Pathotype Diversity of *Pyrenophora teres* f. *teres* on Barley

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We thank Larry Yost for technical assistance and Marion E. Stanley, Farm Advisor, Monterey County, for help in locating barley fields. This research was supported in part by the California Crop Improvement Association.

Accepted for publication 5 July 1991 (submitted for electronic processing).

ABSTRACT

Steffenson, B. J., and Webster, R. K. 1992. Pathotype diversity of *Pyrenophora teres* f. *teres* on barley. Phytopathology 82:170-177.

From 91 isolates collected in California, 13 pathotypes of *Pyrenophora teres* f. *teres*, the causal organism of net blotch of barley, were identified on 22 differential barley genotypes. Pathotype 3-10-15-19-21 (numbers correspond to the differential barley hosts that exhibit a high infection response [HIR] to this pathotype) was most prevalent, comprising 28.6% of the isolates. The next most common pathotypes included 15, 15-20, and 3-10-15-19-20-21 with 19.8, 15.4, and 8.8% of the isolates, respectively. Pathotype 3-10-15-19-20-21 was virulent on the greatest number of host genotypes (six), whereas pathotype 0 was not virulent on any of the hosts. Prato, Kombar, and Atlas were the individual host differentials susceptible (exhibited HIRs) to the greatest percentages of isolates: 82.4, 56.0, and 50.5%, respectively; however, 10 of the 22 differential genotypes were resistant (exhibited low infection responses) to all of the pathotypes identified in California. Pathotypes from Minnesota, Mexico, and England were distinctly different from each other. Mexican pathotypes were similar to the prevalent California pathotypes in virulence on specific differential genotypes. Pathotypes from Minnesota (1-6-13-16-18) and England (22) differed from two rare California pathotypes (6-13-16-18 and 11-22) for virulence on one differential genotype. The structure and diversity of isolates from two populations were compared. One population consisted of 25 isolates sampled randomly at a field station (Armstrong population) and the other of 59 isolates collected from random fields around the state (off-station population). The most complex pathotypes, 3-10-15-19-21 and 3-10-15-19-20-21, were present at a higher frequency in the Armstrong population (72.0%) than in the off-station population (23.7%); however, the latter population was genetically more diverse (Shannon index = 1.96) than the former (1.30). The set of barley differentials used in this study was effective for typing the virulence phenotypes of a wide collection of isolates of *P. t.* f. *teres* and should be useful to other workers investigating this host/parasite system.

Net blotch of barley (*Hordeum vulgare* L. emend Bowden) is caused by the fungus *Pyrenophora teres* Drechs. f. *teres* Smedeg. (anamorph: *Drechslera teres* [Sacc.] Shoem. f. *teres* Smedeg.) and is common throughout the major barley-growing regions of the world. Mathre (29) reported losses nearing 100% in some highly susceptible barley cultivars, but losses of 10–40% are more common with this disease. In 1979, a severe epidemic of net blotch developed on the cultivar Kombar (CI 15694) in California (44). This epidemic may have resulted because of an increase in a previously rare pathotype (subdivision of a species based on specific characters of virulence exhibited on a set of differential host genotypes—adapted from Holliday [18]); however, it is also possible that Kombar was susceptible to the prevalent pathotypes in the area but was not thoroughly evaluated for resistance. Regardless of the reason, this epidemic demonstrates the importance of determining the variation in virulence among isolates of *P. t.* f. *teres* and the need for rigorous evaluations of germ plasm for disease resistance.

The net blotch pathogen is variable in a number of cultural and morphological characters (40). In addition, this pathogen can induce different symptoms in barley. Smedegård-Petersen (42) recognized two forms of *P. teres* based on symptomatology: *P. t.* f. *teres* (the net form) causes typical net blotch lesions that have dark brown striations extending longitudinally and transversely within a lesion to form a netlike pattern, whereas *P. t.* f. *maculata* (the spot form) produces dark brown circular or elliptical lesions without netting. Spot form lesions are encircled by varying widths of chlorosis and are often associated with water-soaked tissue (42). To date, only *P. t.* f. *teres* has been found in California.

Variation in virulence among isolates of *P. t.* f. *teres* was first reported by Pon in 1949 (34). A number of studies have given indirect evidence for the existence of pathotypes by documenting a susceptible response on barley genotypes previously reported to be resistant in other countries. From these reports, it is difficult to state with certainty the presence of distinct pathotypes because this host:parasite interaction is markedly altered by environmental, nutritional, and ontogenetical factors in the host and pathogen (40). Pathotypes of *P. t.* f. *teres* have been reported from Australia (22,26), Canada (46–48), Denmark (42), Egypt (10), Israel (20), Morocco (3), Poland (13), Tunisia (3), the United Kingdom (B. C. Clifford, *personal communication*), the United States (3,14,41), and the USSR (2,27). The objective of this study was to determine the degree of variation in virulence among California isolates of *P. t.* f. *teres*. Additionally, isolates from Minnesota, Mexico, and England were included for comparison.

MATERIALS AND METHODS

Survey and collection of isolates. Ninety-one isolates of *P. t.* f. *teres* were collected from barley in the major cereal-producing regions of California during 1984–1986. Twenty-five of the 91 isolates were collected in random samplings of unknown barley genotypes at the Armstrong Plant Pathology Field Station in northeast Solano County. These isolates allowed us to characterize the virulence spectrum of *P t.* f. *teres* near the area where most of the California barley germ plasm is screened for resistance to the pathogen. Collections outside the field station were made as far north as Butte County and as far south as Kern County. The largest plantings of barley in California are located in the southern San Joaquin Valley and the south-central coast region, primarily in Fresno, Kings, San Luis Obispo, Tulare, Kern, and Monterey counties (listed in descending order of hectares planted); however, most of the *P. t.* f. *teres* isolates were collected in Monterey, San Luis Obispo, and Kings counties, since there was a higher incidence and severity of disease in these districts during

the years of the survey. Survey stops were made at commercial fields (the identity of cultivars was not known) approximately every 15 km or until the next barley field could be located. Two or three separate leaf samples were made about 10 m apart in each field following a single diagonal transection. For the characterization of the virulence spectrum of isolates, a single leaf sample from each field was used, except in seven cases where two different leaf samples from the same field were evaluated. The surveys were made from February to April of each year, when the growth stages of barley ranged from early tillering to mid-dough. Counties were grouped into valley regions (19) as follows: the Northern Valley included Butte, Colusa, Glenn, Shasta, and Sutter counties; the Middle Valley included Sacramento, San Joaquin, Solano, Stanislaus, and Yolo counties; the Southern Valley included Fresno, Kern, Kings, Madera, Merced, and Tulare counties; and the Coastal Valley included Monterey, San Benito, and San Luis Obispo counties. The consolidation of these counties into regions was done primarily for convenience of discussion; however, some general differences between the Northern and Southern Valley regions are apparent. For example, rainfall is consistently higher in the Northern than in the Southern Valley and barley is usually planted earlier in the former (L. F. Jackson, *personal communication*). Isolates from Minnesota (MN1A), Mexico (MexLagA, MexLagB, MexBatA, and MexStC) and England (UK80-12A) were kindly provided by Dr. Linda Treeful (University of Minnesota, St. Paul), Dr. Hugo Vivar (CIMMYT, El Batan, Mexico), and Dr. V. W. L. Jordan (University of Bristol, Long Ashton, England), respectively.

Isolation and increase of isolates. Leaf specimens were allowed to air dry after collection. In the laboratory, tissue with net blotch lesions was cut into 3-cm-length pieces, wetted in 95% ethyl alcohol for 10 s, and surface-sterilized in a 10% sodium hypochlorite solution (5.25% NaOCl) for 90 s. This tissue was rinsed in sterile distilled water, blotted on tissue paper to remove excess water, and aseptically transferred to plates containing 2% water agar. The samples received natural indirect sunlight (north exposure; photoperiod 11-15 h; and 9,688–10,106 ergs cm^{-2} sec^{-1}) on a laboratory bench at 19–23 C. All subsequent incubations of the fungus were made under the same environmental conditions. Sporulation from leaf tissue usually occurred within 3 days after transfer. Single conidia were transferred from the leaf tissue of each sample to a second water agar plate so that the germinability of the spore could be verified. After 1 day, germinating conidia were transferred to 17.7% V8 juice agar (177 ml of V8 juice, 16 g of agar, and 3 g of $CaCO_3$ per liter of H_2O) where they were allowed to grow for 2 wk.

The virulence and sporulation of the net blotch pathogen can vary greatly, especially after successive subculturing (20,28). To reduce such variation, all monoconidial isolates were increased in barley plants (cultivar Kombar) by needle injecting a water suspension of conidia (about 2 ml of 2,000 conidia per milliliter) into the stems of 4-wk-old plants. This infected tissue was used to produce the inoculum for all subsequent inoculations.

Differential genotypes. Twenty-two differential host genotypes were selected on the basis of whether they had been reported to possess specific genes for resistance to the net blotch pathogen, possessed uncharacterized resistance that was thought to differ from that previously reported, or had been used by earlier workers to study variation in virulence of *P. t.* f. *teres*. The number(s) of resistance genes reported for each differential genotype (including references) and a citation list of studies that have previously used the individual differentials to type virulence in *P. t.* f. *teres* are given in Table 1. Kombar, Atlas (CI 4118), and Prato (CI 15815) were included because they have been grown as cultivars in California and are susceptible to many isolates of the net blotch pathogen.

Inoculum preparation and inoculation. All isolates used in this study were taken from source material originally increased in Kombar barley. Leaf sections (2 cm^2) were placed on V8 juice agar and incubated under the conditions described above. Two weeks later, conidia were harvested by adding about 2 ml of sterile distilled water to the plate and scraping the culture with a rubber spatula. This suspension was mixed with a magnetic stirrer, and then poured through a double layer of cheesecloth into a beaker. Conidia per milliliter were counted with a hemacytometer. The concentration of the suspension was adjusted to 2×10^4 conidia per milliliter because preliminary results indicated that this concentration was great enough to prevent disease escape and was low enough not to obscure individual infection responses.

The host differentials were sown (five to seven seeds per clump) in metal flats (50 × 35 × 9 cm) containing U.C. mix (30) and grown in a greenhouse at 15–22 C. Plants were inoculated about 2 wk after planting, when the second leaf was fully expanded.

TABLE 1. Barley genotypes used to evaluate the virulence phenotype of isolates of *Pyrenophora teres* f. *teres*

Genotype	CI[a] number	Number of resistance genes	Literature citations	
			Genetics of resistance	Use as a differential host
1. Tifang	4407-1	1-2	3,6,23,31,38	3,11,13,26
2. Canadian Lake Shore	2750	1-2	14,31	1,2,13,14,27,33
3. Atlas	4118	?	...	10,14,21
4. Rojo	5401	1	12	47[b]
5. Coast	2235	1	3	22
6. Manchurian	739	1	31	13[b]
7. Ming	4797	1-2	23,31	25,26
8. CI 9819	9819	2-3	3,6,23	3,5,13,26
9. Algerian	1179	?	...	13,21,22,25,26
10. Kombar	15694	?	...	...
11. CI 11458	11458	2	32,39	...
12. CI 5791	5791	1-3	3,7,23,26,32,39	3,5,13,14,21,25,26,37, 42,46,47,48
13. Harbin	4929	1	31	1,2,13,25,27,33
14. CI 7584	7584	1-2	3,6	3,5,21,22,25,26
15. Prato	15815	?	...	...
16. Manchuria	2330	1-2	3,9,23	13,25,26
17. CI 5822	5822	1	39	13,14
18. CI 4922	4922	2	31	...
19. Hazera	12673	?	...	...
20. Cape	1026	?	...	...
21. Beecher	6566	1	39	21,25,26
22. Rika	8069	?	...	41

[a] CI = Cereal Investigation number.

[b] Used to determine the virulence spectrum of *P. t.* f. *teres* in the United kingdom cereal pathogen virulence survey (B. C. Clifford, personal communication).

TABLE 2. Mode and range of infection responses exhibited on 22 barley genotypes to 13 pathotypes of *Pyrenophora teres* f. *teres* differentiated from 91 isolates collected from California

	0	10	15	3-10	11-22	15-20
Genotype	Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range
1. Tifang	1/1-2[b]	1/1	1/1-2(3)	2,1/1-2	1/1	1/1-3
2. Can. Lk. Sh.	1/1-3	1,2/1-3	1/1-3	2,3/1-3	1,2/1-2	1/1-3
3. Atlas	2,1/1-2(3)	5,4/4-5	2,3/1-3(4)	9,10/8-10	2,1/1-2	3,2/1-3(4,5)
4. Rojo	1/1-2	1,2/1-2	2,1/1-3	1,2/1-2(3)	2,3/2-3	1,2/1-3
5. Coast	1,2/1-2	2,3/1-3	1,2/1-2(3)	2,3/1-3(4)	2,3/1-3	1,2/1-3
6. Manchurian	3,4/3-4(5)	3/1-3(4)	3,4/2-4(1,5)	4,5/2-5(1)	3,4/3-4	4,3/2-5(1)
7. Ming	1,2/1-3	1,2/1-3	1,2/1-3(4)	2,1/1-3(4)	1,2/1-2	3,4/1-4
8. CI 9819	1,2/1-2	1,2/1-2	1,2/1-3	2,1/1-2	1,2/1-2	1,2/1-2(3,4)
9. Algerian	2,1/1-3(4)	2,1/1-2(3)	2,3/2-4(1,5)	2,1/1-3	3,2/2-3	3,2/2-4(1)
10. Kombar	2,1/1-2(3,4)	8,9/8-9(7)	2,3/1-3(4,5)	9,10/9-10	3,4/3-4	2,3/2-4(1)
11. CI 11458	1/1-2	1,2/1-3	1,2/1-3	1,2/1-2	8,9/8-9	2,3/1-3(4)
12. CI 5791	1,2/1-2	1,2/1-2	1,2/1-3	2,1/1-2	3,4/3-4	1,2/1-2(3)
13. Harbin	1/1-2(3)	1,2/1-3	1,2/1-3(4)	2,1/1-3(4)	2,1/1-2	1,2/1-3(4)
14. CI 7584	2,1/1-2	2,3/1-3	1,2/1-3(4)	3,2/2-4(5)	2,1/1-2	2,3/1-3
15. Prato	5,4/4-5	4,5/4-5	9,10/8-10(7)	5,4/3-5(2)	2,3/2-3	9,10/8-10(7)
16. Manchuria	3,4/2-4(5)	4,5/3-5(2)	4,3/2-5(1)	2,4/1-4(5)	3,4/3-4	4,5/2-5(1)
17. CI 5822	2,1/1-2	1,2/1-2	1,2/1-2(3)	2,1/1-3	2,1/1-2	1,2/1-3
18. CI 4922	1,2/1-2	2,3/2-3(1)	2,1/1-3(4)	2,3/2-4(1)	2,1/1-2	2,3/1-4
19. Hazera	3,2/2-4(1)	4,5/4-5(6)	2,3/1-3(4)	4,5/4-5(6)	2,1/1-2	3,4/2-4
20. Cape	4,5/3-5(2)	4,5/3-5(2)	5,4/3-5(2,6)	3,5/3-5(2)	3,4/3-4	8,9/7-9(6,10)
21. Beecher	1,2/1-2(3)	4,5/4-5(6)	2,3/1-3(4)	5,4/4-5(6)	2,1/1-2	2,3/1-3(4)
22. Rika	1,2/1-2(3)	1/1	1,2/1-2(3)	1,2/1-3	9,10/9-10	1,2/1-3
Total isolates:	6	3	18	4	1	14

(*continued on next page*)

[a] Isolates of *P. t.* f. *teres* were differentiated into pathotypes based on the infection response (IR) exhibited on the barley genotypes. The IRs were assessed based on the rating scale of Tekauz (45). Ratings from 1 to 5 were classified as low infection responses (LIRs) and those from 6 to 10 as high infection response (HIRs). The pathotype designation corresponds to the number of the barley genotype(s) (given in column 1 of this table) exhibiting a HIR. The 0 designation represents a pathotype that is not virulent (does not confer a HIR) on any of the 22 barley genotypes.

[b] The mode represents the most common or two most common (most prevalent type listed first) IRs observed on the barley genotypes to isolates within a designated pathotype. The range includes all IRs identified on the barley genotypes including those rarely observed (in parentheses).

A DeVilbiss atomizer was used to deliver the conidial suspension (12 ml per flat) onto the plants in each flat. Next, the plants were placed in a mist chamber (maintained near saturation by a 2.5 min misting period per 5-min cycle) at 18–22 C with a 12-h photoperiod (2,474 ergs cm^{-2} sec^{-1}). After 48 h, the plants were transferred back to a greenhouse and allowed to incubate at 20–27 C. Infection responses on the plants were scored 12–14 days after inoculation. One differential set was inoculated with distilled water as a control, and in each case, no infections were observed.

Assessment of the infection response. Infection responses were assessed based on the 10-point pictograph scale of Tekauz (45) for net blotch. This scale is qualitative and is based on lesion size and morphology. Ratings from 1 to 5 were classified as low infection responses (LIRs) and those from 6 to 10 as high infection responses (HIRs). Our interpretation of infection response 5 was different from that of Tekauz (46), who considered the lesion type indicative of host susceptibility because it exhibited considerable chlorosis and the ability to expand quickly. In this study, an infection response 5 was assigned to lesion types that exhibited a larger necrotic area than type 4, possessed a narrow band of chlorosis around the lesion, and remained restricted in width 14 days after inoculation. LIRs could be easily differentiated from HIRs because they exhibit little or no chlorosis and usually extend linearly (approximately 0.5–15 mm in length) along the veins of the leaves in a narrow stripe (approximately 0.5–1.25 mm in width). In contrast, HIRs are large (over 15 mm in length and over 1.25 mm in width) and are associated with distinct chlorotic zones that enlarge in length and especially width over time. Isolates of *P. t.* f. *teres* were differentiated into pathotypes on the basis of their virulence phenotype (the LIR/HIR criterion) on the 22 host genotypes. The isolates were retested on the barley hosts at least once using a completely randomized design.

Pathotype designations. A system for designating pathotypes is proposed based on the HIRs elicited on the 22 host genotypes to isolates of *P. t.* f. *teres* (Table 2). For example, a pathotype designated 3-10-15 denotes that this culture is virulent (results in a HIR) on 3-Atlas, 10-Kombar, and 15-Prato (the numbers of the differential genotypes correspond to those given in the first column of Tables 1 and 2) and one designated 15 is virulent only on 15-Prato. This system is similar to that devised by Black *et al* (4) and modified by Watson and Luig (49).

Measure of genetic diversity. To compare the genetic diversity of the population of isolates collected from the Armstrong field station ($N = 25$) with the one comprised of isolates collected outside the station area ($N = 59$, seven isolates were omitted from this population because they were duplicates from individual fields), the Gleason, Shannon, and Simpson indices were employed. The Gleason index describes the number of distinct phenotypes in a given sample size, whereas the Shannon and Simpson indices reflect both the number and evenness of distinct phenotypes (15). Equations for these three indices as well as considerations for their use are given by Groth and Roelfs (15). Poole (35) lists statistical procedures for calculating the variance and standard error of the Shannon index only.

RESULTS

A full range of infection responses was observed on the host genotypes to isolates of *P. t.* f. *teres* from California (Table 2). Distinct differences in infection response were exhibited on some of these host genotypes, and on the basis of the LIR/HIR criterion, 13 pathotypes were differentiated. Two different (usually consecutive) infection responses were commonly observed on individual leaves for each specific isolate × host genotype interaction; however, one and sometimes three consecutive infection responses were also recorded. The range of infection responses for isolates within a designated pathotype varied within and not between the LIR and HIR groups, although exceptions did occur; with a few isolates, a rare infection response 6 was observed among

(*continued from preceding page*)

Pathotype[a]						
3-10-15	3-10-21	10-15-19	3-10-15-19	6-13-16-18	3-10-15-19-21	3-10-15-19-20-21
Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range
1/1-2	2,1/1-2	1,3/1-3	2,1/1-2	4,5/4-5	1,2/1-3(4)	1,2/1-3
1,2/1-3	2,1/1-2	1/1	3,4/3-4	4,5/4-5	2,3/1-3(4)	1,2/1-3
8,9/7-9(6,10)	8,9/8-9	5,4/4-5	8,9/8-9	3,4/3-4	9,10/8-10(7)	9,10/8-10(6,7)
1,2/1-2	1/1	1/1-2	2,3/2-3	1,2/1-2	1,2/1-3	1,2/1-2(3)
1,2/1-2(3)	3,4/3-4(2)	1,2/1-2	4,3/3-4	2,3/2-3	2,3/1-3(4)	2,3/1-3(4)
3,4/2-5	3,4/3-4	4,3/3-5	4,3/3-4(5)	9,10/9-10	4,3/2-4(5)	5,4/3-5
1,2/1-3(4)	2,1/1-2	2/1-3	4,3/3-4	4,5/4-5	2,3/1-3(4,5)	2,1/1-4
1,2/1-2	2,1/1-2	2/1-3	3,2/2-3	2,1/1-2	2,1/1-3	1,2/1-2(3)
2,3/1-3(4)	2,1/1-2	2,3/1-3(4)	3,2/2-3	2,1/1-2(3)	3,2/1-3(4,5)	3,2/2-4(1,5)
9,10/8-10(7)	9,10/9-10	8/8-9	9,10/9-10	5,4/4-5	9,10/8-10(7)	9,10/9-10
2,1/1-3	1,2/1-2	1,3/1-4	4,5/4-5	4,5/4-5	2,1/1-3(4)	2,3/1-3
1,2/1-2	1,2/1-2	1,2/1-2	3,4/3-4	2,1/1-2	1,2/1-2(3)	1,2/1-3
1,3/1-3(4)	3,2/2-3	2/2-3	3,4/3-4	9,10/9-10	2,3/1-4	2,3/1-4
2,3/1-3	3,2/2-3	1,2/1-3	4,3/3-4	4,3/3-4	2,3/1-4	3,2/1-4
9,8/8-10	4,5/4-5	8,9/8-9	8,9/8-9	3,4/3-4(2)	9,10/8-10(6,7)	9,10/9-10(8)
4,5/3-5	2,3/2-3	2,5/2-5	5,4/4-5	9,10/9-10	4,3/3-5(1,2)	5,4/4-5
1,2/1-2	2,1/1-2	1,2/1-2	2,1/1-2	2,1/1-2	2,1/1-3	1,2/1-3
2,1/1-3	2,1/1-2	2,3/2-3	3,4/3-4	9,10/9-10	2,3/1-4(5)	3,4/2-4(5)
5,4/4-5(6)	5,4/4-5(6)	8/7-9	8,9/8-9	4,3/3-4	8,9/7-9(6,10)	9,10/8-10(7)
5,4/4-5(6)	3,2/2-3	4/3-5	5,4/4-5	5,4/4-5(6)	5,4/3-5(6)	8,9/7-9(6,10)
5,4/4-5(2,6)	8,9/8-9	3,5/3-5(6)	5,4/4-5	4,3/3-4	8,7/7-9(6,10)	8,9/7-10
1,2/1-2	1,2/1-2	1,2/1-2	2,3/2-3	2,1/1-2	1,2/1-3	1,2/1-3
6	1	2	1	1	26	8

predominant types 4–5 on the host genotypes Hazera, Cape, and Beecher.

Pathotype 3-10-15-19-20-21 was most complex, i.e., was virulent on the greatest number of host differentials, whereas pathotype 0, which was not virulent on any of the differentials, was the simplest. Two pathotypes had markedly different virulence combinations: 11-22 was the only pathotype virulent on CI 11458 or Rika (CI 8069), and 6-13-16-18 was the only one virulent on Manchurian (CI 739), Harbin (CI 4929), Manchuria (CI 2330), or CI 4922.

Pathotype 3-10-15-19-21 was most common, comprising 28.6% of the isolates (Fig. 1). Pathotypes 15, 15-20, and 3-10-15-19-20-21 were the next most prevalent with frequencies of 19.8, 15.4, and 8.8%, respectively. All other pathotypes were found in frequencies of less than 7.0%. Prato, Kombar, and Atlas were the individual host differentials susceptible (exhibited HIRs) to the greatest percentages of isolates: 82.4, 56.0, and 50.5%, respectively (Fig. 2). The next most widely susceptible group of hosts included Hazera (CI 12673), Beecher (CI 6566), and Cape (CI 1026), which gave HIRs to 40.6, 38.5, and 25.3% of the isolates, respectively. Manchurian, CI 11458, Harbin, Manchuria, CI 4922, and Rika were susceptible to fewer than 2% of the isolates. The remaining differential hosts Tifang (CI 4407-1), Canadian Lake Shore (CI 2750), Rojo (CI 5401), Coast (CI 2235), Ming (CI 4797), CI 9819, Algerian (CI 1179), CI 5791, CI 7584, and CI 5822 were resistant (exhibited LIRs) to all isolates from California.

It is difficult to generalize about the distribution of pathotypes in any one year or from any one region because the sample sizes were small; however, some salient features can be discerned from the data in Table 3. The four most common pathotypes, 3-10-15-19-21, 15, 15-20, and 3-10-15-19-20-21 were found in all three years, except pathotype 15 which was not identified in 1985. The most complex pathotypes, 3-10-15-19-21 and 3-10-15-19-20-21, comprised 72% (18/25) of isolates collected at the Armstrong Field Station (Armstrong population) in Solano County and only 23.7% (14/59) of isolates collected outside the station (off-station population). Genetic diversity was greater in the off-station population (Gleason = 2.70, Simpson = 0.85, Shannon = 1.96) than in the Armstrong population (Gleason = 1.55, Simpson = 0.70, Shannon = 1.30). The difference between the two populations for the Shannon index was statistically significant at $P = 0.01$.

Four of six isolates identified as pathotype 0 were found in an area from southwestern Tulare County and into northeastern Kern County from 1985 and 1986 (Table 3). Clusters of four or more isolates of the same pathotype also occurred in northern Kings County, pathotype 15 (cluster of seven) from 1984 and 1986; southern Monterey County, pathotype 3-10-15-19-21 (cluster of five) from 1985 and 1986; and northeastern San Luis Obispo County, pathotype 3-10-15 (cluster of four) all from 1985. In contrast, mixtures of different pathotypes within a locale were also observed, especially in sections of Solano, Monterey, San Luis Obispo, and Tulare counties, where a larger number of samples was taken. Additionally, different pathotypes were found within individual fields. In two of seven fields where more than one leaf sample was evaluated, two distinct pathotypes were found: in 1984, pathotypes 10 and 15 were found in a field in Kings County and in 1985, pathotypes 3-10-15 and 3-10-15-19-21 were found in a field in San Luis Obispo County. The markedly different pathotypes, 11-22 and 6-13-16-18, were collected from northeastern Fresno County in 1984 and in east-central Tulare County in 1986, respectively.

The isolate from Minnesota (MN1A) was virulent on Tifang, Manchurian, Harbin, Manchuria, and CI 4922; this isolate was designated pathotype 1-6-13-16-18 (Table 4). Three different pathotypes were identified from Mexico: isolates MexLagA and MexLagB were not virulent on any of the differentials (pathotype 0); isolate MexBatA was virulent on Kombar (pathotype 10); and isolate MexStC was virulent on Kombar and Prato (pathotype 10-15). Isolate UK80-12A from England was virulent only on Rika and was thus designated pathotype 22.

DISCUSSION

Marked differences in virulence were detected among isolates of *P. t.* f. *teres* as 16 pathotypes were identified on 22 barley differentials from all collections tested in this study. With respect to California, 14.3% (13/91) of the isolates were distinct pathotypes. This degree of variation is not surprising because the sexual stage of the net blotch fungus has been observed in the field in California (8). Mature ascospores, however, are relatively rare in pseudothecia growing on barley straw. Conidia arising from oversummering mycelium in barley stubble are

considered the most important infective propagule in the epidemiology of the net blotch disease in California (unpublished data).

It is difficult to compare the results of this study with those of other workers because different host differentials, methods, and assessment protocols were used; however, variation in virulence in the net blotch pathogen is common. Bjarko (3) found five pathotypes of *P. t.* f. *teres* in Montana, and Singh (41) found 10 from a limited sample of North American isolates. In Canada, two pathotypes were identified in a 1974 survey (47) and 45 in a subsequent 1985 survey (46). Khan and Boyd (22) found three pathotypes in Australia using Algerian and either CI 7584, CI 2235, or CI 9776 as differential host groups. El-Fahl et al (10) found 21 pathotypes in Egypt, but this number was reduced to four when the differentials of Khan and Boyd were employed. Bjarko (3) reported seven pathotypes from a collection of 15 isolates from the Mideast and North Africa. Using cluster analysis, Harrabi and Kamel (17) classified 33 isolates from North Africa, Egypt, and Cyprus into four virulence groups. A high degree of variation in virulence was reported from the USSR, where 80 pathotypes of *P. t.* f. *teres* were identified on seven differentials (2). In contrast, Frecha (11) and Roth and Schafer (37) did not differentiate pathotypes of the net blotch pathogen even though their isolates exhibited some pathogenic variation on the host. If only the differentials used by Khan and Boyd are considered, one pathotype would be described from California in this study and would conform to pathotype W.A.-1. The California pathotypes apparently differ from those found in Australia (22), the Mideast and North Africa (3,10), Montana (3), Poland (13), and the USSR (2) based on the infection responses of common differentials in the respective studies.

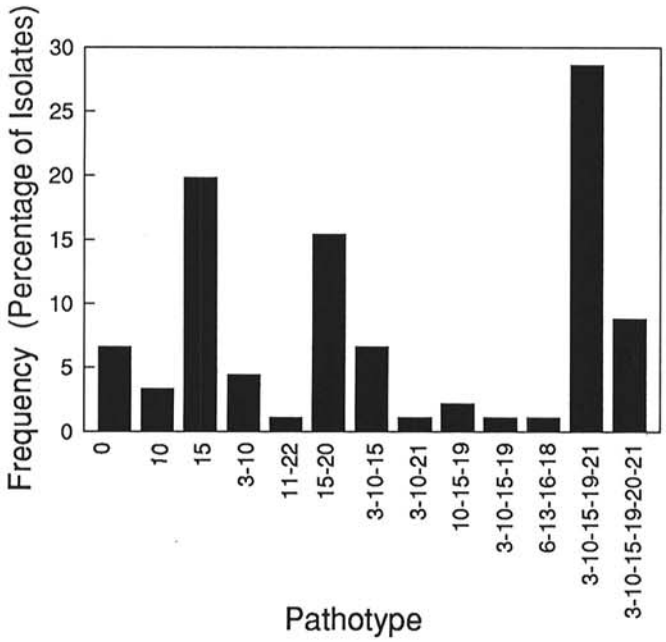

Fig. 1. Frequency (in percent) of pathotypes identified from 91 isolates of *Pyrenophora teres* f. *teres* collected in California. Designations for pathotypes are described in Table 2.

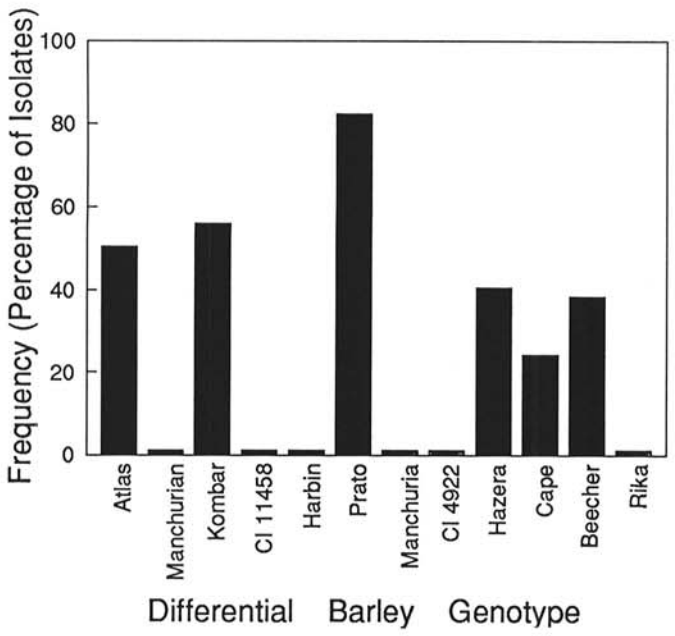

Fig. 2. Frequency (in percent) of pathotypes of *Pyrenophora teres* f. *teres* with virulence (conferring a high infection response) on individual barley genotypes. A total of 91 isolates from California were evaluated for their virulence phenotype on the host differentials. The barley genotypes Tifang, Canadian Lake Shore, Rojo, Coast, Ming, CI 9819, Algerian, CI 5791, CI 7584, and CI 5822 were resistant (exhibited low infection responses) to all 91 isolates.

Three of the four Mexican isolates studied (MexLagA, MexLagB, and MexBatA) conformed to the same virulence phenotype as pathotypes identified from California (Tables 2 and 4). The fourth isolate, MexStC, was virulent on Kombar and Prato, the differentials attacked by the greatest number of California pathotypes, but the virulence combination of this isolate was different from those found in California. Thus, the Mexican isolates were similar to the prevalent California isolates in virulence on specific barley differentials. The California pathotypes, 6-13-16-18 and 11-22, differed in virulence on only one differential host from isolates found in Minnesota, 1-6-13-16-18, and England, 22, respectively. From this limited sample, it appears that isolates of the net blotch pathogen from Minnesota, Mexico, and England are distinctly different because no common differential genotype was susceptible to the isolates from these respective regions.

Knowledge about the variation in virulence of pathogen populations is important for programs concerned with breeding for disease resistance. With such data, plant pathologists and breeders can wisely deploy sources of resistance that are likely to be effective against the spectrum of pathotypes in a given area. Resistance to California pathotypes of *P. t.* f. *teres* was common in the differential host set: six genotypes were resistant to 12 of 13 pathotypes and 10 were resistant to all pathotypes. Every differential used in this study was resistant to six or more pathotypes; thus, each differential host possesses genes for resistance to some pathotypes of *P. t.* f. *teres*. These results indicate that there are a number of effective sources of resistance to all pathotypes of *P. t.* f. *teres* found in California. The resistance of these genotypes is also effective in the adult plant stage under field conditions (43).

Khan (21) in Australia and Afanasenko (1) and Petrova (33) in the USSR reported that the population structure of the net blotch pathogen depends on the composition of cultivars grown in a region. From the present study, it is difficult to infer the possible influence that predominant cultivars may have on the structure of the pathotype population because only two (Prato and Atlas) of the 10 most common cultivars were evaluated to all the isolates, and the genetics of resistance to specific pathotypes of *P. t.* f. *teres* in many of the barley cultivars is not known. The isolates of *P. t.* f. *teres* evaluated in this study were collected from unidentified barley cultivars; thus, the possible relationship between specific pathotypes and the host genotype of origin cannot be determined.

Levitin and Afanasenko (27) found distinct differences in the composition of pathotypes within certain regions and indicated that this information should be used in breeding for resistance.

TABLE 3. Distribution of pathotypes of *Pyrenophora teres* f. *teres* by region, county, and year in California from 91 isolates

Region	County	Pathotypes by year[a]		
		1984	1985	1986
Northern Valley	Butte	...	...	15-20(1)
	Sutter	...	...	3-10-15-19-21(1)
Middle Valley	Solano	15(2) 3-10-15(1) 10-15-19(1) 3-10-15-19-21(4) 3-10-15-19-20-21(1)	10(1) 10-15-19(1) 3-10-15-19-21(4)	10(1) 3-10-15-19-21(5) 3-10-15-19-20-21(4)
	Stanislaus	...	...	15(3)
Southern Valley	Fresno	11-22(1) 3-10-15-19-21(1)	...	0(1) 15(1) 3-10-15-19-21(2)
	Kern	...	0(1)	0(1)
	Kings	10(1) 15(3) 15-20(2)	3-10-15-19-21(1)	15(4)
	Merced	15-20(2)	15-20(1)	...
	Tulare	15-20(1) 3-10-15-19-21(1)	15-20(1)	0(2) 6-13-16-18(1)
Coastal Valley	Monterey	15-20(2)	15-20(1) 3-10-15-19-21(2) 3-10-15-19-20-21(1)	0(1) 15(3) 3-10(2) 15-20(1) 3-10-15(1) 3-10-15-19-21(3)
	San Benito	15(1) 15-20(1)	...	...
	San Luis Obispo	...	3-10-15(4) 3-10-15-19(1) 3-10-15-19-21(2) 3-10-15-19-20-21(2)	15(1) 3-10(2) 15-20(1) 3-10-21(1)

[a] Pathotype designations are as given in Table 2. The number enclosed in parentheses denotes the number of isolates of that pathotype found in each county.

In this study, only tentative conclusions can be made about the distribution of pathotypes in California because the number of samples from some regions was small. Clusters of the same pathotype were found in certain locales, but mixtures of different pathotypes were also observed, sometimes within a single field. Thus, these data fail to confirm any distinct geographical differences in the distribution of pathotypes within California. When barley is bred for resistance to the net blotch pathogen in California, a wide spectrum of pathotypes should be considered.

Interesting differences were found between the Armstrong population and the off-station population: the most complex pathotypes, 3-10-15-19-21 and 3-10-15-19-20-21, were found in a much higher percentage in the former than in the latter population, and the latter population was genetically more diverse, as indicated by the diversity indices, than the former population. It is possible that some selection for more complex pathotypes has occurred at the field station where the barley breeding materials are routinely planted for disease evaluation; however, this result could also have occurred by chance. The greater diversity of pathotypes in the off-station population is not surprising considering the number of different cultivars grown in the state (up to 12, with most occupying no more than 7% of the total number of hectares planted) and the diverse regions that were sampled. The two populations differed significantly with respect to the Shannon index, but this measure of diversity can be sensitive to sample size (15). This factor is important to consider because the off-station population had over twice as many samples as the Armstrong population.

We are not aware of any attempt to gain acceptance for a set of standard differentials to type the virulence phenotypes of *P. t.* f. *teres*. Without a standard set, it is difficult to interpret and compare studies dealing with virulence in the pathogen and resistance in the host. The genetics of resistance in each host genotype (including references) and a list of previous studies employing the genotypes to differentiate isolates of *P. t.* f. *teres* are summarized in Table 1. There are a number of conflicts in the literature regarding the identity and number of genes in some of the differential genotypes, therefore no attempt was made to assign gene designations. Some of the disputes on the genetics of resistance to *P. t.* f. *teres* arose because workers have used different susceptible parents in crosses, different cultures of the pathogen, or different environmental conditions when evaluating the reactions of crosses. The barley genotypes selected for this study were thought to possess the greatest potential for differentiating isolates of *P. t.* f. *teres*, even though some have common genes for resistance (24). More research is needed on the genetics of resistance in these differential genotypes and on the development of near-isogenic lines for the resistance genes.

In addition to using the same set of differentials, it is also important for workers to standardize their inoculation, incubation, and disease assessment protocols. The methods used in this study proved to be quite reliable as differences of only 1–2 on the infection response scale were found when the isolates were retested. These differences were in the range of the LIR or HIR group—only rarely did the infection response vary across the two classes. Thus, the isolates used in this study did not exhibit the extreme variability for virulence as was reported in Israel by Kenneth et al (20).

In this study, the presence of extensive chlorotic zones surrounding large expanding lesions was an easily identifiable phenotype for separating HIRs from LIRs and was manifested most clearly 12–14 days after inoculation. It is certain that further differentiation of isolates could also be made on the basis of infection responses within the LIR or HIR groups (e.g., infection

TABLE 4. Mode and range of infection responses exhibited on 22 barley genotypes to five pathotypes of *Pyrenophora teres* f. *teres* differentiated from isolates collected in Mexico, England, and Minnesota

	Pathotypes[a]				
	0	10	22	10-15	1-6-13-16-18
Genotypes	Mode/Range	Mode/Range	Mode/Range	Mode/Range	Mode/Range
1. Tifang	1,4/1-4[b]	4,3/3-4	1/1	3,2/3-2	8-9/7-9(10)
2. Canadian Lake Shore	1,4/1-4	4,3/3-4	1/1-2	5,4/4-5	5,4/4-5
3. Atlas	2,5/2-5	5,4/4-5	1,2/1-2	4,5/4-5	3,2/2-5
4. Rojo	1,2/1-2	2,1/1-2	1,2/1-2	2,1/1-2	2,1/1-3
5. Coast	2,1/1-2	2,1/1-2	2,1/1-2	2,1/1-2	2,1/1-3
6. Manchurian	1,4/1-4	4,5/4-5	1,3/1-3(4)	3,4/3-4	9,10/9-10
7. Ming	1,2/1-3	5,4/4-5	1/1-2	4,3/3-4	3/3-5
8. CI 9819	1,3/1-3	2,1/1-2	1,2/1-2	2,1/1-2	1,2/1-3
9. Algerian	1,2/1-3	2,1/1-2	2/1-3	3,2/2-3	2,1/1-3
10. Kombar	2,5/2-5	9,10/9-10	2,3/1-3(4)	9,10/9-10	5,4/3-5
11. CI 11458	1,2/1-3	4,3/3-4	2/1-3	2,3/2-3	2/2-5
12. CI 5791	1,2/1-2	2,1/1-2	2/1-3	2,1/1-2	2,1/1-3
13. Harbin	2,4/2-4	5,4/4-5	2/1-3	5,4/4-5	9,10/9-10
14. CI 7584	2/1-3	2,1/1-2	2/1-3	2,1/1-2	2,1/1-3
15. Prato	3,4/2-4	4,5/4-5	2/1-3	9,10/9-10	2,1/1-3
16. Manchuria	3,4/3-5	3,4/3-4	2/1-3	4,3/3-4	9,10/8-10
17. CI 5822	2/1-3	2,1/1-2	2/1-3	2,1/1-2	3,2/2-4
18. CI 4922	1,4/1-4	4,3/3-4	2,1/1-2	4,5/4-5	8,9/7-9
19. Hazera	2,3/2-5	4,5/4-5	2/1-3	5,4/4-5	3,4/2-5
20. Cape	2,4/2-5	2,3/2-3	2,4/2-5	2,1/1-2	5,4/4-5(6)
21. Beecher	1,2/1-2	4/4	2/1-3	4,3/3-4	3,2/2-4
22. Rika	2/1-3	2,1/1-2	9,10/8-10	3,2/2-3	3,2/2-3

[a] See Table 2. Pathotype 0 was composed of isolates MexLagA and MexLagB from Mexico; pathotype 10, isolate MexBatA from Mexico; pathotype 22, isolate UK80-12A from England; pathotype 10-15, isolate MexStC from Mexico; and pathotype 1-6-13-16-18, isolate MN1A from Minnesota.
[b] See Table 2.

response 1,2 vs. 4,5), but the present classification system has practical implications in breeding for net blotch resistance. In a number of barley genotypes, the LIR exhibited in the seedling stage is correlated with adult plant resistance in the field (43).

The nomenclatural system used for pathotypes in this study was selected for convenience and is based on the systems of Black et al (4) and Watson and Luig (49). Isolates were virulent on only a few host genotypes, and thus a relatively short code could be used to describe the virulence phenotype with the aid of a differential host list. This system would admittedly be cumbersome if a large number of differential genotypes prove susceptible. A similar system is used with *P. t.* f. *teres* in the United Kingdom cereal pathogen virulence survey (B. C. Clifford, personal communication). In another study, Afanasenko (2) used the nomenclatural system of Habgood (16) to designate pathotypes of *P. t.* f. *teres*, but this scheme is too complicated for workers who only occasionally use the system.

Investigations on the specificity of pathogens from surveys are important for monitoring the frequency of potentially threatening pathotypes, characterizing pathotypes useful in studies on the genetics of host/parasite interactions, studying the distribution of pathotypes in a region, identifying virulence markers useful in population biology studies, and evaluating the stability of pathotypes over time (36). The set of differentials employed in this investigation has been effective for typing the virulence phenotypes of *P. t.* f. *teres* isolates from diverse regions and should be useful to other researchers studying this system. The adoption of a standard set of differentials and protocols for studying the virulence spectrum of *P. t.* f. *teres* would greatly facilitate the interpretation of results among international workers in this host/parasite system.

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