogenic races had not been fully investigated. Therefore, work was undertaken to determine the range of pathogenic variability of *R. secalis,* to identify existing races, and to determine their frequency and distribution in California. We sought this information to provide a basis upon which to develop breeding programs for scald resistance.

MATERIALS AND METHODS

Collection and isolation of the fungus.—A representative sample of the fungus as it occurred on barley in California was obtained by isolating from naturally infected barley in 23 counties during the spring of 1973. Most of the isolates were from the Sacramento Valley and the San Joaquin Valley. Isolates also were obtained from the coastal valleys (Fig. 1). Leaves with fresh lesions were collected from barley fields and brought to the laboratory for isolation of the fungus.

Leaf pieces with lesions were wet in 70% ethyl alcohol for 10 seconds, surface-sterilized in a 1:9 dilution of commercial bleach (Clorox, 5.25% NaOCl) for 90 seconds, placed in petri dishes on Whatman No. 2 filter paper moistened with sterile distilled water, and incubated at 15 C for 48 hours to induce sporulation of the fungus. Spores then were scraped from the lesions with a wire transfer loop and streaked onto plates of potato-dextrose agar (PDA). The plates were incubated at 15 C for 24-48 hours to allow the spores to germinate. Glass needles with tips approximately the diameter of the germ tube of the spore (about 1 μm) were used to pick up single germinated spores and transfer them to fresh plates of PDA to establish the single-spore isolates used for subsequent race identification.

Differential host range.—Race identification rests on the ability of selected barley cultivars to react differentially, with regard to resistance and susceptibility, to isolates of the fungus. Cultivars with known specific genes for resistance to scald were thus selected for inclusion in a differential host range. Fourteen cultivars were ultimately included in the set of differentials, including the six cultivars used by Schein to differentiate seven races of *R. secalis* (21). The differential cultivars and their identified genes for resistance to scald are shown in Table 1.

Inoculation procedure.—To minimize the influence of environmental variation on disease expression and subsequent race determination, methods of preparing the

inoculum and the host range, as well as the inoculation process itself, were standardized.

Inoculum for each isolate was produced by seeding PDA plates with 1 ml of a spore suspension obtained from the stock culture, incubating the plate at 15 C for 2 weeks, harvesting the new crop of spores in sterile distilled water, and adjusting the concentration to the desired level using a Spencer Brightline hemacytometer. The spore concentration of 2×10^5 spores/ml was selected for use in all inoculations after initial tests with concentrations ranging from 1×10^2 to 2×10^5 spores/ml were carried out.

Fig. 1. Collection sites for *Rhynchosporium secalis*. Each dot on the map of California represents a barley field from which *Rhynchosporium secalis* was isolated.

The differentials were planted together in U.C. soil mix C-2 (12) in metal flats; each flat contained the complete host range, with each cultivar represented by a row of five seedlings. The barley was grown in a greenhouse maintained at 15-25 C and inoculated at the one-and-a-half- to three-leaf stage, about 11 days after sowing. The day prior to inoculation the plants were placed in a controlled environment chamber maintained at 15 C. The inoculum for each isolate, 50 ml of a spore suspension adjusted to 2×10^5 spores/ml, was atomized onto the plants with a DeVilbiss Adjustable Tip Atomizer 15 attached to an air hose. The inoculum was allowed to dry on the barley for a period of 3 hours, after which the controlled environment chamber was brought to 100% relative humidity through the use of a series of atomizers attached to a combination of air and distilled-water outlets which produced a fine mist in the chamber. After 48 hours, the barley was returned to the greenhouse where symptom expression permitted disease evaluation 2 weeks later. One uninoculated control set, sprayed with distilled water only, was included with every set of inoculated differentials.

Disease rating.—A disease rating scale of 0-4 was utilized in which each seedling was scored for foliar symptoms on the following basis: 0 = no visible symptoms; 1 = very small lesions confined to leaf margins; 2 = small lesions not confined to leaf margins; 3 = large coalescing lesions, involving a majority of the leaf area; and 4 = total collapse of the leaf. The seedlings of each cultivar-isolate combination were evaluated and a disease rating equal to the highest score given was assigned to that combination.

Twelve randomly selected isolates were retested to establish the constancy and repeatability of the method and to determine a numerical range for resistance and susceptibility. Another 12 randomly selected isolates were inoculated onto 10 of the cultivars 6 weeks after planting

TABLE 1. Differential host range used to identify races of *Rhynchosporium secalis* occurring on barley in California

Cultivar	Previously identified genes for resistance	Gene action	Reference(s)
Atlas C.I. 4118	*Rh2*	Dominant	4
Atlas 46 C.I. 7323	*Rh2, Rh3*	Both dominant	4
Brier C.I. 7157	*Rh*	Dominant	2, 4
California 1311 (Modoc C.I. 7566) C.I. 2376[a] C.I. 5831[a]	*Rh*^{2}*4* and an unnamed recessive gene	Dominant; recessive	4, 17
Hudson C.I. 8067	*Rh*	Dominant	6
Kitchen C.I. 1296	*Rh9*	Incompletely dominant	1
La Mesita C.I. 7565	*Rh4*	Dominant	4
Osiris C.I. 1622	*Rh4*	Dominant	4
Steudelli C.I. 2266	*rh6, rh7*	Complementary recessive	1
Trebi C.I. 936	*Rh4* and an unnamed recessive gene	Dominant; recessive	4, 17
Turk C.I. 5611-2	*Rh3, Rh5*	Both dominant	4
Wisconsin Winter × Glabron C.I. 8162	*Rh*^{3}	Incompletely dominant	6

[a]Both C.I. 2376 and C.I. 5831 have at least two dominant genes for resistance (M.S. Mohamed and C. W. Schaller, *unpublished*).

to compare the disease reaction on older plants with that on seedlings.

Race differentiation.—Isolates were classified into races on the basis of the differential reactions of the barley cultivars in the host range. Disease ratings of 3 and 4 were grouped as susceptible reactions and ratings 0, 1, and 2 were grouped as resistant reactions. Individual isolates were characterized by the reactions that they caused on the entire host range. Isolates that caused the same reaction as other isolates on all of the differentials were placed in a single race. The identified races were numbered so that the lower-numbered races represented those pathogenic to few of the differentials, and the higher-numbered races represented those pathogenic to progressively more of the differentials. The list culminated in race 75, which was pathogenic to all of the differentials.

Race distribution and frequency.—In order to facilitate study of the distribution of the races in California, the sampled areas were consolidated into the following four regions: (i) a Northern Valley region which included Butte, Colusa, Glenn, Shasta, and Sutter counties; (ii) a Middle Valley region which included Sacramento, San Joaquin, Solano, Stanislaus, Tuolumne, and Yolo counties, (iii) a Southern Valley region which included Fresno, Kern, Kings, Madera, Merced, and Tulare counties; and (iv) a Coastal Valley region which included Alameda, Contra Costa, Monterey, San Benito, San Luis Obispo, and Santa Clara counties. The races were consolidated into three groups based on the number of differentials to which they were pathogenic. Group I consisted of races pathogenic to 0-3 differentials; Group II, 4-9 differentials; and Group III, 10-14 differentials.

RESULTS AND DISCUSSION

Determination of an optimum inoculum level.—The spore concentration used in race differentiation must consistently induce either a resistant or a susceptible host response in a given cultivar. Three isolates were tested at each of six different spore concentrations (1×10^2, 1×10^3, 1×10^4, 5×10^4, 1×10^5, and 2×10^5 spores/ml) on the 14 differentials. The two highest spore concentrations resulted in the clearest differentiation between resistance and susceptibility on all cultivars in the differential host range. The lower concentrations allowed some cultivars that were susceptible at the higher concentrations to escape. The spore concentration of 2×10^5 spores/ml was chosen for use in race differentiation.

Repeatability of the method for race differentiation.—The constancy and repeatability of the method for race differentiation was determined by retesting 12 random isolates on the 14 differentials. There was some variation in individual numerical ratings for some cultivar-isolate combinations, but all variability was either in the 0-2 or the 3-4 range. There was always a clear delineation between ratings of 2 and 3, justifying the break in resistance and susceptibility at that point.

Validity of the seedling test for race differentiation.—The predictive value of the seedling test for the disease reaction of an older, established barley plant was shown by the inoculation of 10 cultivars with 12 previously characterized isolates when the barley was 6 weeks old. The plants were judged susceptible if they developed large coalescing lesions or if the entire leaf collapsed, and resistant if they developed at most small isolated lesions. Cultivars resistant at the seedling stage were resistant at the later stage; and those susceptible at the seedling stage were susceptible at the later stage.

Race differentiation, distribution, and frequency.—The characterization of 175 isolates on the differential host range resulted in the identification of 75 pathogenic races. A broad spectrum of pathogenicity was encountered. There was a race pathogenic to all of the differentials, a race pathogenic only to the universal suscept, Wong, as well as 73 races pathogenic to from one to 13 of the 14 differentials (Table 2).

When the pathogenic variability of the fungus had been demonstrated, the frequency and distribution of the individual races took on added importance. Of the 75 races identified, four races were represented by 65 of the 175 isolates (37%), 71 other races by four or fewer isolates, and 46 races by single isolates only. Of particular interest in the controversy over the theory of stabilizing selection in which race fitness is equated to race simplicity (13, 23) is the fact that the four most frequent races were races designated 2, 9, 72, and 73, and that these included two races from each end of the race spectrum. Since the isolates were obtained from commercial California barley lacking known specific resistance, the phenomenon of stabilizing selection may not apply to the barley scald system.

Individual race distribution is meaningful only for the four most frequent races since most of the other races were represented by only one to four isolates. Races 2 and 9 were most frequent in the Northern and Middle Valley regions, whereas races 72 and 73 predominated in the Southern Valley region. That pattern was substantiated by the distribution of consolidated groups of the individual races. Group I consisted of 65 isolates, representing races 1-20; Group II consisted of 57 isolates, representing races 21-61; and Group III consisted of 53 isolates, representing races 62-75. Of the 175 isolates that were characterized, 33 were from the Northern Valley region, 60 were from the Middle Valley region, 68 were from the Southern Valley region, and 14 were from the Coastal Valley region (Table 3). Although there were representatives of each race group in each area surveyed, Group I races predominated in the Northern, Middle, and Coastal Valley regions, making up 54.5, 51.6, and 57.2%, respectively, of the isolates from those regions, whereas Group III races predominated (60.3%) in the Southern Valley region. The significance of the above distribution is that the Southern Valley region, the dominant barley-producing area of California, hosts a *R. secalis* population dominated by Group III isolates; in fact, it contains 77.3% of all Group III isolates. Since Group III isolates represent the races that are able to overcome most of the known genes for resistance to scald, the chances are slight that a scald-resistant cultivar could be developed for that region, at least by utilizing the known genes for resistance.

TABLE 2. Disease reactions of the differential barley cultivars to races of *Rhynchosporium secalis* in California

	Resistance/susceptibility to *R. secalis* races of barley cultivars:														
Race no.	Atlas	Atlas 46	Brier	Calif. 1311	C.I. 2376	C.I. 5831	Hudson	Kitchen	La Mesita	Osiris	Steu-delli	Trebi	Turk	Wisc. Win. × Glab.	Wong[c]
1	–[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	+
2	+[b]	–	–	–	–	–	–	–	–	–	–	–	–	–	+
3	–	–	–	–	–	–	–	–	+	–	–	–	–	–	+
4	–	–	–	–	–	–	–	–	–	–	–	–	–	+	+
5	–	–	–	–	–	–	–	–	–	–	+	–	–	–	+
6	+	–	–	–	–	–	–	–	–	–	–	–	–	+	+
7	+	–	–	–	–	–	–	–	–	+	–	–	–	–	+
8	+	–	–	–	–	+	–	–	–	–	–	–	–	–	+
9	+	–	–	–	–	–	–	–	–	–	+	–	–	–	+
10	+	–	–	–	–	–	–	+	–	–	–	–	–	–	+
11	+	–	–	–	–	+	–	–	+	–	–	–	–	–	+
12	+	–	–	–	–	–	–	–	+	–	+	–	–	–	+
13	+	–	–	–	–	+	–	–	–	–	+	–	–	–	+
14	+	–	–	–	–	–	–	–	–	–	+	–	–	+	+
15	+	–	–	–	–	–	–	–	–	+	+	–	–	–	+
16	+	–	–	–	–	–	–	+	+	–	–	–	–	–	+
17	+	–	–	–	–	–	–	+	–	–	+	–	–	–	+
18	–	–	–	–	–	–	+	–	–	–	+	–	–	+	+
19	–	–	–	–	–	–	–	+	+	–	+	–	–	–	+
20	–	–	–	–	–	–	–	+	+	+	–	–	–	–	+
21	+	–	–	–	–	+	–	–	–	–	+	–	–	+	+
22	+	–	–	–	–	+	+	+	–	–	–	–	–	–	+
23	+	–	–	–	–	+	–	+	–	–	+	–	–	–	+
24	+	–	–	–	–	–	+	+	–	–	+	–	–	–	+
25	+	–	–	–	–	–	–	+	–	+	+	–	–	–	+
26	+	–	–	–	–	–	–	+	+	–	+	–	–	–	+
27	+	–	–	–	–	–	–	+	–	–	+	–	–	+	+
28	+	+	+	–	–	–	–	–	–	–	–	–	–	+	+
29	+	–	–	–	–	–	–	+	+	+	+	–	–	–	+
30	+	–	–	–	–	–	–	+	+	–	+	–	–	+	+
31	+	–	–	–	+	+	–	–	–	–	+	–	–	+	+
32	+	+	+	–	–	–	–	–	–	–	–	–	+	+	+
33	+	–	–	–	–	–	–	+	+	+	+	–	–	+	+
34	+	–	–	–	–	+	–	+	–	+	+	–	–	+	+
35	+	–	–	–	–	+	–	+	+	+	+	–	–	–	+
36	+	–	–	+	–	–	–	+	+	+	+	–	–	–	+
37	–	+	+	+	+	–	–	+	+	–	–	–	–	–	+
38	–	–	–	–	+	–	–	+	+	+	+	+	–	–	+
39	+	+	+	–	–	–	+	+	–	–	–	–	+	+	+
40	+	+	+	–	–	–	+	–	–	–	+	–	+	+	+
41	+	–	–	+	–	+	–	+	+	+	–	–	–	+	+
42	+	–	–	–	–	+	–	+	+	+	+	–	–	+	+
43	+	–	–	–	–	–	+	+	–	+	+	+	–	+	+
44	+	–	–	+	+	+	–	+	–	–	+	–	–	+	+
45	+	–	–	+	+	–	–	+	+	–	+	–	–	+	+
46	–	+	+	+	–	–	–	–	+	+	–	+	–	+	+
47	–	+	+	–	–	–	+	+	–	–	+	–	+	+	+
48	–	–	–	+	+	+	–	+	+	+	–	+	–	–	+
49	–	–	–	+	+	–	–	+	+	+	+	+	–	–	+
50	+	+	+	–	–	–	+	+	–	–	+	–	+	+	+
51	+	+	+	+	–	–	+	+	–	–	–	–	+	+	+
52	+	–	–	–	+	–	–	+	+	+	+	+	–	+	+
53	+	–	–	+	+	–	–	+	+	–	+	–	+	+	+
54	+	–	–	+	–	–	–	+	+	+	+	+	–	+	+
55	+	–	–	+	–	+	–	+	+	–	+	+	–	+	+

TABLE 2. (continued)

Race no.	Resistance/susceptibility to *R. secalis* races of barley cultivars: Atlas	Atlas 46	Brier	Calif. 1311	C.I. 2376	C.I. 5831	Hudson	Kitchen	La Mesita	Osiris	Steu-delli	Trebi	Turk	Wisc. Win. × Glab.	Wong[c]
56	−	+	+	+	−	−	−	+	+	−	−	+	+	+	+
57	+	+	+	+	−	−	+	+	−	−	+	−	+	+	+
58	+	−	−	+	+	−	−	+	+	+	+	+	−	+	+
59	+	−	−	+	−	+	−	+	+	+	+	+	−	+	+
60	−	−	−	+	+	+	−	+	+	+	+	+	−	+	+
61	−	+	−	+	+	−	−	+	+	+	+	+	−	+	+
62	+	+	−	+	+	−	−	+	+	+	+	+	−	+	+
63	+	+	−	+	+	+	−	+	+	+	+	+	−	+	+
64	−	+	+	+	+	−	+	+	+	−	+	+	+	−	+
65	−	+	+	+	+	−	−	+	+	+	−	+	+	+	+
66	−	+	+	+	−	−	+	+	+	+	−	+	+	+	+
67	+	+	−	+	+	+	−	+	+	+	+	+	−	+	+
68	−	+	+	+	−	−	+	+	+	+	+	+	+	+	+
69	−	+	+	+	+	−	−	+	+	+	+	+	+	+	+
70	−	+	+	+	+	−	+	+	+	+	−	+	+	+	+
71	−	+	+	+	+	+	+	+	+	+	−	+	+	+	+
72	−	+	+	+	+	−	+	+	+	+	+	+	+	+	+
73	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+
74	+	+	+	+	+	−	+	+	+	+	+	+	+	+	+
75	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

[a]− = Resistant reaction, 0-2 rating.
[b]+ = Susceptible reaction, 3-4 rating.
[c]The cultivar Wong was included as the universal suscept.

TABLE 3. Regional distribution of 175 isolates of 75 races of *Rhynchosporium secalis* in California

California		
Region	County	Races of *R. secalis*
Northern Valley	Butte	1, 40
	Colusa	2(3)[a], 9(4), 10, 17, 40, 74
	Glenn	1, 11, 15, 16, 17, 25(2), 26, 30, 32, 33, 39, 50, 51, 55, 57
	Shasta	9
	Sutter	2(2), 67
Middle Valley	Sacramento	2(2), 9(2), 10, 14, 17, 26, 35, 52, 58
	San Joaquin	2, 5, 9, 18, 26, 31, 45, 53, 54, 72(2), 74
	Solano	9, 52
	Stanislaus	2(4), 4, 7, 20(2), 24, 38, 41, 44, 47, 49, 50, 68, 72
	Tuolumne	2, 9, 48, 60
	Yolo	1, 2, 3, 9(4), 12, 19, 27, 43, 58, 64, 75
Southern Valley	Fresno	62, 65, 70(4), 72(3), 73(3)
	Kern	6, 28, 37, 46, 71, 72
	Kings	30, 65, 72(2), 73(2)
	Madera	8, 15, 21, 22, 25, 29, 34, 36, 56, 63, 66, 72(2), 73(4), 75
	Merced	2, 9, 17, 23, 26, 34, 35, 42(2), 59, 72(3), 73(2), 75
	Tulare	9, 14, 56, 69, 72(3), 73(3)
Coastal Valley	Alameda	2, 9, 61
	Contra Costa	2
	Monterey	2, 13(2), 71
	San Benito	8(2), 71
	San Luis Obispo	40
	Santa Clara	48, 71

[a]Number in parentheses signifies the number of isolates of that race isolated from the particular county.

Previous race differentiation studies.—Schein differentiated eight isolates into seven races, designating them U.S. 1 through U.S. 7, with a differential host range consisting of six differentials and the universal suscept Wong (21). The present set of differentials includes all of those used by Schein so the newly identified races can also be characterized on his set. Theoretically, six differential cultivars may distinguish 64 ($=2^6$) races. The present 75 races are reduced to 17 when characterized on Schein's set of differentials. As many as 12 different races could be grouped under a single race designation. Races 1, 2, 5, 7, 8, 9, 10, 13, 15, 17, 23, and 25 all corresponded to U.S. 8; races 4, 6, 14, 21, 27, 31, and 34 all corresponded to U.S. 6; races 39, 40, 47, and 50 all corresponded to U.S. 9, races 51 and 57 both corresponded to U.S. 4 [U.S. 8 and U.S. 9 were added to Schein's original seven races by Dyck and Schaller (4)]. The heterogeneous nature of theU.S.-type race designation has important implications, especially with regard to breeding programs for scald resistance. For instance, a breeding program based on resistance to isolates of each U.S. race may develop a worthless product if those isolates do not reflect the dominant pathotypes of *R. secalis* existing in the field.

The California race situation can be contrasted to that of Great Britain where Fowler and Owen (5) suggested that only two races of *R. secalis* exist. Their conclusions were supported by Williams and Owen (25) who differentiated only two races in tests involving 122 isolates on a differential set of 12 cultivars. Williams and Owen subsequently reduced their set to three cultivars: Deba Abed, the suscept; Dea, later replaced by Senta (H. Owen, *personal communication*), and Osiris. Race U.K. 1 was described as pathogenic to Deba Abed and race U.K. 2 was described as pathogenic to Deba Abed and Senta. In order to learn more about the California races, 71 isolates representing 35 races were tested on the three British differentials. Six of the possible eight races were differentiated; 21 isolates fitted the U.K. 1 classification, two isolates fitted the U.K. 2 classification, and the remaining 48 isolates fitted into four race classifications that lacked identified British counterparts. Fundamental differences appear to exist between the two different *R. secalis* populations. Deba Abed, the British universal suscept, was resistant to some California isolates. Osiris, resistant to all British isolates, was susceptible to many California isolates. If the California isolates were characterized only on the three British differentials, heterogeneous races would result. Conversely, the three British differentials revealed a certain amount of heterogeneity in the California races themselves. Two races from Group I (races 1 and 8), one race from Group II (race 56), and three races from Group III (races 65, 70, and 71) had representatives in more than one British differentiated race. Since the precision of race separation depends on the number and quality of differentials used, and additional differentials may or may not reveal heterogeneity in a given race, extreme care should be exercised in equating the races described in different populations of the fungus.

Relative effectiveness of the sources of resistance to scald.—The characterization of 175 isolates into races revealed the relative effectiveness, or ineffectiveness, of the different sources of resistance against *R. secalis* as it occurs in California. The cultivar that displayed the most effective resistance, C.I. 5831, was ineffective against 46 isolates, representing 21 races and 26% of the sample (Table 4). Such vulnerability of the best source of resistance in this study brings into question the advisability of seeking specific resistance for the control of scald. The three most ineffective sources of resistance were Steudelli, Kitchen, and Atlas, which were susceptible to 68, 64, and 62%, respectively, of the sample. The salient point here is that Atlas, although susceptible to the great majority of Group I and Group II isolates, exhibited the highest degree of resistance to Group III isolates, being susceptible to only 15% of them. It is obvious that factors other than those previously identified are being expressed in the reaction elicited by Group III isolates.

Re-evaluation of the designated genes for resistance to scald.—Differential cultivars assumed to have genes for resistance in common did not react identically to all of the races, indicating previously undisclosed differences in the genes described or suggesting the presence of previously unidentified genes for resistance. Three examples serve to support this contention. La Mesita and Osiris have the *Rh4* gene in common (4) yet La Mesita was susceptible to 13 races to which Osiris was resistant whereas Osiris was susceptible to five races to which La Mesita was resistant. Brier and Hudson have the *Rh* gene in common (6) yet Brier was susceptible to seven races to which Hudson was resistant whereas Hudson was susceptible to four races to which Brier was resistant. Even though Trebi and California 1311 were originally believed to have both a dominant and a recessive gene in common (17), California 1311 was susceptible to nine races to which Trebi was resistant and Trebi was susceptible to three races to which California 1311 was resistant. Either of the two hypotheses that have been advanced becomes an acceptable resolution to the seemingly paradoxical situation when the methods of identifying genes for resistance and assigning them to more than one resistant cultivar are re-examined in light of the currently disclosed pathogenic variability of *R. secalis*.

Studies on the inheritance of resistance to *R. secalis* have utilized very few races of the fungus, resulting in genes for resistance going undetected in the absence of the

TABLE 4. Susceptible reactions of barley cultivars to *Rhynchosporium secalis* in California

Barley cultivar	Number of races	Number of isolates	Percent of sample
Steudelli	50	119	68
Kitchen	54	112	64
Atlas	50	109	62
Wisconsin Winter × Glabron	46	96	55
La Mesita	43	93	53
Osiris	35	84	48
California 1311	32	74	42
Trebi	26	69	39
Atlas 46	26	69	39
C.I. 2376	24	66	38
Brier	22	65	37
Turk	20	63	36
Hudson	19	60	34
C.I. 5831	21	46	26

races with the pathogenicity necessary to identify them, and in closely linked genes and alleles of the same gene not being differentiated from each other in the absence of the races that would make such differentiation possible. Workers have characteristically extended the range of identified genes to other resistant cultivars on the basis of resistant F_2 populations from crosses between resistant cultivars (1, 17, 22, 24). Although such F_2 analysis may be sufficient to determine whether the genes involved are independent, the possibility of closely linked genes or multiple alleles cannot be eliminated. In view of the extent of the pathogenic variability of *R. secalis,* it must be emphasized that the assignment of a gene for resistance to cultivars other than the one in which it was first identified, unless the cultivars are directly related, can only be tentative.

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