Evolutionary Response of Barley Composite Cross II to *Rhynchosporium secalis* Analyzed by Pathogenic Complexity and by Gene-by-Race Relationships

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ABSTRACT

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Evolutionary changes in host-parasite interactions between barley composite cross II (CCII) and 61 races of *Rhynchosporium secalis* were analyzed in terms of pathogenic "complexity" and in terms of specific gene-by-race resistance-pathogenicity reactions. Races identified as complex by a differential set of host cultivars were, in general, able to infect more of the 28 parents of CCII than simple races and simple races were more often countered by the development of genetic resistance in the host population than were complex races. There were, however, many exceptions and it was concluded that pathogenic complexity provides a tenuous base for the analysis of host-pathogen interactions at the population level. Analysis of gene-by-race relationships showed that increases in the frequency of alleles for resistance occurred for each of the five genes known to govern disease reaction in CCII. Patterns of change in allelic frequencies indicated that selection favored alleles for resistance in seasons when scald disease was prevalent but that resistance alleles were detrimental to reproductive capacity in seasons that were unfavorable for scald. The results also suggested that many gene-by-race interactions, in addition to the five that were studied, affect evolutionary response in the CCII-*R. secalis* host-pathogen system. Studies required for a more comprehensive gene-by-race analysis of evolutionary change are discussed.

Additional key words: barley scald, disease resistance, pathogenic variability, selection.

The fungus *Rhynchosporium secalis* (Oud.) Davis causes a foliar disease (scald) on barley (*Hordeum vulgare* L.) in places where the growing season is cool and humid. Workers in several countries have studied genetic variability in the pathogenicity of this fungus and the corresponding genetic variability for resistance in barley (2,9,10,12,13). A series of such studies has been conducted by our group at Davis, CA. Jackson and Webster (13) tested cultivars with known specific genes for resistance against 175 isolates of the fungus as it occurs throughout California and ultimately selected a differential set of 14 cultivars that separated the 175 isolates into 75 distinct races. The pathogenicity of these races remains stable when the races are kept in isolation, whether in culture or on barley plants (13,14); the races thus represent pathogen phenotypes governed by genotypes differing in pathogenicity. However, when barley plants are infected by a mixture of races under either natural or experimental conditions, new races quickly appear (14). *R. secalis* (9,14), an imperfect fungus, thus has effective mechanisms for the production of new genotypes for pathogenicity; however, it is not known whether these mechanisms involve an unobserved sexual cycle, parasexuality, spontaneous mutation, or other phenomena.

In sexual organisms, the existence of genes for pathogenicity in the pathogen and corresponding genes for resistance in the host can be demonstrated by formal genetic studies (6,7). Such studies are, however, formidable in practice, and in few, if any, cases have gene-for-gene relationships been analyzed sufficiently completely to provide an adequate basis for field studies. With asexual pathogens, the difficulties are greater because standard Mendelian analyses are precluded and the existence of genes for pathogenicity can only be inferred by assuming that a gene-for-gene correspondence exists with the host. However, data obtained by determining the ability of isolates of pathogens to cause disease on "differential" host strains with unknown, or incompletely known, genotypes for resistance have been suggested as a means for quantification of the extent of variability in pathogenicity and resistance in terms of the degree of "complexity" of races of the pathogen and complexity of strains of the host. In the literature of disease resistance in plants (5,17,18,20), a complex race of a variable pathogen is defined as one capable of producing disease on a larger number of differential strains of the host than a "simple" race of the same species; i.e., complex races are pathogenic to more differential strains of the host, each resistant to different isolates of the pathogen, and/or they are pathogenic to host strains that are resistant to more isolates of the pathogen than a simple host strain. Thus, under the gene-for-gene hypothesis, relative complexity rankings are assumed to provide a measure of the number of genes for pathogenicity carried by different races of the pathogen and the number of genes for resistance carried by different strains of the host. A complex host strain is resistant to more races of the pathogen and/or to more complex races than a simple host strain. Complexity of both pathogen and host is consequently relative. Each set of differential strains of the host is expected to produce a different characterization of the complexity of a given set of isolates of a pathogen, depending on the number of pathogenicity genotypes in the pathogen set. Similarly, each set of isolates of the pathogen is expected to produce a different characterization of the complexity of a set of differentials of the host, depending on the number of different genotypes for resistance in the host set.

The 75 races of *R. secalis* identified by Jackson and Webster (13) encompass a broad sample of the diversity in pathogenicity of this species in a major barley-growing region. The 75 races, which were numbered in order from simplest to most complex based on the set of 14 differential cultivars used to delimit them, include a race unable to infect any of the differentials (race 1) and a race able to infect all of them (race 75). The composite crosses of barley, developed by intercrossing cultivars from diverse barley-growing regions of the world and propagating the resulting hybrid materials thereafter in large plots under agricultural conditions without conscious selection, represent a broad sample of genetic diversity for reaction to scald disease. Seed stocks from various generations of the composite crosses have been saved and are available for study of evolutionary changes over sequences of generations. The barley composite cross-*R. secalis* host-pathogen system thus provides materials that appear to be useful for the study of evolving host-pathogen interactions at the population level.

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Jackson et al (12) determined frequencies of resistant individuals to four races of *R. secalis* in early, intermediate, and late generations of three barley composite crosses (CC), CCII, CCV, and CCXXI, which differ in parentage and method of synthesis. These investigators found that the frequency of plants resistant to races 40 and 61 (races of intermediate complexity) and 74 (a highly complex race) increased from early to late generations in CCII but that little if any change occurred in the frequency of plants resistant to race 72 (also a complex race). This finding, which has been repeated in two subsequent studies of CCII tested against races 40, 61, 72 and 74 (16,19), indicates that CCII is able to counter both simple and complex races through genetic resistance. The studies reported in this paper were undertaken to determine the pathogenicity of California races of *R. secalis* on the parents and on genotypes drawn from various generations of CCII and also the resistance of different host genotypes to individual races of the pathogen. Evolutionary changes in CCII were analyzed in two ways: in terms of pathogenic complexity of the races of *R. secalis* relative to the complexity of the resistance genotypes in the host and in terms of resistance to individual races of the pathogen produced by specific known genes in the host population, i.e., in terms of gene-by-race relationships.

MATERIALS AND METHODS

The materials used in this study were 61 of the 75 races of *R. secalis* identified by Jackson and Webster (13), the 28 parents from which CCII was synthesized (11), and samples of seeds drawn at random from 19 different generations of CCII.

The races used in this study were taken from the collection maintained by the Department of Plant Pathology, University of California, Davis. The 61 races chosen for study, which are listed in Table 1, included races that varied from one race able to infect only one of the 14 cultivars in the differential set of Jackson and Webster (13) to one race that was able to infect all 14 differentials; thus the sample included races from racial complexity classes I–XIV (race 1, complexity class 0, is no longer available). The numbers of races to which the 14 members of the differential set are resistant are also given in Table 1; the members of the set varied from one differential that was resistant to 21 races (host complexity class I) to one that resisted 56 races (host complexity class XIV). CCII, which was synthesized in 1929 by mixing equal numbers of F_1 seeds obtained by crossing its 28 parents in all 378 possible pair-wise combinations (11), has subsequently been grown annually at Davis, CA, for more than 50 generations. Seeds for the present study were taken from reserve stocks maintained by the Department of Genetics, University of California, Davis.

Three experiments were performed. In experiment 1, 44 races, including representatives of complexity classes I–XIV (Table 1), were tested for ability to produce disease on the 28 parents of CCII. In experiment 2, races 40, 61, 72, and 74 were tested for ability to infect plants of 19 generations of CCII, including 12 generations that had not been assayed in previous studies (12,16,19). In experiment 3, the pathogenicity of 53 races was determined on generations 8, 14, 24, and 46 of CCII.

Assays of the disease reaction of individual races were conducted on seedlings grown from samples of seeds taken from various generations of CCII. We used the procedures described by Jackson and Webster (13) for inoculation and incubation of seedlings and for scoring disease reactions. The number of seedlings (N) tested in each generation of CCII varied from 109 to 479 in experiments 2 and 3. The standard error of estimate of the frequency of resistant (p) and susceptible plants (q), given by $SE = (pq/N)^{1/2}$, varied widely from sample to sample depending on sample sizes and the values of p and q. In judging the significance of differences between samples, for convenience, we have adopted a uniform criterion. If the difference in frequency of resistant plants for any pair of estimates was smaller than 0.1 (approximately two standard error units for the smaller samples), the samples were judged not to differ significantly, whereas samples that differed in frequency by more than 0.1 were considered to be significantly different.

RESULTS AND DISCUSSION

Host-pathogen complexity analyses. *Pathogenicity of 44 races on the parents of CCII.* Experiment 1 was conducted to determine the ability of 44 races to produce disease on the parents of CCII. Seven to 15 seedlings of each parental cultivar were inoculated with each of the 44 races, and the reaction of each parent to each race was uniform; i.e., there was no sample in which some seedlings were scored as resistant and others as susceptible. Table 2 gives the disease reaction of each of the 28 parents to each of the 44 races. As shown in the first column of Table 2, all 44 races produced disease on nine of the parents of CCII and five of the races were able to infect all 28 parents (bottom row, Table 2). This sample of races is thus highly pathogenic to the parents of CCII; all of the races are pathogenic on more than one-half of the parents and one-third of the races are able to infect all, or all except one, of the parents. Resistance to the 44 races is widely distributed among the parents of CCII (Table 2). Resistance was found in the parents to 39 of the

TABLE 1. Complexity classification for 75 races of *Rhynchosporium secalis* and 14 host differential cultivars[a]

Racial complexity[b]					Complexity of host cultivars[c]		
Cultivars infected (no.)	Racial complexity class	Races in complexity class	Races included in study				
			No.	Races[d]	Cultivar	Races resisted (no.)	Host complexity class
0	0	1	0	...	Kitchen	21	I
1	I	2–5	2	2,5	Atlas, Steudelli	25	II
2	II	6–10	2	9,10	Wisc. Winter × Glabron	29	III
3	III	11–20	6	14–18,20	La Mesita	32	IV
4	IV	21–28	7	21–27	Osires	40	V
5	V	29–32	4	29–32	Calif. 1311	43	VI
6	VI	33–38	3	33,36,38	Trebi, Atlas 46	49	VII
7	VII	39–49	11	39–49	CI 2376	51	VIII
8	VIII	50–56	7	50–56	Brier	53	IX
9	IX	57–61	5	57–61	CI 5831	54	X
10	X	62–66	5	62–66	Turk	55	XI
11	XI	67–70	4	67–70	Hudson	56	XII
12	XII	71–72	2	71,72			
13	XIII	73–74	2	73,74			
14	XIV	75	1	75			

[a] From data of Jackson and Webster (13).
[b] Based on numbers of differential host cultivars diseased by each race.
[c] Based on numbers of races to which each cultivar was resistant.
[d] Designation of Jackson and Webster (13).

44 races. Each of the 19 parents having any resistance had its own unique pattern of reaction to the 44 races. Nevertheless, the parents of CCII, when employed as a set of differential host cultivars, separated the 44 races into only 26 different groups; this result is not surprising because Jackson and Webster's (13) smaller set of differentials was selected to provide maximum differentiation among these particular racial isolates, whereas the parents of CCII represent a random set with respect to resistance to California races of *R. secalis*.

The data of Tables 1 and 2 allow comparison of the correspondence between levels of racial complexity, as measured by the 14 differentials of Jackson and Webster (13) (Table 1) vs. the parents of CCII. Inspection of these two tables shows that races that infected few of the 14 differentials also generally infected fewer of the parents of CCII than the more pathogenic races of Table 1. Race 16, for example, which infected only three of Jackson and Webster's differentials, is the least pathogenic of the 44 races tested on the parents of CCII (complexity class I, Table 2), whereas race 75, the most pathogenic race of Table 1, was also among the races that infected all of the parents of CCII (complexity class IX, Table 2). The correlation between the paired measures of racial complexity for the 44 races, $r = 0.559^{***}$, indicates highly significant correspondence between degree of racial complexity as measured by the two sets of host differentials. There are, however, many striking exceptions, e.g., race 2, the least complex among the 44 races (complexity class I, Table 1) was able to infect all except three of the parents of CCII, which places it in complexity class IV in Table 2; races 61 and 64, on the contrary, are quite complex races (complexity class IX and X, respectively) in Table 1, but they are among the simpler races (complexity class III and IV) in Table 2. The coefficient of determination, $r^2 = 0.559^2 = 0.312$, indicates that about one-third of the total variability in racial complexity, as measured by one set of host differentials, is explained by racial complexity, as measured by the other set of host differentials.

The comparisons of host complexity that can be made, as measured by the two sets of host cultivars, are limited because Atlas and Trebi are the only two among Jackson and Webster's (13) differentials that are also parents of CCII. Atlas is a simple host (complexity class II) and Trebi a host of intermediate complexity (complexity class VII) in Table 1, whereas Atlas is a host of intermediate complexity (complexity class V) and Trebi a quite complex host (complexity class VIII) in Table 2.

Many comparisons bearing on interactions between host complexity and pathogen complexity can be made from the data of Tables 1 and 2, and among these comparisons a number of striking exceptions to the generally positive relationships between complexity of host and pathogen were found. Some examples are: Race 2, the simplest race of Table 1, was able to infect all except the three most complex parents of CCII (Trebi, Han River, and Maison Caree); and races 20 and 38, which are quite simple races (complexity classes III and VI, respectively, Table 1) were both able to infect Maison Caree, the most complex parent of CCII. Atlas, although a simple host (complexity class II, Table 1; class V, Table 2) is, however, resistant to nine of the most complex races (60, 64, 65, 68, 69, 70, 71, 72, and 73). Races 40, 61, 72, and 74, which are common in California, fall into complexity classes VII, IX, XII, and XIII, respectively, in Table 1 and in complexity classes III, IV, VIII, and VI, respectively, in Table 2. This supports the conclusion of Jackson and Webster (13) that ability of a race to prosper in a major barley-growing area is independent of level of complexity.

Pathogenicity of races 40, 61, 72, and 74 on the parents and generations of CCII. Table 3 reports the frequencies of plants resistant to races 40, 61, 72, and 74 observed over a range of generations in three previous studies of CCII and in experiments 2 and 3 of the present study. The generations tested were not identical in these five experiments because seed stocks of certain generations were no longer adequate in the later experiments; when substitutions were necessary, the nearest generation for which

TABLE 2. Susceptibility (S) of the 28 parental cultivars of barley composite CCII to 44 races of *Rhynchosporium secalis* (experiment 1)

Race	a[z]	Pamella Blue	Calif. Mar.	Manchuria	Lion	Everest	Oderbrucker	Flynn	Meloy	Arequipa	Good Delta	Lyallpur	Algerian	Alpha	Atlas	White Smyrna	Glabron	Trebi	Han River	Maison Caree	Racial complexity class
16	S		S	S	S		S	S				S			S						I
29	S	S		S	S	S	S	S	S			S			S						II
32	S	S	S	S	S	S	S	S	S	S	S		S	S	S						III
15,27	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S						IV
18,61	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S					IV
22	S	S	S	S	S	S	S	S	S	S	S	S	S		S	S					IV
40	S	S	S	S	S	S		S	S	S	S		S	S	S	S					III
31	S	S	S	S	S	S	S	S	S	S	S		S	S	S	S					IV
5,17,25	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S					V
53	S	S	S	S	S		S	S	S	S	S	S		S	S		S				III
26,36	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S				V
10	S	S	S	S	S	S	S	S		S		S	S	S	S	S	S				IV
2,14,24,30	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S				IV
58	S	S	S	S	S	S	S	S	S	S		S	S	S	S	S		S			V
43	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S			VII
21	S	S	S	S	S	S	S	S	S	S	S		S		S	S	S		S		V
23	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S		VII
74	S	S	S	S	S	S	S	S	S		S	S	S	S	S	S		S	S		VI
54	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S		VIII
20	S	S	S		S	S	S	S	S	S	S	S		S		S	S			S	IV
64	S	S	S	S		S			S	S	S	S		S		S	S	S		S	III
60	S	S	S	S	S	S	S			S	S	S	S			S	S	S	S	S	V
65,68–73	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S	S	S	S	S	VIII
63	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S	S	VIII
38	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S		S	VIII
48,59,62,67,75	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	IX
Nonpathogenic races (no.)	0	1	1	1	1	2	2	2	3	3	4	4	5	5	5	6	11	17	18	19	
Host complexity class	0			I			II			III		IV			V	VI	VII	VIII	IX	X	

[z]a = nine susceptible parents (Club Mariout, Golden Pheasant, Hannschen, Horn, Minia, Multan, Orel, Sandrel, and Wisconsin Winter).

adequate numbers of seeds were available was selected.

Among the 28 parents of CCII, 20 were susceptible to races 40, 61, 72 and 74, and eight parents were resistant to one or more of the four test races (Table 2), as follows: resistant to race 40—Glabron, Han River, Lyallpur, Maison Caree, Oderbrucker, and Trebi; resistant to race 61—Atlas, Glabron, Han River, Maison Caree, and Trebi; resistant to race 72—Atlas; and resistant to race 74—Arequipa, Glabron, and Maison Caree. Jackson et al (12) reported an identical pattern of resistance to these four test races. Thus, among the 28 parents of CCII, six (21%), five (18%), one (4%), and three (11%) were resistant to races 40, 61, 72, and 74, respectively. With respect to these four test races, Glabron and Maison Caree had the most complex genotypes for resistance (resistant to three races), followed by Atlas, Han River, and Trebi (resistant to two races), Arequipa, Oderbrucker, and Lyallpur (resistant to one race) and the remaining 20 parents (resistant to none of the races).

The data on the various generations of CCII show that the frequencies of resistant plants observed for a given race in a given generation were similar in each of the five studies (Table 3). However, the pattern of change in frequency over generations was not the same for the different races. Two main patterns are discernible, one for race 72 and another for races 40, 61, and 74. The frequency of plants resistant to race 72 was consistently low (≤ 0.10) in all generations in each of the five studies, indicating that no significant change (>0.10) in frequency of plants resistant to that race occurred in CCII over the 46-generation span that was monitored. In contrast, the pattern for races 40, 61, and 74 was one of decrease in the frequency of resistant plants below parental levels in the early generations followed by increases in the later generations. Thus, the mean frequency of plants resistant to race 40 decreased in the early generations (F_7, F_8, F_{13}, F_{14}, and F_{15}) from the parental level of 0.21 to about 0.06; the mean frequency of resistant plants then increased to about 0.20 in the two intermediate generations (F_{23} and F_{24}) and to about 0.75 in the latest generations tested (F_{45}, F_{46}, and F_{47}). The pattern was similar for races 61 and 74, but the changes were smaller. These results establish that substantial and statistically significant changes in the frequencies of plants resistant to each of these three races occurred over generations in CCII.

In experiment 2 of the present study, the reaction to races 40, 61, 72, and 74 was determined for seedlings grown from seed samples taken from 19 generations of CCII, including 12 generations not assayed in previous experiments. The data of this experiment (Table 4) provide additional evidence that the frequency of plants resistant to race 72 was low in all generations of CCII, although frequencies of resistant plants reached 0.09 in generations F_{17} and F_{22} and 0.14 in generation F_{18}. The data also contain additional information concerning the dynamics of change for races 40, 61, and 74. The frequency of plants resistant to races 40 and 61 remained below parental levels until the transitions from F_{18} to F_{19}, at which time the frequencies of plants resistant to both races increased sharply (Table 4). No further significant changes in the frequency of plants resistant to race 61 occurred thereafter; however, the frequency of plants resistant to race 40 had increased sharply by generation F_{39} and, in the late generations (F_{40}–F_{46}), large increases as well as large decreases often occurred from generation to generation. In contrast to races 40 and 61, the frequency of plants resistant to race 74 remained below parental levels throughout the intermediate generations; however, as was the case for race 40, the frequency of resistant plants increased sharply in the interval from generation F_{23} to F_{39} and then fluctuated widely in generations F_{40} to F_{46}. It should be noted that, although each of the generations of experiment 2 was tested against all four races simultaneously, tests of certain of the generations were done at different times. Thus, although attempts to maintain standard conditions were made throughout the study, some of the observed differences in frequencies of resistant plants may have resulted from variations in disease development associated with differing environmental conditions during the several test runs. In summary, the data of experiment 2 show that: the dynamics of change were less similar for races 40, 61, and 74 than indicated by the earlier studies involving fewer generations; changes in frequency of resistant plants were often large from generation to generation; significant decreases in the frequency of resistant plants sometimes occurred, which suggests that resistance to scald may have detrimental effects on reproductive capacity under some environmental conditions; and changes in the frequency of plants resistant to different races were not always concordant in a given generation transition, which suggests that environments that favor infection of CCII by one race may not favor infection by other races. Ali and Boyd (1) noted that inter- and intra-isolate variability in both host reaction and isolate pathogenicity depends on specific host-isolate combinations and also environmental conditions.

TABLE 3. Frequencies[a] (f) of plants resistant to four races of *Rhynchosporium secalis* in generations (G) of CCII in three earlier studies and in experiments 2 and 3 of this study

	Jackson et al, 1978		Muona et al, 1982		Saghai et al, 1983		Experiment 1		Experiment 2		
Race	G	f	G	f	G	f	G	f	G	f	Mean
40	F_7	0.05	F_8	0.16	F_8	0.12	F_8	0.03	F_8	0.01	0.07
	F_{15}	0.08	F_{13}	0.05	F_{13}	0.10	F_{14}	0.06	F_{13}	0.02	0.06
	F_{25}	0.18	F_{23}	0.33	F_{23}	0.16	F_{24}	0.20	F_{23}	0.13	0.20
	F_{47}	0.85	F_{45}	0.60	F_{45}	0.88	F_{46}	0.79	F_{45}	0.56	0.74
61	F_7	0.11	F_8	0.04	F_8	0.04	F_8	0.00	F_8	0.03	0.04
	F_{15}	0.12	F_{13}	0.00	F_{13}	0.00	F_{14}	0.02	F_{13}	0.02	0.03
	F_{25}	0.23	F_{23}	0.20	F_{23}	0.08	F_{24}	0.08	F_{23}	0.19	0.16
	F_{47}	0.78	F_{45}	0.47	F_{45}	0.34	F_{46}	0.27	F_{45}	0.35	0.44
72	F_7	0.02	F_8	0.10	F_8	0.08	F_8	0.00	F_8	0.08	0.06
	F_{15}	0.01	F_{13}	0.04	F_{13}	0.02	F_{14}	0.00	F_{13}	0.04	0.02
	F_{25}	0.01	F_{23}	0.07	F_{23}	0.08	F_{24}	0.00	F_{23}	0.06	0.04
	F_{47}	0.04	F_{45}	0.01	F_{45}	0.06	F_{46}	0.00	F_{45}	0.03	0.03
74	F_7	0.17	F_8	0.06	F_8	0.02	F_8	0.00	F_8	0.01	0.05
	F_{15}	0.14	F_{13}	0.01	F_{13}	0.06	F_{14}	0.00	F_{13}	0.06	0.05
	F_{25}	0.28	F_{23}	0.18	F_{23}	0.12	F_{24}	0.09	F_{23}	0.02	0.14
	F_{47}	0.92	F_{45}	0.84	F_{45}	0.86	F_{46}	0.49	F_{45}	0.29	0.68

[a] Frequencies were calculated assuming that the plants parental to families segregating for resistance to races 40, 72, and 74 were resistant and that plants parental to families segregating for resistance to race 61 were susceptible; alleles for resistance to races 40, 72, and 74 are dominant whereas allele(s) for susceptibility to race 61 are recessive (Gurusinghe [8]).

TABLE 4. Frequencies[a] of plants resistant to four races[b] of *Rhynchosporium secalis* (40, 61, 72, and 74) in 19 generations of barley composite cross II (CCII) (experiment 2)

Generation of CCII	Race of *R. secalis*			
	40	61	72	74
F_8	0.01, 0.03	0.00, 0.03	0.00, 0.08	0.00, 0.01
F_{13}	0.02	0.02	0.04	0.06
F_{14}	0.06	0.03	0.00	0.00
F_{17}	0.08	0.10	0.09	0.01
F_{18}	0.09	0.09	0.14	0.02
F_{19}	0.17	0.13	0.06	0.02
F_{20}	0.22	0.20	0.04	0.03
F_{21}	0.23	0.24	0.04	0.03
F_{22}	0.15	0.17	0.09	0.02
F_{23}	0.13	0.19	0.06	0.02
F_{24}	0.20	0.08	0.00	0.09
F_{39}	0.56	0.23	0.02	0.21
F_{40}	0.64	0.27	0.01	0.21
F_{41}	0.74	0.30	0.02	0.42
F_{42}	0.48	0.27	0.04	0.37
F_{43}	0.53	0.30	0.01	0.15
F_{44}	0.54	0.30	0.02	0.26
F_{45}	0.56	0.35	0.03	0.29
F_{46}	0.79	0.27	0.00	0.49

[a] Seeding samples varying from 190 to 479 individuals were assayed to determine the frequency of resistant plants in each generation; mean sample size was 285.

[b] Designation of Jackson and Webster (13), see Table 1.

Pathogenicity of 53 races on generations of CCII. Table 5 reports the results of experiment 3 in which the frequency of plants resistant to 53 races was determined for generations F_8, F_{14}, F_{24}, and F_{46} of CCII. In this experiment, data for each race in each generation were taken simultaneously by the same observer; consequently changes in the frequency of plants resistant to a given race can be compared over generations with greater confidence than in the earlier studies. The patterns of change differed from race to race (Table 5). However, most of the races appeared to follow a pattern of change similar to one of the patterns previously observed for races 40, 61, 72, or 74. Patterns of change for the remaining races were similar to those of either race 16 or race 46. These six patterns, expressed numerically in terms of frequencies given in Table 5 for generations F_8, F_{14}, F_{24}, and F_{46}, respectively, and the number of races we placed in each class (in parentheses) are:

race 40—0.03, 0.06, 0.20, 0.79 (14);
race 61—0.00, 0.02, 0.08, 0.27 (15);
race 72—0.00, 0.00, 0.00, 0.00 (11);
race 74—0.00, 0.00, 0.09, 0.49 (3);
race 16—0.47, 0.56, 0.74, 0.89 (7);
race 46—0.24, 0.17, 0.12, 0.17 (3).

The distribution in these six categories of races from the different complexity classes of Table 1 is not at random. (The distribution was also not random when patterns of change were classified in several other ways.) Thus, among the 15 simplest races (complexity classes I–V) of experiment 3, the patterns of frequency change of 12 races were similar to those of either race 40 (races 2, 21, 23, 24, 25, 27, and 32) or race 61 (races 17, 18, 22, 26, and 31) and the patterns for three races (races 9, 16, and 29) were similar to that of race 16. Among the 12 races of low intermediate complexity (complexity classes VI and VII), the patterns of seven races were similar to those of either race 40 (races 40, 42, 44, and 45), or race 16 (races 41, 43, and 47). However, two races (races 48 and 49) followed the pattern of race 72, one race (race 39) the pattern of race 61, and one race (race 33) the pattern of race 46. Among the 12 races of high intermediate complexity (complexity classes VIII and IX), the majority of races (races 53, 54, 55, 56, 57, 58, 59, and 61) followed a pattern similar to race 61, whereas three races (races 50, 51, and 52) were classified in racial group 40 and one race (race 60) in racial group 72. Among the 14 most complex races (complexity classes X–XIV), 11 races fit into either racial group 72 (races 62, 65, 69, 70, 71, 72, 73, and 75) or racial group 74 (races 63, 67, and 74), whereas the three remaining races (races 64, 66, and 68) appeared to fit into racial groups 46, 16, and 61, respectively. The data of Table 5 therefore indicate that the evolutionary responses in CCII, as measured by patterns of increase in the frequency of resistant plants, were usually rapid and large for the simple races (e.g., race 16, complexity class III, Table 1), generally slower but still large for races of low intermediate complexity (e.g., race 40, complexity class VII), still slower and smaller for races of greater complexity (e.g., races 61 and 74, complexity classes IX and XIII), and consistently very small for races similar to race 72 (complexity class XII).

In summary, complexity analyses of the data of experiments 1, 2, and 3 show that, in general, races of *R. secalis* identified as complex by Jackson and Webster's (13) set of differential cultivars were able to infect more of the host genotypes in CCII than were simple races; hence, according to the definition of pathogenic complexity, complex races carry more alleles for pathogenicity than simple races. Complex host gentoypes generally resist more races of the pathogen than simple strains and, by the definition of host complexity, carry more alleles for resistance than the simple strains. The data of experiments 1, 2, and 3 indicate further that a general correlation exists between evolutionary responses of CCII and pathogenic complexity of races; simple races more often were countered by development of genetic resistance in the host population than were complex races. There were, however, many exceptions that lead us to the conclusion that estimates of

TABLE 5. Frequencies[a] of plants resistant to 53 races[b] of *Rhynchosporium secalis* in four generations of barley composite cross II (CCII) (experiment 3)

	Complexity class[c] and races														
	I	II	III			IV									
Generation	2	9	16	17	18	21	22	23	24	25	26	27			
F_8	0.05	0.20	0.47	0.00	0.01	0.01	0.00	0.03	0.01	0.05	0.00	0.02			
F_{14}	0.08	0.12	0.56	0.00	0.28	0.02	0.00	0.01	0.00	0.16	0.00	0.01			
F_{24}	0.22	0.32	0.74	0.02	0.17	0.18	0.00	0.13	0.17	0.18	0.09	0.12			
F_{46}	0.84	0.87	0.89	0.48	0.21	0.65	0.11	0.46	0.69	0.70	0.52	0.60			
	V			VI	VII										
	29	31	32	33	39	40	41	42	43	44	45	46	47	48	49
F_8	0.08	0.00	0.12	0.00	0.00	0.03	0.25	0.02	0.24	0.08	0.11	0.24	0.33	0.01	0.00
F_{14}	0.19	0.00	0.08	0.00	0.00	0.06	0.28	0.02	0.28	0.06	0.05	0.17	0.41	0.01	0.01
F_{24}	0.33	0.13	0.17	0.01	0.02	0.20	0.18	0.17	0.25	0.23	0.23	0.12	0.51	0.02	0.03
F_{46}	0.87	0.28	0.58	0.21	0.22	0.79	0.88	0.65	0.86	0.88	0.79	0.17	0.92	0.04	0.06
	VIII						IX								
	50	51	52	53	54	55	56	57	58	59	60	61			
F_8	0.02	0.01	0.04	0.01	0.00	0.00	0.01	0.02	0.00	0.00	0.03	0.00			
F_{14}	0.01	0.04	0.02	0.00	0.00	0.02	0.00	0.03	0.06	0.01	0.01	0.02			
F_{24}	0.15	0.18	0.19	0.01	0.03	0.08	0.01	0.05	0.08	0.05	0.01	0.08			
F_{46}	0.76	0.60	0.77	0.12	0.10	0.19	0.02	0.24	0.17	0.25	0.02	0.27			
	X					XI				XII		XIII		XIV	
	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
F_8	0.00	0.01	0.26	0.01	0.39	0.01	0.05	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
F_{14}	0.00	0.01	0.32	0.01	0.33	0.00	0.08	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
F_{24}	0.00	0.03	0.37	0.00	0.30	0.02	0.18	0.00	0.00	0.00	0.00	0.00	0.09	0.00	
F_{46}	0.00	0.24	0.12	0.01	0.59	0.40	0.17	0.00	0.00	0.00	0.00	0.00	0.49	0.00	

[a] Seedling samples varying from 109 to 309 individuals were assayed to determine the frequency of plants resistant to each race in each generation; mean sample size was 186.
[b] Designation of Jackson and Webster (13); see Table 1.
[c] See Table 1.

pathogenic complexity provide an uncertain base for analyzing the ability of a set of races to infect a given set of host strains or to induce an evolutionary response in a host population. Similarly, measures of the complexity of host genotypes appear to be of little value in analyzing the population biology of host-pathogen interaction systems. The data of experiments 1, 2, and 3 thus indicate that the specific characteristics of individual genes for pathogenicity or resistance are more important than their numbers; i.e., estimation of complexity does not provide important information concerning specific pathogenicity-resistance reactions. In the next section we analyze evolutionary response in terms of the pathogenicity of the individual races of experiments 1, 2, and 3 in interaction with the specific resistance vs. susceptibility reactions of the five genes presently known to govern resistance in CCII.

Gene-by-race analysis. In a recent study of the inheritance of reaction to scald disease in hybrids between several of the parents of CCII (crossed with each other and/or with several lines isolated from various generations of CCII), Gurusinghe (8) found that resistance vs. susceptibility is governed by a single locus for each of race 40, race 61, and race 74. Alleles for resistance are dominant and alleles for susceptibility are recessive for the loci governing reaction to races 40 and 74, whereas resistance is recessive and susceptibility is dominant for the locus governing the reaction to race 61. Assuming a gene-for-gene relationship between resistance and pathogenicity, this result implies that races 40, 61, and 74 each carry a single allele for pathogenicity toward the corresponding resistance gene in CCII. Gurusinghe (8) also reported that resistance vs. susceptibility to race 72 is governed by two complementary loci: individuals with at least one dominant allele at each locus (R_1-R_2-) are resistant, whereas individuals with genotypes R_1-r_2r_2, $r_1r_1R_2$-, and $r_1r_1r_2r_2$ are susceptible. Assumptions concerning a gene-for-gene relationship between resistance and pathogenicity are more tenuous for race 72; clearly, it is idle to speculate about the nature of the relationship in the absence of formal genetic studies of the inheritance of pathogenicity. The loci governing resistance to races 40, 61, and 74 are loosely linked; the locus governing resistance to race 74 is located between the other two loci at map distances of approximately 33 centimorgans in each case. The two complementary loci governing resistance to race 72 segregate independently of each other and the other three loci.

In a population such as CCII that mates by a proportion s of selfing and a proportion $t = 1-s$ of random outcrossing, the expected frequencies of plants resistant to each of the four races can be calculated for successive generations as illustrated in the following computations for race 40. Six among the 28 parents of CCII are resistant (genotype RR) and 22 are susceptible (genotype rr) to race 40. Thus, in the initial generation of CCII (F_1 generation), 15 of the 378 F_1 hybrids from which the population was synthesized resulted from crosses between RR parents, 132 were from crosses between RR and rr parents, and 231 were from rr × rr crosses. The frequencies of RR, Rr, and rr individuals were therefore $15/378 = 0.0396$, $132/378 = 0.3492$, and $231/378 = 0.6111$, respectively, in the F_1 generation and the inbreeding coefficient (F) calculated by Wright's (24) Fixation Index $\hat{F} = 1 - H/2pq$, in which H is the frequency of heterozygotes and p and q are the frequencies of alleles R and r, was thus $\hat{F} = 1 - 0.3492/2(0.2143)(0.7857) = -0.0371$. Assuming selective neutrality, change in F over generations in a population in which $s = 0.994$ and $t = 0.004$ (15) is given by

$$F^{(n)} = [(1-t)/(1+t)][1-(s/2)^{(n)}] + (s/2)^{(n)}F^{(0)}, \qquad (1)$$

in which $F^{(0)}$ and $F^{(n)}$ are inbreeding coefficients in the initial generation and in any generation n, respectively. Substituting $t = 1 - s = 0.006$ and $F^{(0)} = -0.0371$ into equation 1 gives the following expected values of F for generations F_1 through F_7: −0.0371, 0.4786, 0.7348, 0.8619, 0.9257, 0.9570, and 0.9861. Thus, by generation F_7 the theoretical inbreeding coefficient is expected to approach its equilibrium value, which is given by $F_e = (1-t)/(1+t) = 0.9881$. Expected frequences of genotypes RR, Rr, and rr for successive generations can be calculated by substituting pq and F into Wright's equilibrium equation,

$$p^2 + F^{(n)}pq[\mathrm{RR}] + 2pq(1-F^{(n)})[\mathrm{Rr}] + q^2 + F^{(n)}pq[\mathrm{rr}] = 1. \qquad (2)$$

Expected frequencies of plants resistant to race 40, and to races 61, 72, and 74 calculated in like manner, are given in graphic form in Figure 1. Note that the expected frequencies of plants resistant to races 40 and 74 are given by the sum of the frequencies of the RR + Rr genotypes, for race 61 by the frequency of rr individuals, and for race 72 by the frequencies of R_1-R_2- individuals.

Observed frequencies of plants resistant to the four test races (Table 3) are also given in Fig. 1. The observed frequencies of plants resistant to races 40, 61, and 74 (Fig. 1A, B, and D) decreased significantly from the parental generation to generations F_8 and F_{14}; an increasing trend then started that continued until generation F_{46}, the final generation assayed. In generations F_8 and F_{14}, the observed frequencies of resistant plants were significantly lower than the expected frequencies; however, observed frequencies increased to expected levels by generation F_{24}, and by generation F_{46} they exceeded the expected frequencies significantly for all three races.

There were no clearly significant departures between the expected and observed values for race 72 (expected frequencies were low in all except the very earliest generations) (Fig. 1). However, some plants resistant to race 72 were observed in nearly all generations, and the presence of resistant plants, even in low frequencies, is probably meaningful because the expected frequency of R_1-R_2- phenotypes is very low in CCII (0.0013 at inbreeding equilibrium). Thus, for example, the observed frequencies of plants resistant to race 72 were 0.09 in generation F_{17} and 0.14 in generation F_{18} (experiment 2, Table 4), and even in the latest generations (F_{45} and F_{47}) of four studies (Table 3), they were 0.04, 0.01, 0.06, and 0.03; these frequencies are from 8 to 108 times larger than the expected frequencies. We conclude that the frequencies of plants resistant to race 72, like the frequencies of plants resistant to races 40, 61, and 74, were higher than expected, assuming selective neutrality, in many generations of CCII.

Various hypotheses can be formulated to account for the above dynamic changes in the frequency of plants resistant to races 40, 61, and 74 in CCII. Perhaps the simplest and most obvious hypothesis is that these three races, or races with similar pathogenic properties, have been a relevant part of the environment of CCII in at least some of the seasons in which this population was grown. In seasons favorable for the development of scald, the reproductive capacity of susceptible plants may have been reduced by disease and resistant plants may have been at a selective advantage; conversely, in seasons not favorable for the development of scald, resistance is irrelevant and resistant plants may have been at a selective disadvantage. The deficiencies of resistant plants in generations F_8 and F_{14} suggest that scald was not an important part of the environments in which many of the early generations of CCII were grown. According to C. A. Suneson, the seasons in which the first 8–10 generations of CCII were grown were generally dry and almost no scald was observed in the population. This leads to the inference that resistance alleles had detrimental effects in those generations and, as a result, their frequencies fell below levels expected under the assumption that alleles governing resistance vs. susceptibility are selectively neutral. The increases in frequency of resistant plants that occurred thereafter suggest that conditions were often favorable for the development of scald and that each of the three races exerted a net selective pressure favoring resistant plants in the intervals from generations F_{14} to F_{24} and F_{24} to F_{46}.

Figure 1 also gives expected frequencies of resistant plants calculated from estimates of selective values made by iteration, using a computer program written by A. Hakim Elahi. The decreases in frequency of resistant plants that occurred from the initial generation to generation 13 are consistent with average selective disadvantages of resistant plants of 14, 24, and 9% per generation, respectively, for races 40, 61, and 74. The selective advantages that give the best fits to the observed frequencies of resistant plants that occurred in the later generations range from 10 to 15% for races 40 and 74 for the intervals from F_{13} to F_{23} and F_{23}

to F_{46}, and from 17 to 6% for race 61. Selective values of this magnitude or even larger have been observed for various other marker loci in composite cross populations (3).

The selective values reported for race 72 in Fig. 1C must be regarded more cautiously because resistant plants were infrequent in all generations and the observed frequency changes were often smaller than their standard errors. It is not surprising that the frequency of resistant plants remained low because, even if resistant plants have a very high selective advantage, the rate of increase in frequency is expected to be small when the targets of selection are a pair of alleles, both necessary for resistance and both initially rare in the population. The fact that plants resistant to race 72 were present in most generations of CCII at frequencies many times higher than expected under selective neutrality indicates that the selective pressures exerted on the population by race 72 were usually strongly in favor of resistant plants.

The most obvious features of directional change in CCII were the large increases in the frequency of plants resistant to races 40, 61, and 74 that occurred at the expense of susceptible plants. Estimates of selective advantages of corresponding resistant genotypes were large on the average, and we can therefore conclude that substantial selection favoring the resistant genotypes took place in the population. This was probably also the case for race 72, as discussed above. However, this does not establish that the resistance alleles at the five loci monitored were themselves the target of the selection. In populations such as CCII, which reproduce by mixed selfing and random mating, the mating system imposes a correlational structure on the entire multilocus structure of the population such that each locus is influenced by the selective effects of all other loci, including even loci located on different chromosomes (4,15). Thus, what is measured by changes in genotypic frequencies at the resistance loci are not only the selective effects of these loci as markers, but also the effects of the flow of selection throughout the entire genome. Because the effects of the marker loci are confounded not only with loci linked with the markers on the same chromosome but also with unlinked loci located throughout the genome, the loci specifically responsible for the selection cannot be identified with certainty. We note, however, that alleles for resistance to each of races 40, 61, and 74 were introduced into CCII from several parents (6, 5, and 3, respectively); also, alleles for resistance to race 72 may have been introduced from more parents than the one parent (Atlas) that was resistant to this race, because some susceptible parents may have been genotypically $R_1R_1r_2r_2$ or $r_1r_1R_2R_2$ and hence susceptible. The parents of CCII were selected for genetic diversity; consequently, alleles for resistance were almost certainly introduced into the population in many different genetic backgrounds, including genetic backgounds that are poorly adapted to the environment at Davis, CA. The fact that alleles for resistance to all 53 races that were tested increased in frequency (increases for race 72 and similar

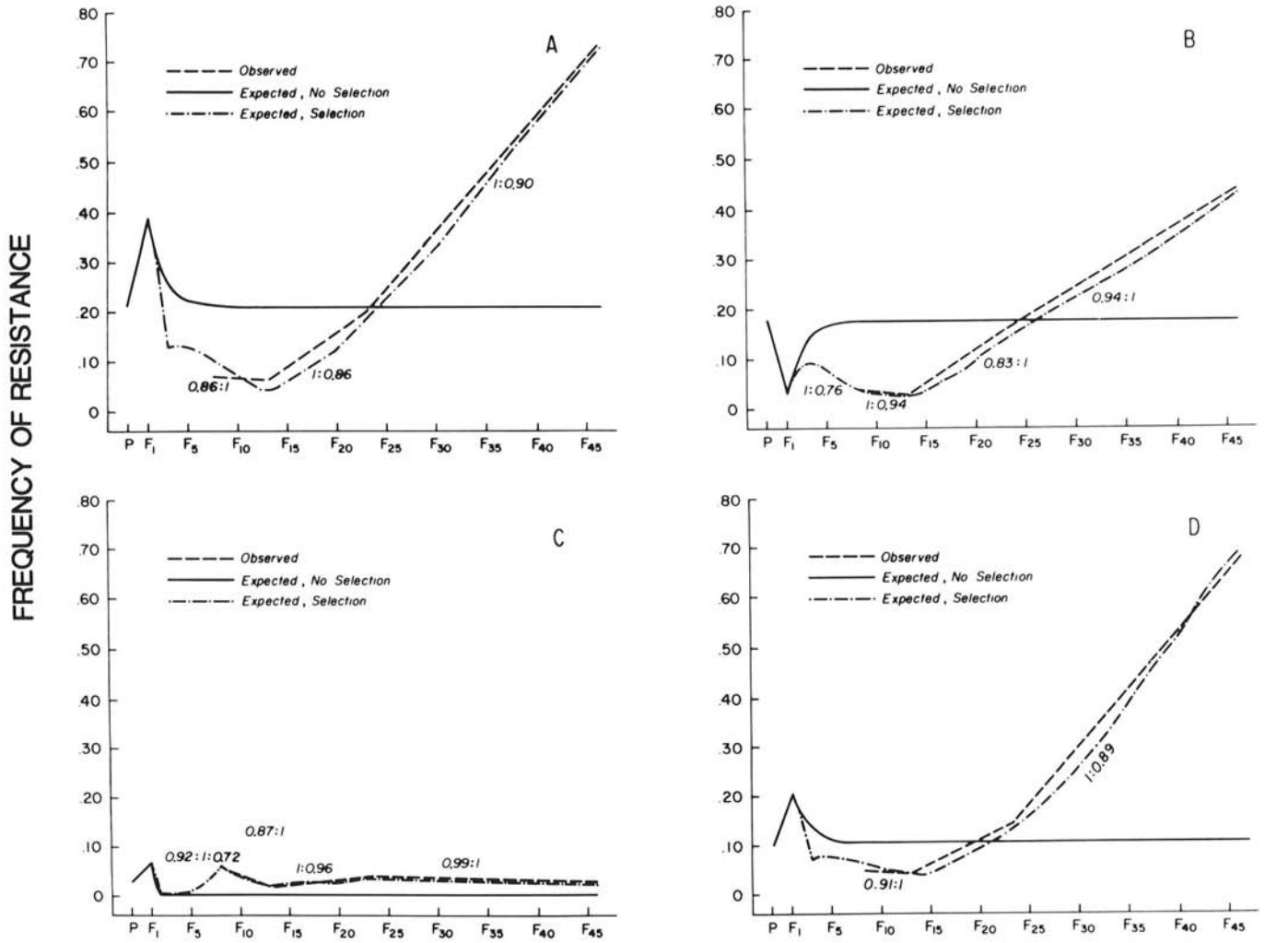

Fig. 1. Observed (mean values, Table 2) and expected (with and without selection) frequencies of plants resistant to four races of *Rhynchosporium secalis*; **A,** race 40; **B,** race 61; **C,** race 72; and **D,** race 74. Expected frequencies of resistant plants were calculated from estimates of selective values made by using an iterative computer program written by A. Hakim-Elahi.

races were usually not statistically significant) suggests that the resistance alleles were not swept passively into high frequency by the flow of selection operating throughout the genome but that the resistant alleles themselves contributed superior reproductive capacity in many generations by protecting the plant from damage from scald disease.

In discussing selection in CCII, it is appropriate to note that substantial numbers of families segregating for resistance vs. susceptibility to races 40, 61, 72, and 74 were reported in generations F_8, F_{13}, F_{23}, and F_{47} of CCII by Muona et al (16) and Saghai-Maroof et al (19). Fixation Indices ($\hat{F} = 1 - [H/2pq]$) estimated from the data of these experiments were nearly always substantially smaller than the theoretical inbreeding coefficient, $F_e = 1 - t/1 + t = 0.9881$, for CCII. Thus, for example, $\hat{F}$ took values of 0.86, 0.80, 0.90, and 0.87 and 0.70, 0.49, 0.50, and 0.58, respectively, for races 40, 61, 72, and 74 (data of Muona et al [16] and Saghai-Maroof et al [19]). Heterozygotes were thus in substantial excess over expectations based on selective neutrality. We conclude that substantial selection favoring heterozygotes has taken place in CCII; again, however, we cannot attribute the excesses solely to the resistance loci because the excesses may reflect an "entire-genome heterosis" associated with the correlated structure imposed on the population by the mating system of predominant self-fertilization (4,22,23). However, heterozygotes at all five of the resistance loci are in much greater excess than for other loci in CCII, e.g., allozyme loci and loci governing morphological markers (3). These large excesses again suggest that the resistance loci were not swept passively into excess frequency by entire genome heterosis but that resistance loci made substantial individual contributions to the heterosis.

The above gene-by-race analysis of evolutionary change in CCII was based on five resistance genes contributed to the population by seven of its parents, Oderbrucker, Atlas, Arequipa, Glabron, Trebi, Han River, and Maison Caree (Table 2). Because each of the 19 parents that had any resistance to the 44 races of experiment 1 had its own unique pattern of resistance, however, it is apparent that the parents of CCII contributed many more resistance genes to the population than the five loci known to govern resistance to races 40, 61, 72, and 74. The number can be estimated as follows. Nine of the parents of CCII (Table 2, column a) are susceptible to all four races. Among the next five parents, one (California Mariout) is resistant to race 29, two (Pamella Blue and Everest) are resistant to race 16, one (Manchuria) is resistant to race 20, one (Lion) is resistant to race 64, and one (Everest) is resistant to race 53. The inheritance of resistance to these five races is not known: one possible mode of inheritance is that the five differences in resistance are governed by five different alleles of a single genetic locus; another possible mode is that they are governed by five different loci, each with only a single allele for resistance. In the latter case, these five parents contributed five new loci affecting scald reaction to the population, additional to the five loci known to govern resistance to races 40, 61, 72, and 74. By similar reasoning, the next five parents (Oderbrucker, Flynn, Meloy, Arequipa, and Good Delta) contributed five more resistance loci, Lyallpur contributed three new loci; Algerian, none; Alpha, one; Atlas, two; White Smyrna, two; Glabron, one; Trebi, three; Han River, one; and Maison Caree, one; bringing the total number of potential resistance loci in CCII to 29. Results of a survey of resistance in the world barley collection by Webster et al (21) are consistent with this possibility. Even if this is a substantial overestimate of the number of resistance loci, it is apparent that the five loci on which the above gene-by-race analysis was based provide far too little information for an adequate analysis of evolutionary change. Formal genetic studies of the differences in resistance cataloged above have been initiated as an essential preliminary to a comprehensive analysis of evolutionary change based on gene-by-race relationships.

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