Resistance in Winter Wheats to Geographically Differing Isolates of *Pyrenophora tritici-repentis* and Observations on Pseudoperithecia

M. DIAZ de ACKERMANN, Plant Pathologist, Estacion Experimental, La Estanzuela, Colonia, Uruguay, and R. M. HOSFORD, JR., Professor, Department of Plant Pathology, D. J. COX, Assistant Professor, and J. J. HAMMOND, Professor, Department of Agronomy, North Dakota State University, Fargo 58105

ABSTRACT

Diaz de Ackermann, M., Hosford, R. M., Jr., Cox, D. J., and Hammond, J. J. 1988. Resistance in winter wheats to geographically differing isolates of *Pyrenophora tritici-repentis* and observations on pseudoperithecia. Plant Disease 72:1028-1031.

The winter wheats (*Triticum aestivum*) Carifen 12 and Red Chief were resistant, Roughrider was moderately resistant, and Brule and ND8001 were moderately susceptible, and the hard red spring wheat ND495 was susceptible, to nine virulent isolates (causing large tan spots on many genotypes) of the fungus *Pyrenophora tritici-repentis* from several areas of the Great Plains of North America. Lesion length separated these wheats for resistance. Isolates PTL1, PYD7, PYR72, Pti2, 1231CDA, 78-64, W38, 78-62, and Embden were highly virulent on the susceptible genotype, moderately to highly virulent on the moderately susceptible genotypes, and of low virulence on the moderately resistant to resistant genotypes. Isolates PT4, PTF3, and Buffalo were of low virulence (causing small tan spots) or avirulent (causing tiny dark flecks) on all genotypes. Isolate × genotype specificity was not evident. Most fungal isolates retained their vigor and virulence after 4 yr in liquid nitrogen. On corn leaves, the fungal pseudoperithecia developed unusually long necks.

Additional keywords: *Drechslera tritici-repentis, Helminthosporium tritici-repentis, Pyrenophora trichostoma*, tan spot, yellow leaf spot

Tan spot of wheat (*Triticum aestivum* L.) and other gramineous plants is caused by the fungus *Pyrenophora tritici-repentis* (Died.) Drechs., anamorph *Drechslera tritici-repentis* (Died.) Shoem. Synonyms of tan spot are yellow leaf spot, yellow leaf blotch, wheat leaf blight, and eyespot (6). In recent years, the prevalence and severity of tan spot have increased to damaging levels, and this disease has been detected around the world in low- to high-rainfall areas under a wide range of temperatures. This increase may be due to changes in cultivars, weather, cultural practices (such as minimum tillage), the fungus, and/or increased recognition of the disease (4,6,16,18–21). Grain yield losses of 2–49% (6,20,21) have been attributed to tan spot. Differences in pathogenicity among isolates of *P. tritici-repentis* (4–6,9,10,15) and differences in severity of spotting among wheat genotypes (2,5–8,10–13,15,18,20) have been reported. Forty isolates from the Great Plains of North America were separated into 12 races for virulence (severity of tan spot) on six spring wheat and durum (*T. turigidum* L. var. *durum*) differentials, and specificity in the fungal strain-wheat genotype relationship was detected (15). On wheat, isolates of *P. tritici-repentis* from bromegrass (*Bromus inermis* Leyss.) had virulence levels comparable to isolates from wheat, but support for specificity was not found (10). Resistance among wheat genotypes is polygenic (1,6), except for a single recessive gene in Carifen 12 (12).

In this host-parasite relationship, percent infection (spots per inoculum concentration) and lesion size increase with rising temperature and increasing duration of free moisture on foliage following inoculation (5–7,11,13). One mechanism of resistance stops infection hyphae and involves papillae formation in some initially invaded epidermal cells (11). A more effective form of resistance, which is diminished and finally eliminated by lengthening periods of postinoculation leaf wetness, retards both mycelial growth and increases in lesion size (7,8,10,11,13). Lesion size on specific genotypes has been related to cell-free culture filtrate, suggesting that host resistance is related to reduced sensitivity to a toxin (22). The rate of lesion development has been reduced by increasing the rate of nitrogen fertilizer and increasing the proportion of nitrogen taken up as ammonium (8). Reduced infection frequency has also been found to be a component of resistance in some (10,13) but not all wheat genotypes (7,8,11,13). Significant differences in lesion lengths among wheat genotypes have been reported (2,7,8,10,11,13) and related among seedlings and adult plants in the greenhouse to severity of tan spot on adult plants in the field (2). Differences have been detected among single ascospore isolates for lesion length, percent infection, and fungal growth, color, and sporulation (3,9). Also, *Cochliobolus sativus* (spot blotch) and *P. tritici-repentis* (tan spot) antagonistically reduce each other's spotting (14).

Our objective was to examine the reaction of several winter wheat genotypes to isolates of *P. tritici-repentis* from several areas of the Great Plains of North America and to examine lesion length as a basis for separating these wheats for resistance. During the study, pseudoperithecia grown on corn leaves developed unusually long necks or beaks.

MATERIALS AND METHODS

Wheat genotypes and fungal isolates. The wheat genotypes used in this study were the winter wheats Carifin 12, Red Chief (CI 12109), Roughrider (CI 17439), Brule (PI 466739), and ND8001 and the spring wheat ND495. Previous studies (2,12,15,18) indicated that these genotypes are resistant, resistant, moderately resistant, susceptible, susceptible, and very susceptible, respectively, to tan spot.

In a prior study (15), the descending order of virulence on ND495 of the fungal isolates used from liquid nitrogen were Pti2 of race 12 from South Dakota, 78-62 of race 12 from Montana, PYR72 of race 12 from Nebraska, PYD7 of race 11 from North Dakota, PTF3 of race 11 from North Dakota, 1231CDA of race 10 from Saskatchewan, PT4 of race 8 from Nebraska, PTL1 of race 7 from North Dakota, W38 of race 5 from North Dakota, and 78-64 of race 5 from Montana. All these isolates had been stored in liquid nitrogen for more than 4 yr. Two fresh isolates from the field, the single-conidium isolates Embden (virulent on ND495) and Buffalo (avirulent on ND495) from tan spots on wheat in North Dakota, were included in one trial. Six isolates were present in all trials, but others were present in fewer trials.

Inoculum. The fungus was grown on potato-dextrose agar (PDA) in petri

Portion of a thesis submitted by the first author in partial fulfillment of the requirements for the M.S. degree at North Dakota State University.

Journal Series No. 1689 of the North Dakota Agricultural Experiment Station.

Accepted for publication 30 June 1988 (submitted for electronic processing).

plates at 22–24 C in continuous light from one each General Electric F40/BLB and Sylvania F40/CW fluorescent tubes 30 cm above the plates, for 10 days. Disks (1 cm in diameter) of the fungus and PDA were transferred, from the gray mycelium halfway between the center and the edge of colonies that had grown to within 1 cm of the edge of the petri plates, to the center of petri plates of modified V-8 agar (150 ml of V-8 juice, 1.5 g of $CaCO_3$, and 20 g of agar in 850 ml of distilled water). The V-8 agar plates were sealed with Parafilm and incubated in a Percival 13LLVL temperature control chamber (Percival Mfg. Co., Boone, IA) at 21 ± 1 C under a 14-hr photoperiod (one each General Electric F20/BLB amd Sylvania F20 GRO-LUX fluorescent tubes 20 cm above the plates) for 10 days. Inoculum was obtained by scraping the growth of the fungus from the agar with a loop and blending in distilled water for 5 min, generally following the technique of Raymond et al (2,7,18). The numbers of conidia, conidiophores, and mycelial fragments in the inoculum were determined with a hemacytometer and their concentration adjusted to 50,000/ml; all three fungal states are infective, but relative infection frequency has been only empirically determined for conidia vs. mycelium (5,6). Four drops of Tween 20 were added to each 100 ml of suspension, and 2.3 ml of inoculum was applied per plant with an atomizer (Model 26, DeVilbiss Co., Somerset, PA). Plants were inoculated at the five-leaf stage of seedling development. This inoculum level is high when compared with some past studies (5,7,15) but not when compared with others (4). We did not detect clumping of inoculum or coalescence of spots on the leaves.

Wheat culture. Winter wheats were grown in flats filled with Sunshine mix (Fisons Western Corporation, Vancouver, BC, Canada; Complete Blend No. 1: peat moss, dolomite limestone, perlite [pH adjustor], vermiculite, wetting agent) to the two- to three-leaf stage in the greenhouse at 21 ± 5 C. These flats were placed at 5 C in 12 hr light/12 hr dark cycles for 6 wk. After vernalization, seedlings were transplanted into 15-cm clay pots of Sunshine mix (three seedlings per pot) and grown in the greenhouse to the fifth-leaf stage of seedling development. Spring wheat was planted in clay pots 10 days before the winter wheats were transplanted, grown in the greenhouse to the fifth-leaf stage, and thinned to three plants per pot.

Experimental design. Three pots (replications) of each wheat genotype containing three plants per pot were randomized and sprayed with an isolate. This group of pots was placed in a sunlit, plastic-covered mist chamber, and the process was repeated with each isolate or water treatment in a trial. Genotypes were randomized and replicated within each of four trials; isolates, however, were replicated only over trials and only six isolates were used in all trials. Therefore, an analysis of variance was computed over trials using a split-plot design with isolate as the main plot effect and genotypes as the subplot effect. The water mist was on for 1-min periods at 10-min intervals over 30 hr at 21 ± 3 C for each trial. Potted plants were fan-dried and placed on a greenhouse bench at 21 ± 5 C. Lesion length in millimeters of the largest lesion on leaf 4 and leaf 5 was measured 7 days after inoculation. Nine plants of each genotype inoculated with a given isolate were rated in each trial.

Observations of pseudoperithecia. Mature pseudoperithecia were produced using two different techniques: 1) Isolates Pti2, PYD7, and HP28 were grown singly and in all possible combinations on autoclaved wheat seed on water agar in petri plates at 22 ± 1 C for 7 days. The plates were placed at 10 ± 1 C for 2 to 3 mo (5). 2) Pti2, PYD7, and PYR72 were grown on autoclaved pieces of corn

Table 1. Length (mm) of lesions caused by isolates of *Pyrenophora tritici-repentis* on six wheat genotypes[a]

Trials	Isolates	Wheat genotypes						LSD (0.05)	Av.
		ND495	ND8001	Brule	Roughrider	Red Chief	Carifen 12		
				Leaf 5					
1–4	PTL1	4.8	3.1	2.6	1.0	0.8	0.6	2.6	2.2
1–4	PYD7	3.9	2.6	1.6	1.1	0.8	0.8	2.1	1.8
1–4	PYR72	3.9	2.5	3.0	1.9	0.8	1.3	2.6	2.2
1–4	Pti2	3.6	3.2	2.8	1.4	0.9	0.6	2.6	2.1
1–4	1231CDA	3.5	1.6	1.2	0.7	0.4	0.3	2.6	1.3
1–4	78-64	3.0	1.9	2.4	0.8	0.8	0.9	2.6	1.6
Average of above six isolates		3.8	2.5	2.3	1.1	0.7	0.7		
P/Diff[b] (0.05)		A	B	B	C	C	C		
2–4	W38	3.9	1.3	1.6	0.8	0.4	1.2	3.0	1.5
Average of above seven isolates		3.9	2.3	2.3	1.1	0.8	0.9		
P/Diff (0.05)		A	B	B	C	C	C		
1–3	78-62	2.3	1.8	2.0	0.4	0.3	0.5	3.0	1.2
Average of above seven isolates, excluding W38		3.1	1.9	1.8	0.9	0.4	0.4		
P/Diff (0.05)		A	B	B	C	C	C		
1 & 2	PT4	0.4	0.0	0.0	0.1	0.0	0.0	3.7	0.1
1	Embden	4.0	2.8	2.2	1.4	0.6	0.5	5.2	1.9
1	PTF3	0.0	0.0	0.0	0.2	0.3	0.2	5.2	0.1
3	Buffalo	0.0	0.4	0.8	0.2	0.0	0.0	5.2	0.2
				Leaf 4					
1–4	PTL1	6.3	4.6	5.9	2.9	1.7	1.3	3.5	3.8
1–4	PYD7	5.1	4.1	3.7	2.2	1.2	1.6	2.8	3.0
1–4	PYR72	5.8	4.4	5.0	3.8	1.5	1.8	3.5	3.7
1–4	Pti2	6.7	4.3	4.5	2.7	1.6	1.6	3.5	3.5
1–4	1231CDA	4.5	2.3	2.9	2.0	1.1	1.1	3.5	2.3
1–4	78-64	5.4	4.6	6.0	2.4	1.5	1.3	3.5	3.5
Average of above six isolates		5.6	4.1	4.7	2.7	1.4	1.5		
P/Diff (0.05)		A	B	B	C	D	D		
2–4	W38	4.9	2.4	3.8	1.7	1.1	3.1	4.0	2.8
Average of above seven isolates		5.8	4.1	4.9	2.7	1.6	2.0		
P/Diff (0.05)		A	B	B	C	C	C		
1–3	78-62	3.3	3.9	4.6	2.0	0.9	0.9	4.0	2.6
Average of above seven isolates, excluding W38		4.4	3.1	3.6	2.1	0.9	0.8		
P/Diff (0.05)		A	B	B	C	D	D		
1 & 2	PT4	0.4	0.1	0.1	0.1	0.0	0.0	4.9	0.1
1	Embden	5.7	4.7	3.6	2.1	0.4	0.8	7.0	2.9
1	PTF3	0.3	0.0	0.0	0.0	0.3	0.0	7.0	0.1
3	Buffalo	1.9	0.6	1.9	0.6	0.2	0.7	7.0	1.0

[a] Six isolates were used in all four trials, others in only some trials. Plants were inoculated at the fifth leaf stage of seedling development, misted for 30 hr at 21 ± 3 C, placed on a greenhouse bench at 21 ± 5 C, and rated 7 days after inoculation. Water-inoculated checks of all genotypes in all trials were not spotted. Lesion lengths (mm) were the least significant means of the longest (largest) lesion on leaf 5 (top leaf) and leaf 4 (leaf below).

[b] Probability of difference; means with the same letter are not significantly different at the 0.05 level.

leaves on water agar at 21 ± 1 C under 14 hr of light/10 hr of darkness in the Percival chamber for 30–35 days (4,16).

RESULTS AND DISCUSSION

Wheat genotypes and fungal isolates. Carifin 12 and Red Chief showed a high degree of resistance (low lesion length, corresponding to small lesion size) to virulent isolates (Table 1). In trials 2–4, however, Carifin 12 had a high mean lesion length with isolate W38 on leaf 4 (3.1 mm). Roughrider was less resistant to virulent isolates, with slightly but not significantly greater mean lesion length on leaf 5 and leaf 4, except for significantly greater length on leaf 4 when isolate 78-62 was included in the analysis. Brule and ND8001 were moderately susceptible, with significantly greater mean lesion length on both leaf 5 and leaf 4 than occurred on Roughrider. ND495 was susceptible to all virulent isolates, with significantly greater mean lesion length than mean lengths on the winter wheats tested; it was not susceptible to isolates PT4, PTF3, and Buffalo (Table 1). This higher resistance of Red Chief and Carifen 12 is in agreement with the findings of earlier researchers (2,10,12,18), as is the high susceptibility of ND495 (2,4,15). Thus, our findings extend these detected resistances to a wider range of isolates from several areas of North America.

While ranking for resistance by lesion length appears to be a good measure of resistance in wheat (2,10), and perhaps of pathogenicity among isolates (9,10) and of nitrogen level and form supplied to susceptible genotypes (8), this parameter does not take into account differences in lesion numbers previously reported on some (10,13) but not other (11) genotypes. The significance of lesion number should be further investigated.

The average lesion length for the six isolates in all four trials (Table 1) increased in the order of decreasing resistance described above on both leaf 5 (0.7, 0.7, 1.1, 2.3, 2.5, and 3.8, respectively) and leaf 4 (1.5, 1.4, 2.7, 4.7, 4.1, and 5.6, respectively), with the lesions on leaf 4 always longer than those on leaf 5. Also, the average lesion length of each isolate on leaf 5 of all six wheat genotypes (2.2, 1.8, 2.2, 2.1, 1.3, and 1.6, respectively) was always shorter than that on leaf 4 (3.8, 3.0, 3.7, 3.5, 2.3, and 3.5, respectively). These results using several isolates support earlier reports (2,18) that lesions are progressively larger on lower leaves. However, as the postinoculation foliage wet period is increased from 6 to 54 hr (30 hr was used in this study), differential resistance of genotypes is progressively lost and lesions become large on all leaves (6). Lesion length did not significantly differ among isolates, which was different from an earlier report (9) and possibly resulted from the low precision placed on isolates in the design of the current study.

The isolate × genotype interaction was not significant; the more virulent isolates did not distinctly differ in virulence on a given wheat genotype (Table 1). The virulent isolates PTL1, PYD7, PYR72, Pti2, 1231CDA, 78-64, W38, and 78-62 from liquid nitrogen and Embden were generally of low virulence on Carifen 12, Red Chief, and Roughrider; moderately to highly virulent on Brule and ND8001; and highly virulent on ND495. The only exception was W38 on leaf 4 of Carifin 12, resulting in a mean lesion length of 3.1, which was not significant. In the one or two trials in which they were tested, PT4, PTF3, and Buffalo were of low virulence or avirulent on all genotypes (Table 1). Using a different set of wheat differentials that included ND495, Luz and Hosford (15) found highly significant differences in virulence among 40 isolates, including most of the isolates used in this study. In this current study, however, isolates PT4 and PTF3 from liquid nitrogen were much less virulent on ND495 than in the earlier study (15). Although these isolates might have lost virulence during storage in liquid nitrogen, we suspect they became avirulent during transfers, before being stored in liquid nitrogen.

Lesions were considerably longer in trial 4 than in trials 1, 2, and 3 (Table 2). This large difference in lesion length among trials may have been caused by uncontrolled factors. Effects of post-inoculation foliage wet period (5,7,11) and temperature (7,11,13) on infection by *P. tritici-repentis* have been studied, but the effects of other factors, such as vigor of the inoculum, merit further research. These might also influence lesion numbers.

In a single additional trial using an older disease severity rating system (6), the isolate × genotype interaction was significant (results not shown), but the relative magnitude of the mean square indicated that this variation was much smaller than the variation among genotypes (3). Because of these differing results, specificity of individual isolates or races (15) could not be supported, and the results of the larger number of trials (four) supported the theory that virulence is multigenic and expressed as aggressiveness (10). In the additional trial, the genotypes had a ranking for severity of spotting similar to that shown in Table 1 (3).

Pseudoperithecia. When isolates Pti2, PYD7, and HP28 were grown singly and in all possible combinations on wheat seed (16 times) and Pti2, PYD7, and PYR72 on corn leaves (37 times), pseudoperithecia containing mature asci were produced on wheat seed by Pti2 and Pti2 × PYD7 after 150 and 210 days, respectively. The pseudoperithecia were located on the seed where they contacted the agar. On corn leaves, all isolates and combinations except PYR72 reproduced sexually after 30 days.

On corn leaves, pseudoperithecia had unexpectedly long necks (Fig. 1) measuring 360–600 (av. 456) μm wide × 960–1,200 (av. 1,068) μm long from the bottom of the pseudoperithecium to the tip of the beak. The beak was 480–720 (av. 600) μm long (3). Mehta (16) obtained pseudoperithecia with a flat base, prominent ostiole, and small beak on autoclaved corn leaves. When mature, the pseudoperithecia measured 445–756 × 445-676 μm. Gilchrist (4) obtained large (800–1,280 μm) pseudoperithecia on sterilized corn leaves. However, neither she nor Wehmeyer (23), who studied the development of the pseudoperithecium in 1954, mentioned the presence of unusually long necks. Recently, mature pseudoperithecia were produced on cellulose-based artificial medium and the influence of N, P, and K levels in the medium were determined; beaks were not mentioned (17). Gilchrist (4) reported that ascospores were not liberated through ostioles of mature pseudoperithecia in Mexico. She suggested that pseudoperithecia produced asexual

Table 2. Least significant means (mm) of lesion length of *Pyrenophora tritici-repentis* for trials 1–4[a]

Leaf	Trial 1	2	3	4	LSD (0.05)
5	1.6	1.1	1.5	3.1	1.2
4	2.4	1.6	2.7	5.8	1.6

[a] Isolates PTL1, PYD7, PYR72, Pti2, 1231CDA, and 78-64 and wheat genotypes ND495, ND8001, Brule, Roughrider, Red Chief, and Carifen 12.

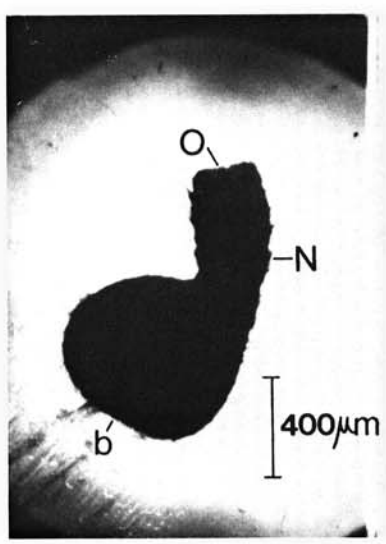

Fig. 1. *Pyrenophora tritici-repentis* pseudoperithecium formed with an unusually long neck when grown on autoclaved corn leaves on water agar. O = ostiole, b = pseudoperithecium base, N = neck or beak.

spores on the external surface that infected wheat in the field. However, ascospores have been detected in the air in Australia, the United States, and Canada (18,19). We have often observed pseudoperithecia on wheat straw in the field containing mature ascospores but having no beak or ostiole (Hosford, *personal observations*). Pseudoperithecial ostiole and beak formation and ascospore liberation appear to vary and warrant further study.

In summary, resistant wheat genotypes maintained their resistance when exposed to isolates of *P. tritici-repentis* from several parts of North America, and the parameter of lesion length specifically identified susceptible and resistant genotypes. Isolate × genotype specificity was not evident, and most isolates retained vigor and virulence when stored in liquid nitrogen for over 4 yr. Some, but not all, isolates produced ascospores, and pseudoperithecia developed unusually long necks on corn leaves.

ACKNOWLEDGMENTS

We thank J. M. Krupinsky and H. A. Lamey for manuscript review and Phyllis Hellem for repeated manuscript typing.

LITERATURE CITED

1. Cantrell, R. G., Elias, E., and Hosford, R. M., Jr. 1985. The inheritance of resistance to tan spot in durum wheat. Agron. Abstr. 77:50.
2. Cox, D. J., and Hosford, R. M., Jr. 1987. Resistant winter wheats compared at differing growth stages and leaf positions for tan spot severity. Plant Dis. 71:883-886.
3. Diaz de Ackermann, M. 1987. Resistance among winter wheats to *Pyrenophora tritici-repentis* isolates and variability in fungal growth, color, sporulation and pathogenicity. M.S. thesis. North Dakota State University, Fargo. 49 pp.
4. Gilchrist, S. Lucy, I. 1982. *Helminthosporium tritici-repentis* (= *Pyrenophora trichostoma*) como agente causal del tizon del trigo, prevalente en el Estado de Michoacan, Mexico. Ph.D. thesis. Colegio de Postgraduados, Chapingo, Mexico. 96 pp.
5. Hosford, R. M., Jr. 1971. A form of *Pyrenophora trichostoma* pathogenic to wheat and other grasses. Phytopathology 61:28-32.
6. Hosford, R. M., Jr., ed. 1982. Tan Spot and Related Diseases Workshop. N.D. Agric. Exp. Stn. 116 pp.
7. Hosford, R. M., Jr., Larez, C. R., and Hammond, J. J. 1987. Interaction of wet period and temperature on *Pyrenophora tritici-repentis* infection and development in wheats of differing resistance. Phytopathology 77:1021-1027.
8. Huber, D. M., Lee, T. S., Ross, M. A., and Abney, T. S. 1987. Amelioration of tan spot-infected wheat with nitrogen. Plant Dis. 71:49-50.
9. Hunger, R. M., and Brown, D. A. 1987. Colony color, growth, sporulation, fungicide sensitivity, and pathogenicity of *Pyrenophora tritici-repentis*. Plant Dis. 71:907-910.
10. Krupinsky, J. M. 1987. Pathogenicity on wheat of *Pyrenophora tritici-repentis* isolated from *Bromus inermis*. Phytopathology 77:760-765.
11. Larez, C. R., Hosford, R. M., Jr., and Freeman, T. P. 1986. Infection of wheat and oats by *Pyrenophora tritici-repentis* and initial characterization of resistance. Phytopathology 76:931-938.
12. Lee, T. S., and Gough, F. J. 1984. Inheritance of Septoria leaf blotch (*S. tritici*) and Pyrenophora tan spot (*P. tritici-repentis*) resistance in *Triticum aestivum* cv. Carifen 12. Plant Dis. 68:848-851.
13. Luz, W. C. da, and Bergstrom, G. C. 1986. Effect of temperature on tan spot development in spring wheat cultivars differing in resistance. Can. J. Plant Pathol. 8:451-454.
14. Luz, W. C. da, and Bergstrom, G. C. 1987. Intereactions between *Cochliobolus sativus* and *Pyrenophora tritici-repentis* on wheat leaves. Phytopathology 77:1355-1360.
15. Luz, W. C. da, and Hosford, R. M., Jr. 1980. Twelve *Pyrenophora trichostoma* races for virulence to wheat in the Central Plains of North America. Phytopathology 70:1193-1196.
16. Mehta, Y. R. 1978. Doencas do Trigo e seu Controle. Editora Agronomica Ceres Ltda. Summa Phytopathol. Brasil. 190 pp.
17. Pfender, W. F., and Wootke, S. L. 1987. Production of pseudothecia and ascospores by *Pyrenophora tritici-repentis* in response to macronutrient concentrations. Phytopathology 77:1213-1216.
18. Raymond, P. J., Bockus, W. W., and Norman, B. L. 1985. Tan spot of winter wheat: Procedures to determine host response. Phytopathology 75:686-690.
19. Rees, R. G., and Platz, G. J. 1980. The epidemiology of yellow spot of wheat in southern Queensland. Aust. J. Agric. Res. 31:259-267.
20. Rees, R. G., and Platz, G. J. 1983. Effects of yellow spot on wheat: Comparison of epidemic at different stages of crop development. Aust. J. Agric. Res. 34:39-46.
21. Tekauz, A., Platford, R. G., and English, N. C. 1982. Pages 60-65 in: Manit. Agron. Proc. Tech. Sci. Pap.
22. Tomas, A., and Bockus, W. W. 1987. Cultivar-specific toxicity of culture filtrates of *Pyrenophora tritici-repentis*. Phytopathology 77:1337-1340.
23. Wehmeyer, L. E. 1954. Perithecial development in *Pleospora trichostoma*. Bot. Gaz. 115:297-310.