Race Composition and Pathogenicity Associations of *Rhynchosporium secalis* in California

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ABSTRACT

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Data from a pathogenicity study involving 723 isolates of *Rhynchosporium secalis,* the causal agent of barley scald, were analyzed for race composition and pathogenicity associations. Isolates were collected from California in 1973, 1983, and 1984. The results showed that the collection made in 1973 differed drastically in race composition from the collections of 1983 and 1984. The 1973 collection was composed mostly of simple races that were capable of producing disease on a few host differentials. The two later collections contained large proportions of complex races that were pathogenic on a large number of differentials. The majority of the pairwise correlations between pathogenicity on different host differentials were highly significant in all three collections, which indicated close associations of genes for pathogenicity among isolates of this fungus. In contrast to the large difference in race composition, most of the significant correlations were in the same direction (positive or negative) in all three collections. The overall structure of pathogenicity association, as determined by cluster analysis, also was very similar for isolates in all three collections. It is inferred that the highly conserved pathogenicity associations were at least partly due to natural selection favoring the development and maintenance of particular pathogenic gene combinations in the fungal populations.

Additional keywords: asexual reproduction, discriminant analysis, evolution.

Rhynchosporium secalis (Oudem.) J. J. Davis causes a serious foliar disease (leaf scald) on barley (*Hordeum vulgare* L.) in places where the growing season is cool and humid. Genetic variability in *R. secalis* populations and corresponding variability in host resistance have been studied by workers in several countries (1,6,7,12,13,18,19). A large number of races have been identified by inoculating host differentials with isolates collected from naturally infected plants. Jackson and Webster (7) separated 175 isolates collected from barley-growing areas in California into 75 races on 14 host differentials, which are known to carry major genes for resistance. Crandall (2) identified an additional 287 races among isolates collected from California in 1983 and 1984 using the same set of 14 differentials. Studies on the barley population composite cross II showed that frequencies of genes for resistance to many of these races increased greatly in later generations as compared with the frequencies in earlier generations (14,17,18). Further studies using four races that were selected on their combined ability to attack the known resistance genes in the 14 differentials indicated that increases in frequencies of genes for resistance to the races 40, 61, and 74 were highly correlated in the composite class II population (14,17). The same three-way association was observed among 350 accessions representing major barley-growing areas of the world (19).

Many changes in resistance of plant hosts have resulted directly in response to evolutionary processes in the pathogen (13). Asso-

ciations between genes for resistance in the host also would be expected to reflect the matching associations of pathogenicity in the pathogen. Conversely, changes in frequency of pathogenicity in the pathogen are frequently directly related to the genetic composition of the host. The dynamic nature of coevolution between the host and pathogen thus allows information obtained on host resistance to be used to infer the pathogenicity structure in the pathogen population. Certain factors, however, may impose serious constraints on the inference of pathogenicity on the basis of host resistance: 1) the influence of human activity often does not permit the establishment of such a correspondence between pathogens and hosts in crop plants; 2) similar environmental factors frequently affect pathogen and host differently, and their optimal conditions often differ; and 3) the genetic mechanisms of evolutionary change in pathogen and host populations are often completely different. Therefore, studies on the genetics of pathogenicity also are necessary to better understand the evolutionary processes that occur in pathogen populations.

Many fungal plant pathogens reproduce predominantly asexually, and this lack of a sexual stage precludes standard Mendelian analysis. Thus, the existence of genes for pathogenicity can only be inferred by assuming a gene-for-gene correspondence with the host (3). Disease reaction data obtained by inoculating host differentials with fungal isolates often have been used to study race composition and pathogenicity associations of asexual populations (4,6,7). Such studies have been useful in revealing important properties of pathogen populations. The studies reported in the present paper were conducted to compare the race composition of *R. secalis* collected in three different years in California and to elucidate the patterns of pathogenicity association in these three collections.

MATERIALS AND METHODS

Collection and isolation of the fungus. Collections were made from naturally infected barley plants during the spring of 1973, 1983, and 1984 in California. A total of 23 barley-growing counties were represented in the 1973 collection, with most of the samples taken from the Sacramento Valley, San Joaquin Valley, and the coastal valley (7). In the collections made in 1983 and 1984 (2), a large portion of the sample was obtained from composite crosses and pure lines grown in three areas: San Luis Obispo, Yolo County, and the University of California at Davis agronomy farm. San Luis Obispo is in the coastal valley, and Yolo County and the agronomy farm are both in the Sacramento Valley. Samples also were collected from commercial fields in 10 counties across California.

Leaves with fresh lesions were collected from barley fields and brought to the laboratory. Single-spore isolates were obtained following the procedures described by Jackson and Webster (7). Isolates were maintained on plates or test tubes containing potato-dextrose agar. The total number of isolates obtained was 175 for 1973, 275 for 1983, and 273 for 1984.

Determining pathogenicity of the fungus. The pathogenicity of the single-spore isolates was determined by inoculating each isolate on a set of differentials consisting of 14 barley lines and cultivars known to carry different genes for resistance to *R. secalis* (Table 1). The procedures for spore preparation and inoculation were essentially the same as those described by Jackson and Webster (7). The differentials were planted in U.C. soil mix C-2 (10) in metal flats with five seedlings per differential and a complete set of the differentials in each flat. A fifteenth line (UC 566 or Wong) also was planted with each set of differentials as a universal susceptible. Each flat was inoculated with 50 ml of a conidia suspension cultured from a single-spore isolate at a concentration of 2×10^5 spores per milliliter. One flat of differentials in each set of tests was sprayed with distilled water as an uninoculated control. After inoculation, flats were placed in a humidity chamber (100% relative humidity) at 15 C for 48 h and then transferred to a greenhouse at 13–25 C for 14 days to allow disease development. Twelve randomly selected isolates of the 1973 collection, retested for constancy and repeatability of the method, gave highly repeatable results (7). A large data base has been generated using this method during the last 15 yr (2,4,5,7,14,18,19). It also has been established that the pathogenicity of the *R. secalis* remains stable when the isolates are kept in isolation either in culture or on barley plants (7,8).

Pathogenicity of the fungal isolates was evaluated using the 0–4 disease scale of Jackson and Webster (7), which was devised to score scald reaction of barley plants. Ratings of 3 and 4, which were taken as susceptible reactions of the host, were considered to be a pathogenic reaction of the fungus. Ratings of 0, 1, and 2, which were taken as resistant reactions of the host, were considered to be nonpathogenic reactions of the isolates. The splitting of the five ratings into two classes corresponds to the effects of major genes for resistance and susceptibility in the host (5,7). According to the gene-for-gene hypothesis (3), we assume that such division also reflects the effects of major genes for pathogenicity.

Isolates were classified into races on the basis of pathogenicity responses to barley differentials. Individuals with the same reactions to all 14 differentials were grouped into one race, whereas those that differed in their reaction to one or more differentials were classified as different races.

Statistical analyses. Potentially, the 14 differentials used could distinguish 16,384 races. A total of 362 races were identified from these three collections. Cluster analysis (9) by case was performed to classify the 75 races identified in 1973 into groups. The reactions of the 75 races to the 14 differentials were used as 75 different cases, and races with similar reactions to the 14 differentials were joined to form groups. The clustering procedures were terminated when joining two groups would cause an average difference of pathogenicity reactions among races in the same cluster to be more than two differentials (i.e., average Mahalanobis distance > 2). The resulting groups were subsequently used for classification of isolates collected in 1983 and 1984.

A discriminant analysis (9) was conducted to assign the isolates of the 1983 and 1984 collections into appropriate groups as resolved by the cluster analysis of the races in the 1973 collection. The classification functions were constructed on the basis of the isolates in the 1973 collection that had been grouped by cluster analysis. An isolate was placed into the cluster that gave the smallest Mahalanobis distance between that isolate and the cluster, and it would not be placed into any cluster if the smallest distance between the isolate and any cluster exceeded 2. The race compositions of these three collections were thus compared on the basis of the resulting race clusters.

Pathogenicity associations among isolates collected in each year were first analyzed using pairwise correlation by treating the

TABLE 1. Host differentials used to identify races of *Rhynchosporium secalis* isolated from barley in California[a]

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

[a]This table is adapted from Jackson and Webster (7).
[b]Both CI 2376 and CI 5831 have at least two dominant genes for resistance (M. S. Mohamed and C. W. Schaller, *unpublished data*).

reactions to the 14 differentials as 14 variables. Cluster analyses (9) by variable were then performed to determine the overall structure of the pathogenicity associations of the reactions of the isolates to the 14 differentials using pairwise correlations as the similarity measurements. At each step of agglomeration, groups of variables with the largest similarity, as measured by the averaged pairwise correlations between members of two clusters, were joined to form new clusters. Grouping was stopped if more than 10 of the 14 host differentials were placed into clusters or if formation of the new cluster would reduce the average correlations among members within a cluster below the critical level at the 0.01 probability level (0.194 for the 1973 and 0.155 for the 1983 and 1984 collections).

RESULTS

A total of 362 races were identified from the 723 isolates collected over these 3 yr, with 75, 180, and 183 for the years 1973, 1983, and 1984, respectively.

Cluster analysis. Six clusters were resolved among the 75 races from the 1973 collection (Table 2). The first cluster contained 34 races representing 88 of the 175 isolates. Races included in this cluster were pathogenic on a mean of 3.4 differentials and had a host range of 10 of the 14 used (Table 3). More than 29% of those races were pathogenic to seven host differentials: Atlas, CI 5831, Kitchen, La Mesita, Osiris, Steudelli, and Wisconsin Winter × Glabron (WW × G).

The second cluster comprised two races that were pathogenic to an average of 4.5 host differentials. Cluster 2 races were pathogenic on Atlas, Atlas 46, Brier, Turk, and WW × G.

The third cluster consisted of three races that were pathogenic on a mean of 7.0 host differentials. The host range of cluster 3 was similar to that of cluster 1, except that one cluster 3 race was pathogenic on Turk and one cluster 1 race was pathogenic on Hudson (Table 3). All of the races of cluster 3 were pathogenic on Osiris, but only 32% of the cluster 1 races could attack that differential. The differentials Ca 1311 and CI 2376, which were resistant to 94% and 97% of the cluster 1 races, respectively, were susceptible to all three races of cluster 3.

Cluster 4 consisted of six races that were, on average, pathogenic to 7.7 host differentials. The host range of this cluster included all of the differentials that were susceptible to races of cluster 2. In addition, 100, 83, and 67% of cluster 4 races were pathogenic to Hudson, Kitchen, and Steudelli, respectively.

The 13 races placed in cluster 5 were pathogenic on a mean of 8.5 hosts. All of the host differentials except one (Hudson) that were susceptible to cluster 1 races also were susceptible to cluster 5 races. In addition, races of cluster 5 also were pathogenic on Atlas 46 and Trebi.

Cluster 6 contained 10 races that were pathogenic on a mean of 11.7 hosts. Eight of the 14 differentials were susceptible to all of the races in this cluster, and one race was pathogenic to all 14 host differentials.

Formation of the above six clusters at a similarity level (average distance) of ≤ 2 left seven races unclassified. Placing any of these seven races into any of the six clusters or joining any two to form a cluster would cause the average distance within the cluster to exceed 2. Hence, clustering procedures were terminated at this stage.

Discriminant analysis. The classification functions of the discriminant analysis, constructed on the basis of the 167 isolates represented by the 68 races in the six clusters, were able to correctly place 166 of the 167 isolates back to their respective clusters. Table 4 lists the number of isolates resolved to each cluster. A χ^2 test showed that, in terms of clusters, the race composition of the 1973 collection was highly significantly ($P < 0.01$) different

TABLE 2. Clusters of *Rhynchosporium secalis* races and the mean number of susceptible host differentials from isolates collected in 1973

Cluster	Races in cluster	Mean susceptible host differentials
1 (34 races)	1–17, 19–21, 23–27, 29–31, 33–36, 41, 42	3.4
2 (2 races)	28, 32	4.5
3 (3 races)	44, 45, 53	7.0
4 (6 races)	39, 40, 47, 50, 51, 57	7.7
5 (13 races)	38, 48, 49, 52, 54, 55, 58–63, 67	8.5
6 (10 races)	65, 66, 68–75	11.7
Unclassified (7 races)	18, 22, 37, 43, 46, 56, 64	

TABLE 3. Number of races of *Rhynchosporium secalis* in each cluster of the 1973 collection pathogenic on each of the 14 barley differentials

Host differential	Cluster						Unclassified races
	1	2	3	4	5	6	
Atlas	28[a] (82)[b]	2 (100)	3 (100)	5 (83)	7 (54)	2 (20)	3
Atlas 46	...	2 (100)	...	6 (100)	4 (31)	10 (100)	4
Brier	...	2 (100)	...	6 (100)	...	10 (100)	4
Ca 1311	2 (6)	...	3 (100)	2 (33)	11 (85)	10 (100)	5
CI 2376	1 (3)	...	3 (100)	...	10 (77)	8 (80)	2
CI 5831	10 (29)	...	1 (33)	...	6 (46)	3 (30)	2
Hudson	1 (3)	...	...	6 (100)	...	8 (80)	4
Kitchen	18 (53)	...	3 (100)	5 (83)	13 (100)	10 (100)	6
La Mesita	13 (38)	...	2 (67)	...	13 (100)	10 (100)	5
Osiris	11 (32)	...	3 (100)	4 (67)	12 (92)	10 (100)	3
Steudelli	22 (65)	...	3 (100)	4 (67)	12 (92)	6 (60)	3
Trebi	...	...	...	...	12 (92)	10 (100)	4
Turk	...	1 (50)	1 (33)	6 (100)	...	10 (100)	2
Wisconsin Winter × Glabron	10 (29)	2 (100)	3 (100)	6 (100)	10 (77)	10 (100)	5

[a]Number of races pathogenic to the differential.
[b]Percentage of the races in that cluster pathogenic to the differential.

TABLE 4. Number of *Rhynchosporium secalis* isolates classified into each of the six race clusters by discriminant analysis

Collection	Cluster						Unclassified isolates	Total
	1	2	3	4	5	6		
1973	88	2	3	9	16	49	8	175
1983	11	57	3	9	56	116	23	275
1984	11	42	9	11	46	126	28	273

from those of the 1983 and 1984 collections, and that the race compositions of the 1983 and 1984 collections were not significantly different from each other. Most of the differences in the race composition between the 1973 collection and the two later collections were due to the predominance of simple races capable of producing disease on a few differentials in the 1973 collection, whereas in the 1983 and 1984 collections complex races pathogenic on a large number of differentials were more frequent.

Pathogenicity association by pairwise correlations. There were 91 possible pairs of phenotypic responses of the isolates to the 14 host differentials in each year. The 273 possible pairwise correlation coefficients for the 3 yr of this study were calculated.

Seventy-nine of the 91 correlations were significant at the $P = 0.01$ probability level in the 1973 collection. Among the 12 nonsignificant correlations, six involved pathogenic reactions to cultivar Steudelli, five included host differential CI 5831, and one was between Steudelli and CI 5831.

Sixty-four correlations were significant at the $P = 0.01$ level in the 1983 collection. Among the 27 nonsignificant correlations, 11 involved reactions of the isolates to Atlas, and nine were due to reactions to Atlas 46. The remaining nonsignificant correlations resulted from reactions to host differentials Brier, CI 5831, Hudson, and Steudelli.

The pairwise correlations among isolates in the 1984 collection were similar to those in the 1983 collection. Sixty-seven correlations were significant at the $P = 0.01$ level. Among the 24 nonsignificant correlations, 10 involved reactions to cultivar Atlas and eight were due to reactions to Atlas 46. The other six nonsignificant correlations resulted from reactions to host differentials CI 2376, CI 5831, and Brier.

Inspection of the correlation coefficients revealed that 62% were simultaneously significant and 4% simultaneously nonsignificant in all three collections. In the 1973 and 1983, 1973 and 1984, and 1983 and 1984 collections, 3.3, 6.6, and 24% of the correlations, respectively, were simultaneously significant or nonsignificant.

Structure of pathogenicity associations by cluster analysis. The structure of pathogenicity associations for the isolates in the 1973 collection can be characterized with two clusters (Fig. 1). The first cluster consisted of six host differentials: Ca 1311, Trebi, CI 2376, La Mesita, Osiris, and Kitchen. The relationship represented by this cluster was such that isolates pathogenic to any one of these host differentials were likely to be pathogenic to the other five host differentials, or vice versa. Similarly, the structure of the second cluster indicated that isolates were likely to be simultaneously pathogenic or nonpathogenic to the five host differentials Atlas 46, Brier, Turk, Hudson, and WW × G.

Table 1 showed that among the six host differentials of cluster 1, four (Ca 1311, La Mesita, Osiris, and Trebi) were known to carry the resistance gene *Rh4,* one (Kitchen) carried *Rh9,* and one (CI 2376) carried some unnamed gene(s). Thus, isolates that were pathogenic to *Rh4* also tended to be pathogenic to *Rh9.* Also, among the five host differentials that were placed in the second cluster, three (Brier, Hudson, and WW × G) were known to carry the resistance gene *Rh,* cultivar Atlas 46 carried genes *Rh2* and *Rh3,* and Turk carried *Rh3* and *Rh5.* Therefore, this cluster indicated that isolates pathogenic to the gene *Rh* also were pathogenic to the gene combinations of *Rh2, Rh3* and *Rh3, Rh5.*

Subjecting isolates of the 1983 collections to cluster analysis resulted in four clusters (Fig. 2). Two host differentials, Atlas and Atlas 46, were included in the first cluster, indicating that isolates that were pathogenic to Atlas also were pathogenic to Atlas 46. It should be noted that Atlas 46 was very closely associated with four other host differentials of the second cluster in the 1973 collection. The second cluster consisted of three host differentials: Brier, Turk, and WW × G. Comparison of Figures 1 and 2 showed that all the three host differentials were members of the second cluster in the 1973 collection. The third cluster was composed of four host differentials—Ca 1311, Trebi, La Mesita, and Kitchen—that were all in the first cluster of the 1973 collection. The fourth cluster included three host differentials, CI 2376, Osiris, and CI 5831. Both CI 2376 and Osiris were in the first cluster of the 1973 collection, whereas CI 5831 was not placed in any cluster in the 1973 collection.

Cluster analysis of the 1984 collection placed 11 host differentials into three clusters (Fig. 3). The first cluster was the same as the first cluster in the 1983 collection. Three of the four host differentials (Brier, Turk, WW × G, and Hudson) placed in the second cluster also were put in the second cluster of the 1983 collection. The third and largest cluster of the 1984 collection included differentials Ca 1311, Trebi, La Mesita, and Osiris, and it was nearly identical to the third cluster of the 1983 collection.

It should be noted that for all three collections, a larger cluster with 12 or more host differentials could have been obtained if the 0.01 level of significant similarity had been used as the only criterion. This suggested that the pathogenic reactions of the isolates to almost all of the host differentials were closely associated.

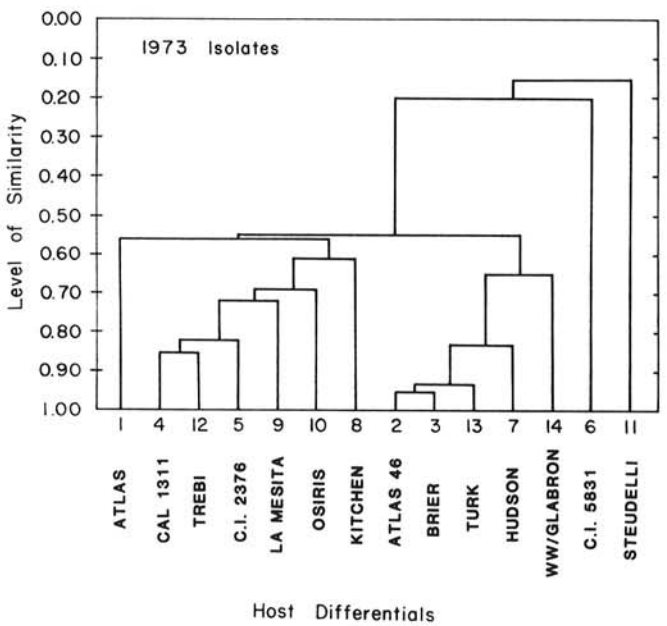

Fig. 1. The cluster structure showing the associations of pathogenicity to 14 barley host differentials among the *Rhynchosporium secalis* isolates of the 1973 collection.

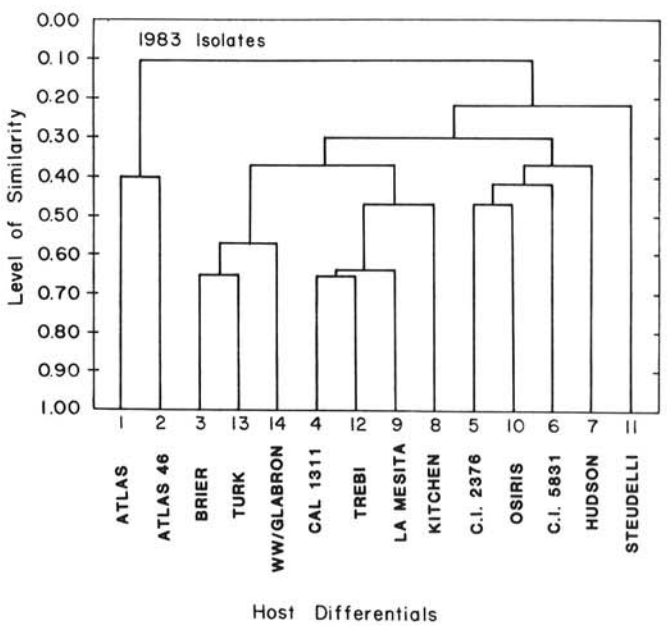

Fig. 2. The cluster structure showing the associations of pathogenicity to 14 barley host differentials among the *Rhynchosporium secalis* isolates of the 1983 collection.

DISCUSSION

The 723 isolates, representing 362 races obtained from the three collections, encompassed a broad spectrum of the race composition of *R. secalis* in California. The sample sizes of 175, 275, and 273 for these three collections would allow races with frequencies > 0.021, 0.014, and 0.014, respectively, to be included in the sample with 95% confidence. Moreover, the total sample size of 723 isolates would allow races with frequency > 0.005 to be included at least once with 95% confidence. Thus, individual races of significant portions, existing in the sampled areas of California in these 3 yr, would have been detected. However, many races were represented only by one isolate in the three collections as a whole, indicating that the frequencies of many races were lower than 0.005. This in turn suggests that the total number of races of *R. secalis* in California, as defined by these 14 differentials, was much larger than 362.

Two possible causes for the observed differences in race compositions among these three collections are nonuniform distribution of the races in the sampled areas and/or temporal changes in race composition in California during these 11 yr. Because of the large number of races and highly variable race populations, the present data cannot distinguish one possibility from the other.

A feature that emerged from the analysis was the large proportion of highly significant correlations between pathogenicity of the isolates on host differentials carrying different genes for resistance. Assuming gene-for-gene correspondence, such correlations suggest close associations of genes for pathogenicity in the pathogen populations.

Another feature is the highly conserved pathogenicity associations in these three collections in contrast to the large differences in race composition. The pairwise correlations in the 1983 and 1984 collections were almost identical in terms of statistical significance, and the majority of such significant correlations were common between the 1973 and the two later collections. In addition to the pairwise correlations, the higher order structures of pathogenicity associations also were very similar in all three collections. Thus, simultaneous pathogenicity or nonpathogenicity of the isolates on many differentials were maintained through both space and time. The apparent lower value of the average correlations estimated for the 1983 and 1984 collections, as compared with that of the 1973 collection, should be noted. About half of the later two collections were composed of complex races that were pathogenic on almost all of the host differentials, hence reducing the estimated values of statistical correlations. Many hypotheses can be formulated bearing on the correlated pathogenicity of the isolates on different host differentials observed in these three collections. An obvious explanation is the restriction of *R. secalis* to asexual reproduction, which may have efficiently held the entire genotype together.

A sexual stage has not been found in *R. secalis,* which makes it impossible to obtain a direct estimate of the effect of asexual reproduction on the pathogenicity association in populations of this fungus. However, some inferences regarding the evolutionary processes underlying the highly conserved pathogenicity associations can be made by comparing the results of the present study with theories and previous experimental findings. Person et al (16) discussed the relationships among recombinantion rate, population size, and number of mutants maintained in the population in asexual populations and suggested that such populations have enormous potential for maintaining new mutants. Maynard-Smith (11) concluded that for haploid populations that were sufficiently large, asexual reproduction need not alter the rate of evolution. More interestingly, Hansen and Mangus (6), Jackson and Webster (8), and Newman and Owen (15) all reported that new races (gene combinations) were generated at high rates in *R. secalis* when the races were mixed, suggesting that recombination may be frequent in this pathogen. Although Hansen and Mangus (6) used almost the same set of host differentials as ours, they did not detect any pathogenicity associations between different host differentials among isolates of *R. secalis* collected throughout Norway.

Thus, theory and experimental evidence suggest that pathogenicity association is not necessarily a feature of asexual populations. Furthermore, asexual reproduction in the absence of selection would not be sufficient to hold the genotype together. In conjunction with the striking differences of race compositions in these three collections that cover a time span of 11 yr, we favor the hypothesis that the highly conserved pathogenicity associations reflect gene complexes in the pathogen genome that were simultaneously favored by natural selection.

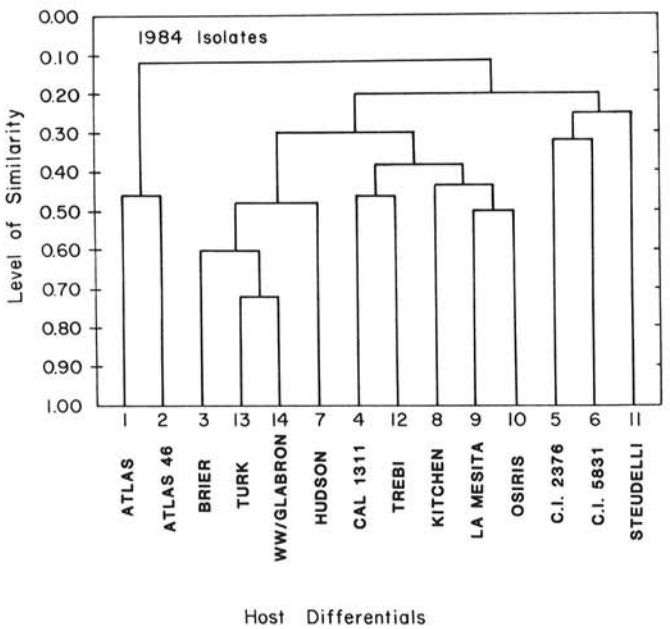

Fig. 3. The cluster structure showing the associations of pathogenicity to 14 barley host differentials among the *Rhynchosporium secalis* isolates of the 1984 collection.

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