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Differentiation of Virulence in *Rhynchosporium secalis* in the Hokuriku District and Sources of Resistance to the Pathogen

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Summary

Variation of virulence in *Rhynchosporium secalis* (Oud.) Davis distributed in the Hokuriku district was investigated by seedling infection test using 14 differential barley cultivars. A total of 38 cultures were isolated from the infected leaves of Minorimugi, a leading variety in the Hokuriku area, in 1992, 1993 and 1995 from various locations. The 38 isolates showed a complex variation in the virulence pattern and could be classified into 36 different pathotypes according to the virulence spectra to the differentials. Based on the cluster analysis of the reactions of 14 differentials, a geographical cline for virulence was suggested; the isolates from the southern part of the district (Fukui, Ishikawa and Toyama) were more virulent to the differentials than those from the northern part (Yamagata and Niigata). This fact suggests that the virulence of the fungus is conditioned by not only race-specific gene (s) but also by some genes with interactions such as additive or complementary effects. Among the 14 differentials, Brier, Turk and Osiris were highly resistant to the 38 isolates. It appeared that the resistance of these 3 cultivars was controlled by different gene (s) from *Rh*, *Rh*^{4}, *Rh5, rh6 or Rh10*, since other differentials with the same genotype were attacked by many of the isolates. The 17 cultivars, which had been already selected for the resistance to the Niigata isolate, were tested by 32 of the 38 isolates above mentioned, and 3 cultivars, Turkey 22, Trukey 208 and Carre 26, were confirmed to be highly resistant to all the isolates. It was concluded that these cultivars including Brier, Turk and Osiris are highly suitable materials for breeding for resistance to scald in the Hokuriku district.

Key Words : *Hordeum vulgare, Rhynchosporium secalis*, barley, genetic resources, resistance, scald, virulence spectrum.

Introduction

Scald which is caused by *Rhynchosporium secalis* (Oud.) Davis is an important disease of barley (*Hordeum vulgare* L.), and is distributed worldwide. The disease can be controlled most effectively by growing resistant cultivars. A total of 13 genes for resistance to scald have been identified (Søgaard and von Wettstein-Knowles 1987, Abbott *et al.* 1992). It is necessary to detect as many available sources of resistance as possible for controlling the disease, and we have already selected 17 cultivars from 1315 world barley accessions by both inoculation tests under field conditions and seedling tests using a culture isolated from Niigata (Fukuyama and Takeda 1992).

For the introduction of such resistance genes into desirable but susceptible cultivars, breeders are confronted with the variability of the pathogen. It is generally recongnized that *R. secalis* displays a large variation in virulence. After the pioneering work of Sarasola and Campi (1947), variation in virulence was reported in many countries (Schein 1958, Owen 1963, Brown 1985, 1990, Ceoloni 1980, Jackson and Webster 1976, Hansen and Magnus 1973, Dodoff 1963, Ali and Boyd 1973, Ali *et al.* 1976, Williams and Owen 1973, Houston and Ashworth 1957). In Japan, Kajiwara and Iwata (1963) recognized 10 races, J1 to J10, within 37 isolates collected from various locations. Thereafter, however, few reports recorded a variation in virulence in our country.

The present study aimed at determining the degree of variation in virulence in *R. secalis* distributed in the Hokuriku district and to assess the suitability of 17 selected resistant cultivars.

Materials and Methods

Leaves of the cultivar Minorimugi infected with *R. secalis* were collected from a number of sites in the Hokuriku district in 1992, 1993 and 1995 (Table 1 and Fig. 1). A single spore aseptically isolated from susceptible lesions was cultured on potato-agar medium with 2% sucrose at 18 ℃ . To avoid the change of virulence, the cultures were used for the inoculation test immediately after the isolation without sub-culture each year. The 14 differential cultivars used to detect the virulence spectrum are listed in Table 2 with their resistance genes to scald. Methods for preparing the inoculum and growing inoculated plants followed those in the previous report (Fukuyama and Takeda 1992). At 1~2 leaf stage for the differentials and Minorimugi as susceptible standard, a spore suspension adjusted to a density of $5x10^5$/m*l* was sprayed on the leaves. The inoculated plants were kept for 48 hours at a high humidity with a humidifier, and transferred to a glass house where the temperature ranged from 15 to 25 ℃ . Three weeks after the inoculation, the reaction of each cultivar was assessed according to the following scale: R; highly

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Table 1. Isolates of *R. secalis* collected from the Hokuriku district

No.	Collection site	Date of collection	Name of isolate
1	Kawanishi, Yamagata	1992,5,21	92Y1
2	Nishikawa, Niigata	1992,5,28	92N1
3	Shirone, Niigata	1992,4,30	92N2
4	Nagaoka, Niigata	do.	92N3-1
5	do.	do.	92N3-2
6	Joetsu, Niigata	do.	92N4-1
7	do.	do.	92N4-2
8	Nyuzen, Toyama	1992,4,29	92T1
9	Kurobe, Toyama	do.	92T2
10	Toyama, Toyama	do.	92T3
11	Fukuoka, Toyama	do.	92T4-1
12	do.	do.	92T4-2
13	Unoke, Ishikawa	1992,4,30	92I1-1
14	do.	do.	92I1-2
15	Komatsu, Ishikawa	do.	92I2
16	Kanazu, Fukui	1992,4,29	92F1
17	Maruoka, Fukui	do.	92F2-1
18	do.	do.	92F2-2
19	Shirone, Niigata	1993,4,30	93N1-1
20	do.	do.	93N1-2
21	Akatsuka, Niigata	do.	93N2
22	Muramatsu, Niigata	1993,4,29	93N3
23	Namerikawa, Toyama	1993,5, 3	93T1
24	Toyama, Toyama	do.	93T2-1
25	do.	do.	93T2-2
26	Kanbayashi, Niigata	1995,5,13	95N1
27	Shirone, Niigata	1995,5,16	95N2
28	Muramatsu, Niigata	do.	95N3
29	Koshiji, Niigata	1995,5,13	95N4
30	Mishima, Niigata	do.	95N5-1
31	do.	do.	95N5-2
32	Kubiki, Niigata	do.	95N6
33	Sanwa, Niigata	do.	95N7
34	Fukumitsu, Toyama	1995,5,20	95T1-1
35	do.	do.	95T1-2
36	Suzaka, Nagano	1995,5	95NG1-1
37	do.	do.	95NG1-2
38	do.	do.	95NG1-3

Table 2. Differential genotypes used in this study and their resistance to scald

Genotype (abbreviation)	Resistance	Genotype (abbreviation)	Resistance
Abyssinian (Ab)	*Rh9*[2)]	Kitchin (Ki)	*Rh9*[2)]
Atlas (At)	*Rh2*[3)]	La Mesita (LM)	*Rh*^{4} *Rh10*[1)]
Atlas 46 (A4)	*Rh Rh2*[1)]	Modoc (Mo)	*Rh*^{2} *rh6*[1)]
Bey (Be)	*Rh Rh5 rh6*[4)]	Nigrinudum (Ni)	*rh8*[1)]
Brier (Br)	*Rh rh6*[1)]	Osiris (Os)	*Rh*^{4} *rh6 Rh10*[1)]
CI 3515 (C3)	*Rh*^{4} *Rh10*[1)]	Rivale (Ri)	*Rh Rh5 rh6*[4)]
CI 8256 (C8)	*Rh*^{4} *Rh10*[1)]	Turk (Tu)	*Rh Rh5 rh6*[1)]

[1)] Habgood and Hayes (1971) [2)] Baker and Larter (1963)
[3)] Dyck and Schaller (1961) [4)] Wells and Skoropad (1963), who could not obtain any segregants in F_2 in the cross with Turk.

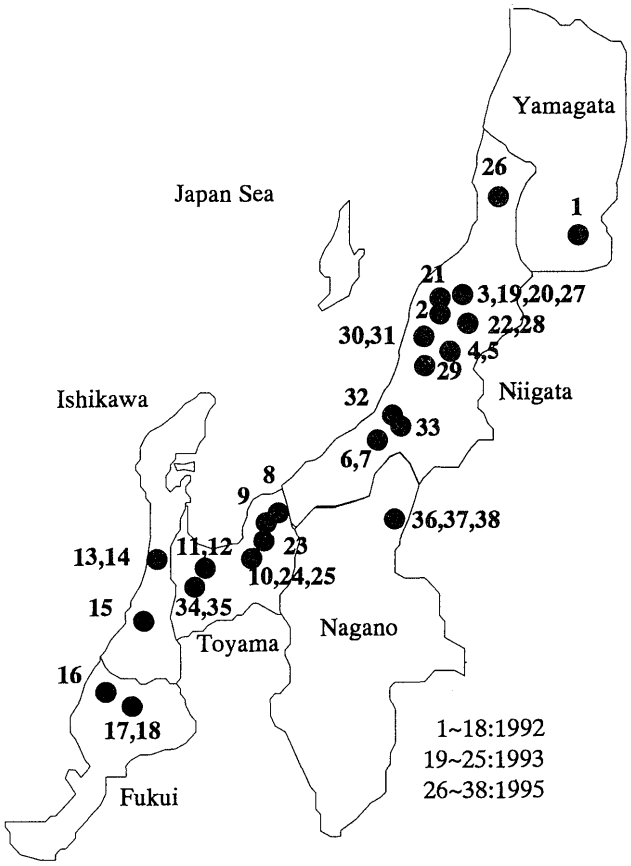

Fig. 1. Collection sites of *R. secalis* in the Hokuriku district. Numerals are cited from Table 1.

resistant without visible lesions or with lesions smaller than 5mm in diameter, with necrosis, M; moderately resistant with somewhat larger lesions from 5 to 10mm with necrosis, and S; susceptible with large blue-gray lesions more than 10mm, or coalescing lesions.

In order to compare the variation of virulence spectra among the 38 isolates, R, M and S reactions of the 14 differentials were given scores of 0, 1 and 2, respectively, and cluster analysis was conducted using a software package SAS6.09 UNIX version by the average linkage method.

For the screening of genetic resources resistant to various pathotypes of *R. secalis* distributed in the Hokuriku district, 17 cultivars, which had been already selected by Fukuyama and Takeda (1992), were also tested by the same as that described above.

Results

Variation in the virulence pattern of the isolates collected from the Hokuriku district

A total of 38 fungus strains could be isolated from the affected leaves within 3 years. The reactions of the 14 differential cultivars to the isolates are shown in Table 3. Highly complex variability in the virulence was detected

Table 3. Reactions of 14 differential genotypes to 38 isolates of *R. secalis* collected from the Hokuriku district

Isolate	Differential genotype [1]														No. of S type	Minori mugi
	Br	Os	Tu	Be	Ri	C3	At	C8	A4	Mo	LM	Ab	Ni	Ki		
92Y1	R[2]	R	R	R	R	R	S	R	R	R	R	R	R	R	1	S
92N1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	0	S
92N2	R	R	R	R	R	M	R	R	R	S	M	R	M	R	1	S
92N3-1	R	R	R	R	R	R	R	R	M	R	R	M	M	S	1	S
92N3-2	R	R	R	R	R	R	R	R	R	M	S	M	S	S	3	S
92N4-1	R	R	R	R	R	R	R	R	R	R	R	S	R	R	1	S
92N4-2	R	R	R	R	R	R	R	R	R	R	S	S	S	S	4	S
92T1	R	R	R	M	R	S	R	M	S	S	S	S	S	S	7	S
92T2	R	R	R	R	R	R	R	R	M	S	S	M	S	S	4	S
92T3	R	R	R	R	R	R	R	S	S	S	S	M	S	S	6	S
92T4-1	R	R	R	M	R	R	R	R	S	S	S	S	S	S	6	S
92T4-2	R	R	R	R	S	R	R	R	M	R	R	R	S	R	2	S
92I1-1	R	R	R	M	R	R	R	R	S	S	S	M	S	S	5	S
92I1-2	R	R	R	R	R	R	R	R	R	R	R	M	S	S	2	S
92I2	R	R	R	M	R	M	R	M	M	S	S	M	S	S	4	S
92F1	M	R	R	R	R	R	R	R	R	M	R	S	M	R	1	S
92F2-1	R	R	R	M	R	R	R	S	M	S	S	S	S	S	6	S
92F2-2	R	R	R	M	R	R	R	S	M	S	S	S	S	S	6	S
93N1-1	R	R	R	M	R	R	R	R	R	M	R	R	R	M	0	S
93N1-2	R	R	R	R	M	R	R	R	R	M	R	M	M	R	0	S
93N2	R	R	R	R	R	R	R	R	R	R	R	R	R	R	0	S
93N3	R	R	R	R	R	R	R	R	R	R	R	R	M	R	0	S
93T1	R	R	R	R	R	R	R	M	S	M	R	R	S	S	3	S
93T2-1	R	R	R	R	R	R	R	R	R	R	S	M	S	S	3	S
93T2-2	R	R	R	M	R	R	R	S	M	S	S	M	S	M	4	S
95N1	R	R	R	R	R	R	R	M	M	R	M	S	R	M	1	S
95N2	M	R	R	R	R	R	S	R	M	R	M	R	R	R	1	S
95N3	R	M	R	R	R	R	R	R	M	M	M	S	S	S	3	S
95N4	R	R	M	R	R	R	S	R	M	R	R	M	S	S	3	S
95N5-1	R	R	R	R	R	S	R	M	R	R	R	S	R	S	3	S
95N5-2	R	R	R	R	M	S	R	S	R	R	R	S	S	S	5	S
95N6	R	R	R	R	R	R	M	R	S	S	R	S	S	S	5	S
95N7	R	M	R	M	R	R	S	R	S	S	R	S	S	S	6	S
95T1-1	R	R	R	R	S	R	S	R	S	R	M	S	M	S	5	S
95T1-2	R	R	R	R	R	R	R	R	R	R	R	M	R	R	0	S
95NG1-1	R	R	R	S	R	S	M	S	S	S	S	S	S	S	9	S
95NG1-2	R	R	R	R	R	R	R	R	R	R	R	R	R	M	0	S
95NG1-3	R	R	R	R	R	R	R	M	R	R	S	S	S	S	4	S
No. of S type	0	0	0	1	2	4	5	6	9	13	14	16	22	23		38

[1] Abbreviations are shown in Table 2.

[2] R, M and S refer to highly resistant, moderately resistant and susceptible, respectively.

among the isolates. No isolates induced an identical reaction in the differentials except for 2 pairs, 92N1/93N2 and 92F2-1/92F2-2. Though 9 pairs of isolates (92N3-1,-2; 92N4-1,-2; 92T4-1,-2; 92I1-1,-2; 93N1-1,-2; 93T2-1,-2; 95N5-1,-2; 95T1-1,-2; and 95NG1-1,-2,-3) were collected in the same year and site, all showed different virulence patterns. Based

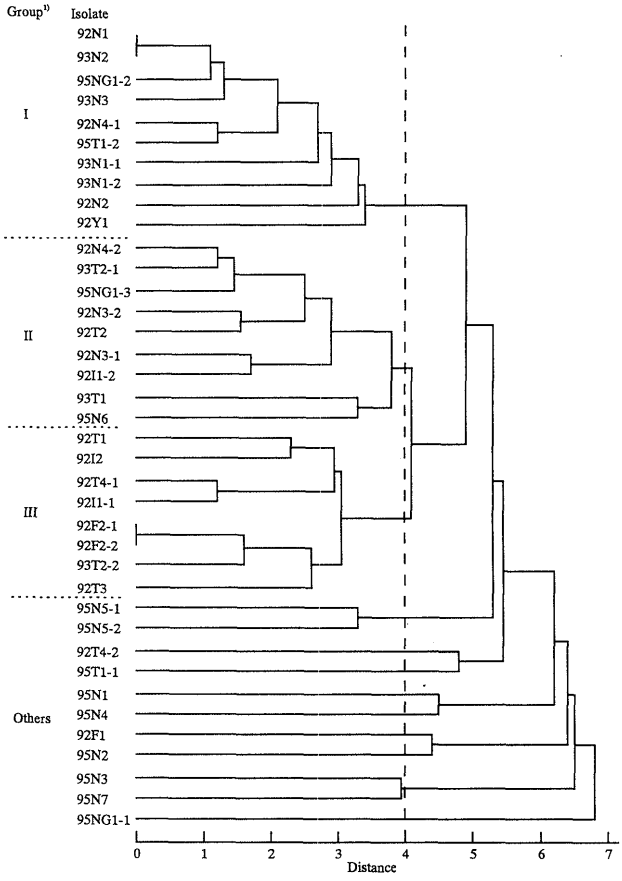

Fig. 2. Dendrogram of 38 isolates based on virulence spectrum to 14 differential genotypes.
[1] See text.

on the numbers of susceptible reactions in the 14 differentials (Table 3), 95NG1-1 displayed the highest virulence, attacking 9 differentials, followed by 92T1, with a virulence range up to 7 differentials. On the other hand, 7 isolates could not affect any of the differentials, and they predominated among the Niigate isolates collected in 1993.

To analyze in more detail the variation in the virulence spectrum to the 14 differentials among the 38 isolates, cluster analysis was conducted and the resulting dendrogram is shown in Fig. 2. When the clusters were tentatively divided at a distance of 4, it became to distinguish 4 or more groups. Group I consisted of 10 isolates which were dominant in Niigata, and their virulence range to the 14 differentials was as narrow as 0 to 1 with an average of 0.4 as shown in Table 3. Group II consisted of 9 isolates with a virulence range from 1 to 5 differentials (average, 3.2). Group III consisted of 8 isolates from Toyama, Ishikawa and Fukui with a range of virulence from 4 to 7 (average, 5.5), which was wider than that of Groups I and II . The remaining isolates were widely separated from Groups, I , II and III , and from each other. They were mainly collected in 1995 and were virulent to 1 to 9 differentials (average, 3.7). Based on cluster analysis, it was suggested that the virulence spectra were geographically different, the

Table 4. Reactions of 17 accessions selected for scald resistance to 32 isolates inthe Hokuriku district

Isolate	Accession[1]																	No. of S type
	T008	B024	T070	E223	E226	T030	T086	E249	T090	I025	E254	E244	E023	E140	E233	E134	T031	
92Y1	R[2]	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	S	2
92N1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	0
92N2	R	R	R	R	R	R	R	R	R	M	R	R	R	R	S	S	M	2
92N3-1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S	S	3
92N3-2	R	R	R	R	R	R	R	S	R	M	R	S	R	R	R	M	R	2
92N4-1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	M	R	S	1
92N4-2	R	R	R	R	R	R	R	R	R	R	R	R	S	R	S	M	M	2
92T1	R	R	R	R	R	R	R	R	R	R	R	R	S	M	S	S	S	4
92T2	R	R	R	R	R	R	M	R	M	R	R	R	M	R	S	S	S	3
92T3	R	R	R	R	R	R	R	R	R	M	R	R	S	S	S	S	S	5
92T4-1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S	S	3
92T4-2	R	R	R	R	R	R	R	R	M	R	R	R	R	R	S	S	S	3
92I1-1	R	R	R	R	R	R	S	R	R	M	R	R	R	S	S	R	M	3
92I1-2	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	M	S	2
92I2	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S	S	S	4
92F1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	M	M	S	1
92F2-1	R	R	R	R	R	R	R	M	R	S	R	R	R	S	S	S	S	5
92F2-2	R	R	R	R	R	R	R	M	M	M	R	M	R	R	R	R	M	0
93N1-1	R	R	R	R	R	R	R	R	R	R	R	R	M	M	M	S	S	2
93N1-2	R	R	R	M	M	R	R	R	M	M	R	M	M	M	M	M	S	1
93N2	R	R	R	R	M	R	R	R	R	M	M	M	M	M	M	M	S	1
93N3	R	R	R	R	R	R	R	R	R	R	R	R	M	R	R	S	S	2
93T1	R	R	R	M	M	M	R	M	S	R	S	M	M	R	M	S	M	3
93T2-1	R	R	R	R	R	R	R	R	R	R	R	R	M	M	S	S	S	3
93T2-2	R	R	R	M	M	R	R	R	M	R	R	R	M	R	M	M	M	0
95T1-1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	1
95NG1-2	R	R	R	M	M	R	R	R	R	R	R	R	R	R	M	R	S	1
95NG1-1	R	R	R	R	M	S	R	R	R	R	R	S	R	R	S	S	R	4
95N3	R	R	R	R	R	R	R	M	R	R	R	M	M	R	S	S	S	3
95N7	R	R	R	R	M	R	R	M	R	R	M	M	M	M	S	S	M	2
95NG1-3	R	R	R	R	R	R	R	R	R	M	S	R	R	R	M	S	S	3
95N5-1	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S	2
No. of S type	0	0	0	0	0	1	1	1	1	1	2	2	3	4	17	18	22	

[1] Accession number given at Barley Germplasm Center, Okayama University.
[2] R, M and S refer to highly resistant, moderately resistant and susceptible, respectively.

isolates from the southern part of the district (Fukui, Ishikawa and Toyama) being more virulent to the differentials than those from the northern part (Yamagata and Niigata).

Sources of resistance to the isolates collected in the Hokuriku district

As indicated in Table 3, 3 differentials, Brier, Turk and Osiris, were highly resistant to the 38 isolates.

The 17 cultivars selected by Fukuyama and Takeda (1992) were tested using 32 of the 38 isolates, and the results are shown in Table 4. Three cultivars, Turkey 22 (accession number T008 given at the Barley Germplasm Center, Okayama University), Turkey 208 (T070) and Carre 26 (B024) showed a high level of resistance to all the isolates inoculated. Two Ethiopian cultivars, Debre Zeit 22 (E223) and Debre Zeit 31 (E226) displayed an intermediate type of resistance to 4 and 7 isolates, respectively, and were not susceptible to any of the remaining isolates. On the other hand, 3 cultivars, Addis Ababa 4 (E233), Ethiopia 402 (E134) and Turkey 91 (T031) were susceptible to 17 or more isolates. The remaining 9 cultivars were affected by 1 to 4 isolates.

Table 4 also indicates that the 32 isolates showed different virulence spectra except for 92N3-1 and 92T4-1, and the isolates 92T3 and 92F2-1 were highly virulent. Two pairs of isolates, 92N1/93N2 and 92F2-1/92F2-2 estimated to be identical based on the inoculation test to 14 differentials gave a very different reaction in the 17 cultivars. Therefore, the remarkable variation in virulence was reconfirmed in this case.

Discussion

The present investigation revealed that the 38 isolates of *R. secalis* collected in the Hokuriku district could be classified into 36 different pathotypes through inoculation tests using 14 differential barley genotypes. Since the genotype for resistance to the Hokuriku isolates among the 14 differentials has not been identified, the 38 isolates could not be referred to any race but were referred to pathotypes following the suggestion by Ali *et al.* (1976). Although our materials were sampled from a restricted area, the Hokuriku district, the percentage of unique isolates indicating different reactions in a set of differentials was as high as 94.7%, a value considerably higher than 26% within the USA isolates tested by Jackson and Webster (1976), or 51-54% by Crandall (1987), although the differentials were not completely identical with those in the USA. It was also found in this study that the virulence pattern was very different among the isolates collected at the same site. This is not surprising, because Brown (1990) reported that spores from the same lesion, as well as spores from different lesions collected at the same location showed a highly variable virulence and could be classified into different pathotypes.

McDonald (1989) reviewed the variability of plant pathogens and pointed out 5 factors affecting genetic change in pathogen population; mutation, genetic drift, recombination, gene flow and selection. McDermott *et al.* (1989) and McDonald *et al.* (1989) reported that the effects of genetic drift and recombination were negli-

gible on the variability of pathogen populations based on the genetic analysis of virulence, isozymes, colony colors and ribosomal DNA RFLPs in a number of *R. secalis* isolates sampled in composite cross II (CC II) and CCV, and they concluded that the genetic structure of both the host and pathogen populations was influenced by coevolutionary processes featuring interactions among loci affecting many different traits, including interactions among host resistance genes and pathogen virulence genes. Although we have no evidence for the origin of the highly complex variation in virulence observed in *R. secalis* distributed in the Hokuriku district, it should be emphasized here that a single genotype, Minorimugi, had been cultivated in this area for more than 10 years. Therefore, factors other than cultivar selection should be considered for the variability of virulence in the Hokuriku district.

Kajiwara and Iwata (1963) classified 37 Japanese isolates of *R. secalis* into 10 groups, J1 to J10, 5 of which, J3, 4, 5, 7 and 9, were distributed in the Hokuriku district (Fukui, Ishikawa, Toyama and Yamagata). They concluded that no distinct geographical differentiation occurred in the Japanese isolates. However, in the present investigation a geographical cline for virulence was assumed based on the cluster analysis; isolates from the southern part of the Hokuriku district (Fukui, Ishikawa and Toyama) showed a higher virulence compared with those from the northern part of the district (Niigata and Yamagata). These results suggest that the virulence of *R. secalis* is controlled by not only "race"-specific gene (s) but also gene (s) with interactions including additive or complementary effects, and/or that the pathogens retain different levels of adaptability to environmental factors. With regard to the mechanism of pathogenic differentiation of the fungus in the Hokuriku district, further studies should be conducted.

Among the 14 cultivars used for differentials, Brier, Turk and Osiris did not show any susceptibility to the 38 isolates. The genotypes for scald resistance of these 3 cultivars were *Rhrh6, RhRh5rh6* and $Rh^4rh6Rh10$, respectively (Habgood and Hayes 1971, Baker and Larter 1963). The commonly involved gene, *rh6,* also occurs in Modoc (Rh^2rh6), which was susceptible to 13 of 38 isolates. Therefore, *rh6* was not effective for development of resistance in the Hokuriku isolates. Likewise, the genes, *Rh,* Rh^4 and *Rh10*, were considered to be ineffective, since Atlas 46 (*RhRh2*), Bey and Rivale (both *Rh*), CI3515, CI8256 and La Mesita (Rh^4Rh10) were all susceptible to many isolates. Bey and Rivale with the same genotype as Turk (Wells and Skoropad 1963) showed a critical susceptibility to some of the isolates. Therefore, it is likely that Brier, Turk and Osiris harbour gene (s) other than those which have already been identified.

Although some of the 17 cultivars selected by Fukuyama and Takeda (1992) were found to be susceptible to many isolates in the present study, Turkey 22 (T008), Turkey 208 (T070) and Carre 26 (B024) were highly resistant to all the Hokuriku isolates. Thus, these cultivars could be used as suitable sources for resistance breeding. Genetical analyses of these cultivars are under way using the hybrids with susceptible cultivar, Minorimugi. It should be mentioned here that Carre 26 and Turkey 22 were attacked by the pathogen in England (Gymer, personal communication). Therefore, a larger number of resistance genes should be identified, and also gene pyramiding should be applied for resistance breeding.

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Literature Cited

Abbott, D.C., A.H.D. Brown and J.J. Burdon (1992) Genes for scald resistance from wild barley (*Hordeum vulgare* ssp. *spontaneum*) and their linkage to isozyme markers. Euphytica 61:225-231.

Ali, S.M. and W.J.R. Boyd (1973) Host range and physiologic specialization in *Rhynchosporium secalis*. Aust. J. agric. Res. 25:21-31.

——, A. H. Mayfield and B.G. Clare (1976) Pathogenicity of 203 isolates of *Rhynchosporium secalis* on 21 barley cultivars. Physiol. Pl. Pathol. 9:135-143.

Baker, R.J. and E.N. Larter (1963) The inheritance of scald resistance in barley. Can. J. Genet. Cytol. 5:445-449.

Brown, J.S. (1985) Pathogenic variation among isolates of *Rhynchosporium secalis* from cultivated barley growing in Victoria, Australia. Euphytica 34:129-133.

——(1990) Pathogenic variation among isolates of *Rhynchosporium secalis* from barley grass growing in South Eastern Australia. Euphytica 50:81-89.

Ceoloni, C. (1980) Race differentiation and search for sources of resistance to *Rhynchosporium secalis* in barley in Italy. Euphytica 29:547-553.

Crandall, B. (1987) Differentiation and distribution of *Rhynchosporium secalis* on composite cross populations and pureline cultivars in California. PhD thesis. Univ. Calif. Davis: 1-122. (cited from Annu. Rev. Phytopathol. 27:77-94).

Dodoff, D.N. (1963) Immunological studies on barley scald—*Rhynchosporium secalis.* Bulletin of the Institute of Plant Protection, Leningrad 5:71-85. (cited from Rev. appl. Mycology 43:1305).

Dyck, P.L. and C.W. Schaller (1961) Inheritance of resistance in barley to several physiologic races of the scald fungus. Can. J. Genet. Cytol. 3:153-164.

Fukuyama, T. and H. Takeda (1992) Survey of resistance to

scald in world collections of cultivated barley. Japan. J. Breed. 42:761－768.

Habgood, R.M. and J.D. Hayes (1971) The inheritance of resistance to *Rhynchosporium secalis* in barley. Heredity 27:25－37.

Hansen, L.R. and H.A. Magnus (1973) Virulence spectrum of *Rhynchosporium secalis* in Norway and sources of resistance in barley. Phytopath. Z. 76:303－313.

Houston, B.R. and L.J. Ashworth (1957) Newly determined races of the barley scald fungus in California. Phytopathology 47:525.

Jackson, L.F. and R.K. Webster (1976) Race differentiation, distribution, and frequency of *Rhynchosporium secalis* in California. Phytopathology 66:719－725.

Kajiwara, T. and Y. Iwata (1963) Studies on the strains of the barley scald fungus, *Rhynchosporium secalis* (Oud.) Davis. Bull. Natn. Inst. Agric. Sci. Ser. C 15:1－72.

McDermott, J. M., B.A. McDonald, R.W. Allard and R.K. Webster (1989) Genetic variability for pathogenicity, isozyme, ribosomal DNA and colony color variants in populations of *Rhynchosporium secalis.* Genetics 122: 561－565.

McDonald, B.A. (1989) The population biology of host－pathogen interactions. Annu. Rev. Phytopathol. 27:77－94.

——, McDermott, J. M., R.W. Allard and R.K. Webster (1989) Coevolution of host and pathogen populations in the *Hordeum vulgare*－*Rhynchosporium secalis* pathosystem. Proc. Natl. Acad. Sci. USA 86:3924－3927.

Owen, H. (1963) Physiologic races of *Rhynchosporium secalis* on cultivated barley. Trans. Brit. mycol. Soc 46:604－608.

Sarasola, J.A. and M.D. Campi (1947) Reaccion de algunas cebadas con respectro a *Rhynchosporium secalis* en Argentina. Revista de Investigaciones Agropecuarias 1:243－260. (cited from Rev. appl. Mycology 28:60).

Schein, R.B. (1958) Pathogenic specialization in *Rhynchosporium secalis.* Phytopathology 48:477－480.

Søgaard, B. and P. von Wettstein－Knowles (1987) Barley: Genes and chromosomes. Carlsberg Res. Comm. 52:123－196.

Wells, S.A. and W.P. Skoropad (1963) Inheritance of reaction to *Rhynchosporium secalis* in barley. Can J. Pl. Sci. 43:184－187.

Williams, R. J. and H. Owen (1973) Physiologic races of *Rhynchosporium secalis* on barley in Britain. Trans. Br. mycol. Soc. 60:223－234.