Variation in Virulence Within the Population of *Pyrenophora tritici-repentis* in New York

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ABSTRACT

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Seventeen isolates of *Pyrenophora tritici-repentis*, originating from diverse locations in New York, Maryland, and Ontario (Canada), differed in their virulence on 12 wheat cultivars, as measured by the percentage of total seedling leaf area that was necrotic. Highly significant cultivar, isolate, and cultivar × isolate effects were observed, indicating a differential host-pathogen interaction. Similarity of isolates of *P. tritici-repentis* was determined by cluster analysis based on their patterns of virulence on the 12 wheat cultivars. Isolates did not differ widely, indicating only a moderate degree of physiologic specialization in the populations of *P. tritici-repentis* under study. Similarity of wheat cultivars was also determined based on their patterns of resistance to 17 isolates of *P. tritici-repentis*. Winter wheat cultivars currently grown in New York possess moderate to high resistance, whereas spring wheat cultivars are susceptible to *P. tritici-repentis*. For wheat breeding purposes, screening wheat genotypes with a few virulent isolates may suffice in selection for a moderate level of resistance to *P. tritici-repentis*. Cluster analysis may be a useful tool to discern similarities among pathogen isolates and among host genotypes in quantitative host-pathogen systems, but experimental variation may influence cluster composition.

Additional keywords: cluster analysis, *Drechslera tritici-repentis*, epidemiology, tan spot, *Triticum aestivum, T. durum*.

The concept of specificity in host-pathogen interactions has both theoretical and applied relevance to the understanding and control of many plant diseases. True specificity implies that genetic variation in the host and the pathogen are correlated and may affect the durability of host resistance to the pathogen (20). Physiologic specialization, i.e., differential adaptation of pathogen isolates to certain host genotypes, could also complicate screening strategies in the development of disease-resistant host varieties. This is certainly important in host-pathogen systems where resistance is governed by major genes and distinct physiologic races can be identified. However, isolate-cultivar specificity should also be a consideration in quantitative host-pathogen systems, where physiologic specialization may be less obvious and based on quantitative differences in disease expression. The latter category includes fungal leaf spot diseases of cereals, such as those caused by *Phaeosphaeria nodorum* (E. Müller) Hedja. (= *Leptosphaeria nodorum* E. Müller), and *Mycosphaerella*

graminicola (Fckl.) Sand. These pathogens have been reported to exhibit some degree of isolate-cultivar specificity (1,9,10,31,33), and concern has arisen about the stability of resistance to these pathogens (1,9,30).

Pyrenophora tritici-repentis (Died.) Drechs. (anamorph: *Drechslera tritici-repentis* (Died.) Shoem.), the incitant of tan spot, a major disease of wheat (*Triticum aestivum* L., *T. durum* Desf.) in many regions of the world (14,18,28,35), has also been reported to exhibit isolate-cultivar specificity (6,19,24), although one study indicated a lack of such (7). Resistance to *P. tritici-repentis* is quantitatively inherited in wheat (15,25,29); however, single genes with major effects have been implicated in resistance in several wheat genotypes (12,13).

Tan spot is an important component of the leaf spot complex on spring and winter wheat in New York (4,5,34). Wheat cultivation in New York differs from that in other regions where tan spot has been studied, in that wheat (primarily soft white winter) is usually grown only once in long-term rotations, and most fields are isolated geographically from each other. Temporal and spatial aspects of host and pathogen distribution in this system of wheat cultivation may have resulted in increased heterogeneity of the populations of *P. tritici-repentis* under study.

The objective of this study was to determine whether isolates of *P. tritici-repentis* from populations in New York and surrounding regions exhibit physiologic specialization, and to select those isolates most appropriate for resistance screening of wheat genotypes. Concurrently, the relative susceptibility of soft white winter wheat and hard red spring wheat cultivars presently grown and recommended in New York was determined.

MATERIALS AND METHODS

The host. Twelve wheat cultivars were selected as differentials for this study, based on a preliminary assessment of the ability of 17 cultivars to differentiate four to seven isolates of *P. tritici-repentis*. In a similar study by da Luz and Hosford (6), the spring wheat cultivars ND 495, BH 1146, Duri, and Chris and the durum cultivar Wells were shown to differentiate isolates of *P. tritici-repentis*. The soft white winter wheat cultivars Houser, Geneva, and Frankenmuth and the hard red spring wheat cultivars Sinton and Max are commonly grown in New York and are the source of all New York isolates of *P. tritici-repentis* used in this study. The Brazilian spring wheat line BR 8 has been reported to have moderate resistance (4), and CI 02057, a winter wheat accession obtained from the Department of Plant Pathology at Cornell University, also showed moderate resistance to *P. tritici-repentis* in preliminary experiments.

Plants were grown in standard potting mix (containing 0.6 kg/m^3 superphosphate and 0.9 kg/m^3 potassium nitrate) in 27 × 53 × 6 cm plastic flats containing 36 cells (Kord Products Inc., Bramalea, Ontario, Canada), at two plants per cell. Plants were grown in a controlled environment chamber at a constant temperature of 22 ± 1 C with 14 hr of fluorescent light (372 μE m^{-2} sec^{-1}) per day.

The pathogen. Seventeen single-conidium isolates of *P. tritici-repentis*, which originated from New York and surrounding regions, were evaluated in this study. Fourteen isolates were collected from separate spring and winter wheat fields in central and western New York, two isolates originated from wheat breeding plots in Keedysville, MD, and one isolate was obtained from wheat plots in Arkell, Ontario (Canada) (Table 1). Isolates of the fungus were maintained on V-8 juice agar (8) in petri plates and slants at 4 C. Because the isolates were collected over a period of 4 yr, some reduction in virulence due to prolonged storage of some of the isolates could have occurred. To minimize variation caused by age differences of the cultures, 3-wk-old plants of the susceptible cultivar Max were inoculated with separate isolates, which were subsequently reisolated from lesions as single conidia. The resulting cultures were used in both repetitions of the experiment.

Inoculum was produced by transferring mycelial plugs to petri plates containing V-8 juice agar. The cultures were incubated at 20–25 C on the laboratory bench for 5 days. The aerial mycelium was then removed by scraping the plates with a flamed metal spatula. The scraped cultures were maintained under near-ultraviolet light (from General Electric F40BL lamps) at 20–25 C for 20–24 hr to induce production of conidiophores, followed by incubation in the dark at 20–25 C for 20–24 hr to induce production of conidia. Conidial suspensions were prepared by flooding the plates with distilled water and scraping the culture surfaces with a metal spatula to dislodge the spores. A hemacytometer was used to make conidial counts. The concentration of each suspension was adjusted to approximately 2,000 conidia per milliliter by dilution with distilled water. A drop of Tween 20 (polyoxyethylene sorbitan monolaurate) was added per 100 ml of suspension to ensure uniform wetting of the leaves.

Inoculation procedure. The experimental design was of a split-plot nature with individual isolates of *P. tritici-repentis* randomly assigned to flats of wheat plants as main plots, and cultivars randomly assigned to cells (two plants per cell) within flats as subplots. Main plots were not replicated. Cultivar subplots were replicated three times in a randomized complete block design within the isolate main plot. At 3 wk of age, all plants in a flat were inoculated at once by being sprayed with a conidial suspension of the assigned isolate until runoff with a hand paint sprayer (model SMC, Sanborn Manufacturing Company Springfield, MN) at a pressure of about 0.5 kg/cm^2.

TABLE 1. Identity of isolates used in the study of physiologic specialization within the population of *Pyrenophora tritici-repentis* in New York and surrounding regions

Isolate number	Isolate	Location	Year of isolation	Wheat cultivar (market class)
1	Ptr002NY85	Onondaga Co., NY	1985	Sinton (HRS)[a]
2	Ptr003NY85	Ontario Co., NY	1985	Sinton (HRS)
3	Ptr004NY84	Ontario Co., NY	1984	Max (HRS)
4	Ptr005NY84	Onondaga Co., NY	1984	Sinton (HRS)
5	Ptr006NY85	Niagara Co., NY	1985	Max (HRS)
6	Ptr007NY85	Ontario Co., NY	1985	Sinton (HRS)
7	Ptr009NY86	Avoca, Steuben Co., NY	1986	Houser (SWW)[a]
8	Ptr010NY86	Newfield, Tompkins Co., NY	1986	Frankenmuth (SWW)
9	Ptr011NY86	Rush, Monroe Co., NY	1986	Frankenmuth (SWW)
10	Ptr012NY86	Waterloo, Seneca Co., NY	1986	Frankenmuth (SWW)
11	Ptr013MD87	Keedysville, MD	1987	Severn (SRW)[a]
12	Ptr014NY86	Fleming, Cayuga Co., NY	1986	Houser (SWW)
13	Ptr015NY87	Weedsport, Cayuga Co., NY	1987	Frankenmuth (SWW)
14	Ptr016MD87	Keedysville, MD	1987	MD73025-51 (SRW)
15	Ptr017NY87	Corfu, Genesee Co., NY	1987	Geneva (SWW)
16	Ptr018ON87	Arkell, Ontario, Canada	1987	Not known
17	Ptr019NY86	Geneva, Seneca Co., NY	1986	Houser (SWW)

[a]HRS = hard red spring wheat, SWW = soft white winter wheat, SRW = soft red winter wheat.

Approximately 5 ml of inoculum was applied to each cultivar subplot, the experimental unit. After a drying period of approximately 30 min to ensure adherence of the spores, the flats were placed in a mist chamber for 30 hr of continuous leaf wetness at 22 ± 2 C. Plants were fan-dried and returned to the controlled environment chamber (22 ± 1 C; 14 hr of fluorescent light [372 μE m^{-2} sec^{-1}] per day). After 7–8 days, disease severity was assessed as the percentage necrosis on the total leaf area, using the disease rating keys devised by James (16). The experiment was performed twice under the same light and temperature regime.

Analysis. Data were analyzed using the SAS computer software package (Statistical Analysis Systems Institute Inc., Cary, NC). Disease severity means (Table 2) of each isolate-cultivar combination in the individual and combined experiments were subjected to an analysis of variance using the SAS ANOVA procedure for a split-plot experiment. An analysis of variance was also performed after a square root transformation of the data. Least significant difference values were calculated for comparison of means within and between rows. A hierarchical agglomerative cluster analysis procedure was applied to the disease severity means (Table 2) to determine the similarity of the isolates of *P. tritici-repentis* based on their patterns of virulence on the 12 wheat cultivars and of the wheat cultivars based on their patterns of resistance to the 17 isolates of *P. tritici-repentis*. Cluster analysis was performed on the results of both experiments, separately and combined. The cluster analysis was executed by the SAS CLUSTER procedure, using the centroid method (32). In the centroid method, which is considered relatively unbiased, the distance between two clusters is defined as the squared Euclidian distance between their centroids or means (32). McQuitty's similarity analysis (SAS CLUSTER procedure) was also applied to the data to determine whether cluster analysis method affected cluster composition.

In addition, the variance among wheat cultivars in disease reaction to specific isolates of *P. tritici-repentis* was regressed against mean virulence of the isolates (percentage of leaf area necrotic). Similarly, the variance among isolates in virulence to specific wheat cultivars was regressed against mean resistance of the cultivars (percentage of leaf area necrotic). This method was proposed by Carson (3) to predict possible modes of host-pathogen interaction in the pathosystem of *P. tritici-repentis*.

RESULTS

All isolates of *P. tritici-repentis* were pathogenic to all wheat cultivars used in the experiment, but differences in virulence, as measured by the amount of necrosis incited on the leaf area, were observed (Table 2). The analyses of variance of the disease severity in the two experiments (Table 3) showed highly significant effects of cultivar, isolate, and cultivar × isolate on disease expression. When the experiments were combined (Table 4), the cultivar and cultivar × isolate effects remained highly significant, but the isolate effect was no longer significant, because of highly significant isolate × experiment and cultivar × isolate ×

TABLE 2. Necrosis (percentage of leaf area) incited by 17 isolates of *Pyrenophora tritici-repentis* on 12 wheat cultivars

	Wheat cultivar											
Isolate	Geneva	Wells	Duri	BR8	BH1146	Houser	Frankenmuth	CI02057	Chris	Max	ND495	Sinton
1	6.5[a]	4.3	4.7	4.5	6.0	7.2	7.5	9.0	17.2	18.8	15.7	29.5
	7.0[b]	2.3	3.0	10.0	6.7	6.5	8.0	11.5	10.0	29.5	15.7	23.5
2	7.1	5.8	8.3	15.5	13.5	9.5	7.5	28.2	26.8	30.5	41.2	50.8
	10.0	7.8	11.8	20.3	12.7	14.5	15.2	23.3	28.0	40.2	39.2	48.0
3	16.2	15.5	14.2	12.0	18.2	21.2	16.0	27.0	45.2	22.8	54.7	45.3
	6.0	10.5	6.0	6.0	12.3	8.2	8.2	8.7	31.5	28.5	34.2	30.0
4	6.2	6.7	7.3	12.0	13.3	10.5	12.2	19.3	21.8	33.7	26.3	39.5
	6.7	5.2	6.2	13.3	8.3	13.7	9.3	19.2	14.2	29.2	30.3	36.7
5	7.0	10.3	8.5	10.8	14.2	6.8	9.3	19.2	24.2	29.3	48.2	45.8
	7.3	9.5	9.3	10.5	8.3	11.5	13.7	21.3	19.3	27.5	22.3	40.0
6	15.5	13.5	12.7	13.2	13.8	13.5	15.5	19.2	32.8	21.3	50.0	33.8
	6.5	8.0	5.4	9.3	11.2	11.5	12.8	11.8	32.3	26.3	27.2	33.3
7	9.3	17.3	10.2	7.5	13.5	13.5	10.5	19.7	31.2	24.7	43.8	39.7
	9.7	12.0	7.3	13.0	16.7	11.2	10.2	14.7	40.3	31.5	49.3	42.5
8	12.7	14.5	10.5	17.0	13.3	12.2	18.3	26.5	31.3	24.7	22.3	44.2
	6.0	9.3	4.3	14.0	14.8	22.8	15.5	19.7	36.7	37.5	39.8	37.3
9	11.2	10.8	12.0	9.7	12.2	15.5	13.8	15.2	51.7	26.7	52.5	37.5
	7.5	6.5	9.3	7.7	13.7	11.7	8.7	12.5	40.2	21.3	32.5	31.3
10	8.3	20.0	8.2	10.7	12.7	11.0	13.3	15.3	38.8	30.5	40.8	40.0
	9.5	8.3	4.2	6.8	10.3	10.7	9.0	11.0	27.7	19.2	25.8	27.8
11	15.0	21.8	4.3	10.2	13.3	7.7	11.3	23.3	22.8	26.8	36.2	42.7
	6.0	7.8	2.5	12.2	6.7	10.7	5.0	18.0	23.8	33.2	22.8	41.0
12	8.7	7.5	3.8	6.0	10.0	8.3	5.0	16.2	25.7	10.3	32.2	23.3
	17.5	21.5	21.3	16.5	20.2	23.7	23.3	19.7	50.8	41.5	58.7	40.3
13	10.2	16.0	7.5	4.8	7.5	17.3	13.8	6.2	34.3	17.0	40.0	29.8
	17.5	37.0	17.5	13.7	19.7	18.5	31.2	24.0	40.8	45.5	53.2	45.8
14	11.7	9.0	16.5	10.5	20.2	14.5	10.7	14.5	33.2	29.0	49.2	38.2
	6.7	7.8	4.7	5.2	8.7	9.2	7.0	9.3	30.0	28.0	38.3	26.3
15	12.8	12.0	6.8	6.7	10.8	10.5	12.5	14.5	30.5	20.0	34.8	29.8
	10.3	6.8	2.7	6.2	13.0	11.3	7.8	13.3	29.2	29.2	24.5	25.8
16	21.3	21.8	22.8	16.8	22.2	23.0	24.2	19.3	44.2	32.7	45.5	42.0
	14.2	15.8	20.0	15.2	21.3	31.2	24.3	26.3	44.2	41.0	46.7	52.5
17	12.7	14.5	10.5	17.0	13.3	12.2	18.3	26.5	31.3	24.7	33.2	44.2
	11.7	14.0	10.0	19.3	14.5	9.7	12.8	25.2	38.8	43.8	46.2	46.7

[a]Experiment 1, $LSD_{0.05} = 5.09$.
[b]Experiment 2, $LSD_{0.05} = 5.98$.

experiment interactions. Cultivar × experiment interactions occurred as well. The cultivar × isolate interaction remained highly significant, however, even when tested against the cultivar × isolate × experiment interaction. Square root transformation of the data did not affect the outcome of the analysis of variance, and the untransformed data were used for the remaining analyses. Differences between the virulence patterns of the isolates of *P. tritici-repentis* are apparent (e.g., compare virulence of isolates 5 and 9 on the wheat cultivars Max and Chris), but not of a large magnitude (Fig. 1). The differences are sufficiently large, however, to make a cultivar appear moderately resistant or susceptible, depending on which isolate is used. Two large groups of similar isolates (isolates 2, 4, 5, 8, 11, 17, and isolates 3, 6, 7, 9, 10, 12, 14, 15, respectively) were identified by cluster analysis of the combined experiments using the centroid method (Fig. 2). Isolates from New York winter wheat fields predominate in the latter group, whereas half of the isolates from New York spring wheat fields are contained in the former group. Maryland isolates 11 and 14 differ from each other, as each is associated with a different group of New York isolates. The virulence pattern of Canadian isolate 16 was quite different from the others, as were the virulence patterns of New York isolates 1 and 13. No strong associations between virulence patterns and isolate origin, with regard to location, wheat cultivar, or market class, were otherwise observed. A trend toward lower average virulence exists, however, among isolates of *P. tritici-repentis* collected from spring wheat in 1984 and 1985, as compared to isolates collected from winter wheat in 1986 and 1987. Thus, average virulence may be correlated with isolate age and/or wheat market class of origin.

Wheat genotypes differed significantly in their degree of resistance to *P. tritici-repentis*, although none were immune. As determined by cluster analysis of resistance patterns in the combined experiments, ND 495, Sinton, Chris, and Max were similarly susceptible to the 17 isolates of *P. tritici-repentis* (Fig. 3). The other cultivars were grouped in a distinctly different cluster. The highly resistant cultivars Geneva and Duri and moderately resistant Wells were very similar in their reaction patterns to the isolates of *P. tritici-repentis*, as were the moderately resistant Houser, Frankenmuth, BH 1146, and BR 8. CI 02057 may be considered intermediate in resistance.

TABLE 3. Analysis of variance for the amount of necrosis incited on 12 wheat cultivars by 17 isolates of *Pyrenophora tritici-repentis*

Source of variation	Experiment 1		Experiment 2	
	df	MS[a]	df	MS[a]
Isolate	16	594.66*	16	1,260.62*
Main plot error	34	11.14	34	20.57
Cultivar	11	6,166.90*	11	6,299.27*
Cultivar × isolate	176	72.15*	176	71.34*
Subplot error	371	10.03	374	13.96
Total df	608	...	611	...

[a] *F-value highly significant ($P < 0.01$).

TABLE 4. Analysis of variance for the amount of necrosis incited on 12 wheat cultivars by 17 isolates of *Pyrenophora tritici-repentis* (experiments 1 and 2 combined)

Source of variation	df	MS[a]
Experiment	1	10.30
Isolate	16	1,030.65
Experiment × isolate	16	824.64*
Cultivar	11	12,262.26*
Cultivar × experiment	11	203.92*
Cultivar × isolate	176	96.35*
Cultivar × isolate × experiment	176	47.15*
Error	813	12.33
Total df	1,220	...

[a] *F-value highly significant ($P < 0.01$).

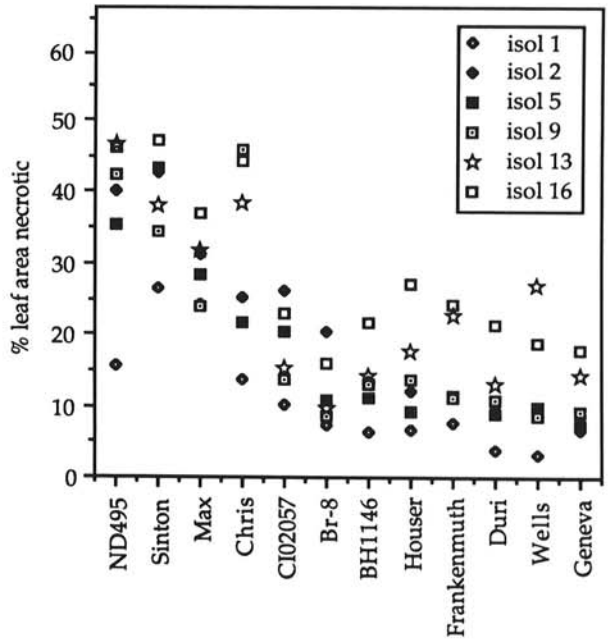

Fig. 1. Amount of necrosis incited on 12 wheat cultivars by isolates representative of the population(s) of *Pyrenophora tritici-repentis* in New York and surrounding regions (average of two experiments).

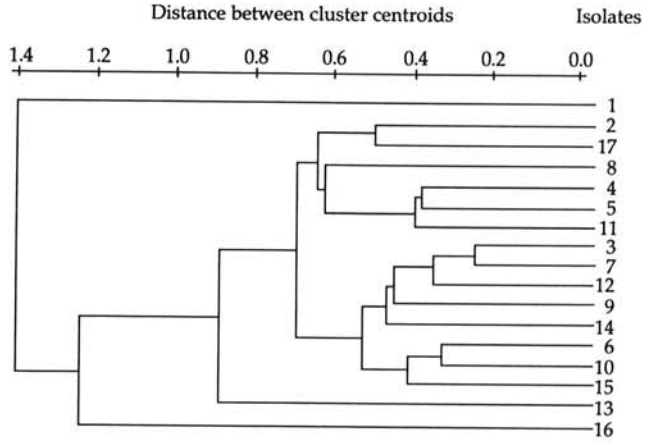

Fig. 2. Dendrogram showing similarity and successive clustering of isolates of *Pyrenophora tritici-repentis* based on their virulence on 12 wheat cultivars (average of two experiments).

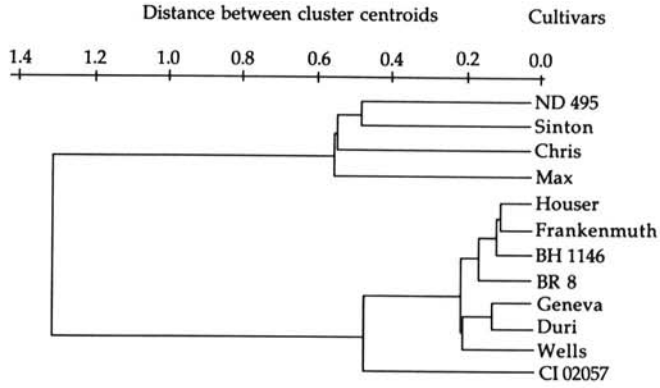

Fig. 3. Dendrogram showing similarity and successive clustering of wheat cultivars based on their resistance to 17 isolates of *Pyrenophora tritici-repentis* (average of two experiments).

Some variation in cluster composition and isolate position within the clusters was observed between the two experiments. Cultivar clusters exhibited less variation between experiments. Cluster analysis was therefore felt to be more reliable if performed on the data of the combined rather than the single experiments. Changing the clustering method affected the similarity relationships within the clusters only slightly.

The variance in virulence among isolates was positively correlated with mean host resistance (Fig. 4), and the variance in disease reaction among wheat cultivars was positively correlated with mean pathogen virulence (Fig. 5). Thus, in general, the more susceptible cultivars differentiated the isolates better than the more resistant cultivars, and the more virulent isolates differentiated the host cultivars better than the less virulent isolates.

DISCUSSION

Specificity in host-pathogen relationships is often indicated by significant isolate × variety interactions in the analysis of variance of an experiment where a number of pathogen isolates are tested in all possible combinations on a set of host genotypes. Nonspecificity is identified by a lack of such interaction (37,38). The use of the analysis of variance method to determine specificity is not without problems, however, as significant cultivar × isolate interactions may occur in nonspecific pathosystems, resulting from cultivar × environment, isolate × environment, and/or isolate × cultivar × environment interactions (20,26). Cultivar × isolate effects may also occur because of the lack of a proper scale for measuring disease severity (39). On the other hand, environmental effects may confound cultivar × isolate interaction terms and make them appear nonsignificant. This occurs if the differential effects depend on environment (17). Rufty et al (31) reported significant differences attributable to experiments when testing isolates of *Phaeosphaeria nodorum* on a series of wheat differentials. The magnitude of the isolate × cultivar interaction in this study, despite significant interactions with uncontrolled environmental factors between experiments, suggests a real differential interaction between isolates of *P. tritici-repentis* and wheat genotypes. This is in agreement with the results of previous studies (6,17,22). Possible factors that may have contributed to the variation between experiments are: differences in microclimate, uniformity of inoculum coverage, and condition of the inoculum. The latter factor may have had the greatest effect, as differences in sporulation and concentration of unquantified infective propagules (mycelial fragments and conidiophores) were observed between experiments for some isolates. Differences in disease reaction between experiments were most obvious on the more susceptible cultivars.

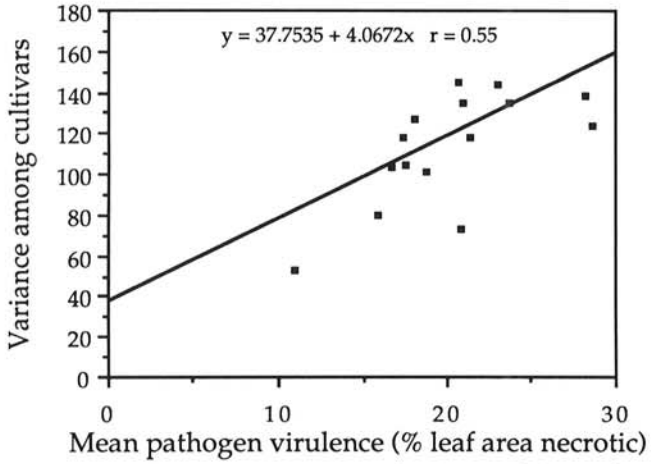

Fig. 4. Relationship of the mean virulence of 17 isolates of *Pyrenophora tritici-repentis* and the variance in disease reaction to the respective isolates among 12 wheat cultivars (average of two experiments).

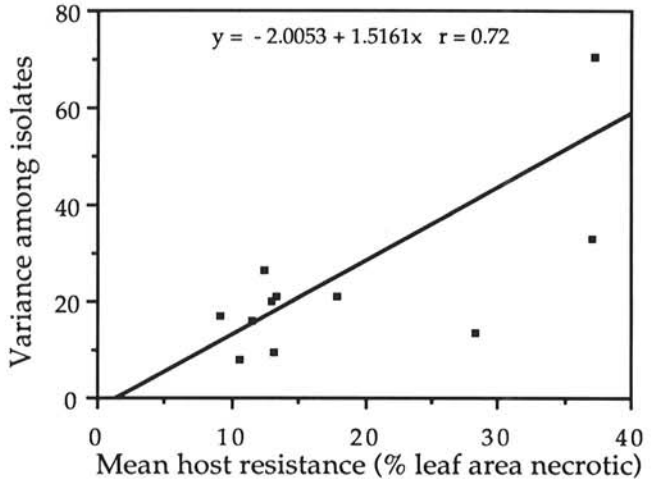

Fig. 5. Relationship of the mean resistance of 12 wheat cultivars and the variance in disease induction on the respective cultivars among 17 isolates of *Pyrenophora tritici-repentis* (average of two experiments).

Although the isolates were relatively few in number, they were quite varied in their origin and may represent a wide range of virulence phenotypes. The virulence patterns that were identified among the 17 isolates did not differ widely and should not be considered to represent actual races. Physiologic specialization therefore appears to be moderate in the population(s) of *P. tritici-repentis* under study, and rapid adaptation to a particular host cultivar and loss of effective resistance appear unlikely. However, results of resistance screening in wheat improvement programs could change considerably, depending on the isolate used.

The mechanisms underlying specificity in the pathosystem of *P. tritici-repentis* are not clear. Resistance in BH 1146 is expressed by restriction of fungal development within the mesophyll tissue (21,23). Differential host sensitivity to the toxin(s) produced by this pathogen and differential accumulation of antifungal substances by the host may be involved (23,36). Other mechanisms of resistance may be present in different cultivars but have not been studied. Observations in this experiment suggest that a reduction in the number of lesions may confer resistance to some cultivars. Differential production of toxin(s) by isolates of *P. tritici-repentis* may also play a role in specificity. Isolates are known to vary in toxin production in vitro (2), but a clear correlation between toxin production and virulence has not been found (A. M. C. Schilder, *unpublished data*).

Based on analysis of data of da Luz and Hosford (6), Carson (3) proposed three possible models of quantitative host-pathogen interaction for the pathosystem of *P. tritici-repentis*: 1) the interaction for susceptibility model (alleles for aggressiveness are expressed only when matched by corresponding alleles for susceptibility in the host), 2) the multiplicative model (final disease reactions are determined by the product of host resistance and pathogen aggressiveness alleles) (11), and 3) the interactive-multiplicative model (a multiplicative model allowing for interaction of host and pathogen alleles). Analysis of theoretical disease severities indicated that resistance in all three models would be stable and would not erode due to increasing numbers of aggressiveness alleles in the pathogen population (3). This followed from the observation that, as resistance genes accumulated in the host population, the genetic variance of the pathogen population decreased, as well as the relative fitness (as measured by disease severity) of the most aggressive pathogen isolates. It was suggested that the most aggressive isolate(s) would maximize the genetic variance among host phenotypes and should be used in screening for resistance to maximize gain from selection in the development of cultivars resistant to *P. tritici-repentis* (3). It should be noted that Carson used the term "aggressiveness" to denote the amount of disease incited by isolates in horizontal, i.e., nonspecific, pathosystems. We use the term "virulence" to describe the amount of disease incited in the pathosystem of *P. tritici-repentis* since a certain degree of specificity is observed. The results of the present study substantiate the findings of Carson,

as an increase was noted in the variance of isolate virulence with increasing average host susceptibility, and a similar increase in the variance of cultivar disease reaction was observed with increasing average virulence of the isolates. Consequently, in screening wheat genotypes for resistance to *P. tritici-repentis*, it is advisable to use the most widely virulent isolate(s). When conditions are conducive to disease development and resistance in currently grown cultivars is low, isolates representing the range of virulence patterns should be used in screening wheat genotypes for resistance to provide a basis for stringent selection. However, the use of only a few virulent isolates for screening purposes appears to be sufficient in situations where high levels of resistance are not required, such as when current wheat cultivars possess adequate resistance and the inoculum level of *P. tritici-repentis* tends to be low. This is generally the case in winter wheat production in New York (34).

The soft white winter wheat cultivars presently grown and recommended in New York have moderate to high, and presumably stable, resistance to the isolates of *P. tritici-repentis* tested. The two common spring wheat cultivars, Sinton and Max, proved to be susceptible to most isolates of *P. tritici-repentis* tested. The differential susceptibility partially explains the greater prevalence of tan spot observed in spring wheat fields (4,5) than in winter wheat fields (34) in New York.

The application of cluster analysis has been suggested previously for assessing genetic similarity and/or dissimilarity in gene-for-gene host-parasite relationships (22,27). The method was used to express exactly the genetic similarity among 48 physiological races of *Bremia lactucae* Regel, the causal agent of downy mildew of lettuce (22). Cluster analysis was useful in understanding the quantitative host-pathogen relationship under study. However, the procedure appears rather sensitive to variation between experiments, which was particularly evident in the clustering of isolates. This may be partially attributed to the relatively small differences in patterns of virulence among the isolates in this pathosystem. Therefore, it may not be advisable to base conclusions about the virulence composition of such a pathogen population on a single experiment.

Cluster analysis also proved to be useful in determining the similarity of wheat cultivars, based on their reactions to 17 isolates of *P. tritici-repentis*. The set of selected cultivars differentiated the isolates reasonably well and may be used to characterize additional isolates. The number of cultivars in the differential set may be reduced by using cluster analysis to those that differentiate the isolates most efficiently.

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