Influence of Temperature and Five Fungicides on Rhizoctonia Root Rot of Hard Red Winter Wheat

J. T. MATHIESON, Research Associate, and C. M. RUSH, Professor, Texas Agricultural Experiment Station, Bushland 79012

ABSTRACT

Mathieson, J. T., and Rush, C. M. 1991. Influence of temperature and five fungicides on Rhizoctonia root rot of hard red winter wheat. Plant Dis. 75:983-986.

Rhizoctonia root rot is a disease associated with early-planted hard red winter wheat in the Texas Panhandle. Studies were conducted to determine the effect of temperature and fungicide seed treatment on disease development. The fungicides tested included triadimenol, imazalil, difenoconazole, UBI1886, and Nusan + Nuzone. When wheat plants were grown in soil infested with a known pathogenic isolate of *Rhizoctonia solani*, anastomosis group 4 (AG-4), at five constant temperatures ranging from 15 to 35 C, emergence decreased significantly ($P = 0.05$) as temperatures increased ($R^2 = 0.50$). Fungicides were tested for their ability to inhibit growth of *R. solani* in vitro on amended potato-dextrose agar and for disease control in infested soil at 15, 25, and 35 C. No fungicide greatly reduced mycelial growth in vitro at concentrations ≤ 0.1 μg a.i./ml, but at concentrations of 1 μg/ml or greater, all fungicides significantly reduced growth. Seed treated with triadimenol had significantly greater emergence and dry matter production at each temperature compared with other treatments. Seed treated with triadimenol had significantly less infection, both on the seed and coleoptile, for 25 days after planting in soil infested with *R. solani* compared with the untreated check.

Hard red winter wheat (*Triticum aestivum* L.) is a major crop in the southern Great Plains. Wheat is grown as a forage crop by cattlemen to increase beef production, which is one of the major industries in the Panhandle region of Texas (21). The Texas Panhandle region alone produces 80% of the beef in Texas, with an approximate annual value of $3.5 billion. To maximize forage production, wheat is planted in late August or early September. Generally, the optimum planting date for grain production in the Panhandle region is the first 2 wk of October depending on soil moisture. One problem often associated with early-planted wheat is seedling disease (12,19). Warm temperatures and high rainfall during this time of year result in soil conditions favorable for disease development, which can lower plant stands and reduce yields. Mean air temperatures for the months of August and September are 24.1 and 20.3 C and average rainfall is 7.0 and 4.6 cm, respectively.

Over the past 2 yr, a seedling disease of wheat caused by *Rhizoctonia solani* Kühn has been found with increasing frequency in the Texas Panhandle. Problems related to *R. solani* on wheat have been identified in Australia, Europe, and parts of the United States (7,10,11,13,16,17,20). Infected seedlings may be killed or severely stunted. Although no single method is available to eliminate the disease, certain practices may help reduce disease incidence.

Methods currently used to reduce the disease are, for the most part, cultural. Crop rotation combined with full season summer fallow may be useful where possible. Planting when temperatures are cool and planting shallow can also be beneficial (19). Root infection can be reduced by disturbing the soil before planting (6) or using conventional tillage practices instead of no-till (8–10).

Growers in the Panhandle often are able to gain higher returns from wheat as forage than grain production. Additionally, their options for controlling disease may be reduced because of a limited number of alternative crops. Chemical control of early seedling disease would allow producers to plant early, protect seedlings until cool soil temperatures slow the activity of pathogens, maximize wheat forage, and potentially reduce grain losses attributable to disease.

The objectives of this study were to 1) determine how temperature affects pathogenicity of *R. solani* and subsequent development of wheat seedling disease, 2) evaluate five fungicides for in vitro toxicity and efficacy in situ for controlling *R. solani*, and 3) determine how long triadimenol protects the seed and coleoptile from infection by *R. solani*.

MATERIALS AND METHODS

Inoculum production and seed treatments. An isolate of *R. solani* from Texas, known to be pathogenic to wheat and identified as anastomosis group 4 (AG-4), was used throughout the study. The anastomosis group of the isolate was confirmed by D. Carling (*personal communication*). Inoculum was produced on barley seed, and soil used in the study was amended with infested barley seed 1% (v/v). The hard red winter wheat cultivar TAM 107 was used in all studies. The following amounts of formulated product were mixed with 50 ml of water and applied to 10 kg of seed: 9.8 ml of Baytan 30 Flowable (triadimenol 30% a.i.); 11 ml of Dividend 3FS (difenoconazole 42.5% a.i.); 19 ml of UBI1886 (PCNB 23.5% a.i. + imazalil 3.1% a.i.); 1.6 ml of Flo Pro 1MZ (imazalil 10% a.i.); and 6 ml each of Nusan 30EC + Nuzone 10ME (TCMTB 30% a.i. + imazalil 10% a.i.). Seed was placed in a cement mixer and the drum rotated as the fungicide-water mixture was applied.

In vitro toxicity test. Chemicals used as seed treatments were first tested in vitro to determine limits of activity. A stock solution was prepared and the necessary amount of solution added to 100 ml of potato-dextrose agar (PDA) to obtain concentrations of 0.0, 0.01, 0.1, 1.0, and 10 μg a.i./ml. Five replicate plates were used for each chemical rate combination. A 10-mm plug from an actively growing culture of *R. solani* was placed in the center of each dish, and the dishes were incubated at room temperature. Radial growth was measured after 72 hr, and results were subjected to ANOVA and regression analysis.

Effects of temperature on disease. A study was conducted to determine the effect of five temperatures on disease development. Temperatures used were 15, 20, 25, 30, and 35 C. Four 8 × 8 cm pots with infested soil and four with noninfested soil, each containing 10 seeds per pot, were maintained at each temperature. Stand counts were made 14 days after planting and data analyzed

Accepted for publication 24 April 1991 (submitted for electronic processing).

using ANOVA and regression analysis. A second study was conducted to determine whether temperature affected the efficacy of fungicide seed treatments. Tests were conducted with infested soil in flats incubated at 15, 25, and 35 C. The seed treatments previously described were planted in four replicate flats at each temperature. Each treatment had 20 seeds per replication planted at a depth of 2.5 cm. Emergence was determined 14 days after planting. Plants in each row of each flat were harvested 21 days after planting, dried at 80 C for 48 hr, and weighed.

Duration of triadimenol activity. To determine the time of infection by *R. solani* and length of fungicide activity, seeds were planted in infested soil and grown in an incubator at 25 C. Seed treated with triadimenol (9.8 ml/10 kg) and untreated seed were planted in 8 × 8 cm pots with 25 seeds per pot. Beginning 48 hr after planting, plants from five pots were harvested for each of the two treatments every 24 hr for 10 days and then every 5 days until 25 days after planting. Seed and seedlings were washed free of soil, washed again for 15 min in running tap water, and dissected. Approximately 100 seed and coleoptile pieces for each treatment were placed on PDA amended with 0.3 g/L of streptomycin sulfate. After an incubation period of 24 hr at room temperature, the number of pieces colonized by *R. solani* were counted and recorded as percent infection. In all studies where emergence was determined, data were analyzed by analysis of variance and regression. Data recorded as percentages were subjected to arcsine transformation before analysis and mean separation was achieved with Duncan's multiple range test. All experiments were repeated at least once.

Table 1. Radial growth (mm) of *Rhizoctonia solani* anastomosis group 4 (AG-4) in vitro after 3 days on potato-dextrose agar amended with fungicides at four rates

	Concentration (μg a.i./ml)					
Fungicide	**0.0**	**0.01**	**0.1**	**1.0**	**10.0**	R^{2y}
Imazalil	75.6	75.5 a[z]	73.6 ab	72.3 a	51.7 a	0.76
Difenoconazole	75.6	75.6 a	71.4 ab	61.2 c	49.2 a	0.80
Nusan + Nuzone	75.6	74.2 a	74.5 ab	67.8 ab	39.2 b	0.82
Triadimenol	75.6	75.8 a	71.1 b	39.9 d	20.1 c	0.95
UBI1886	75.6	74.0 a	76.6 a	62.9 bc	14.4 d	0.96

[y] All regression values were significant at $P = 0.05$.
[z] Treatment means within a column followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

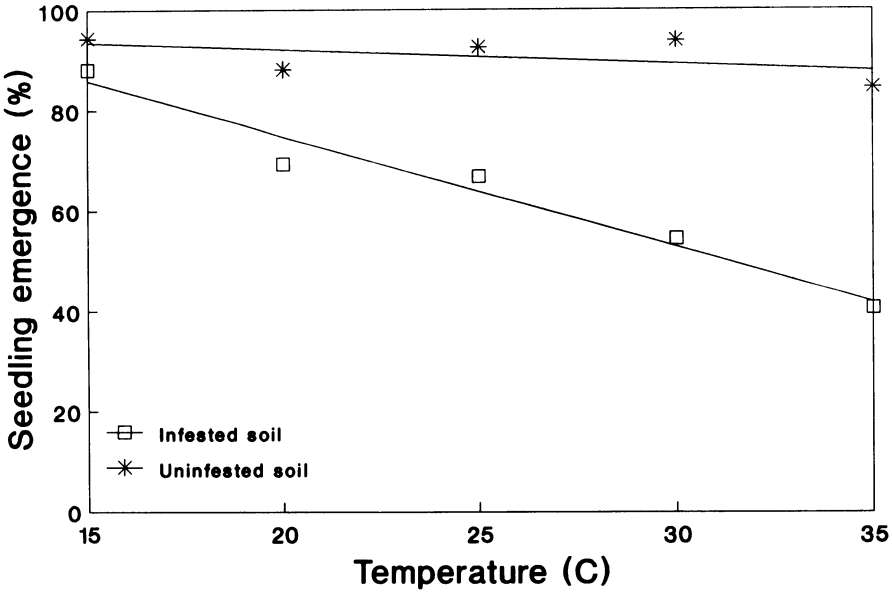

Fig. 1. Relationship between temperature and wheat seedling emergence in soil uninfested ($R^2 = 0.09$) or infested ($R^2 = 0.50$) with *Rhizoctonia solani* AG-4.

Table 2. Percent seedling emergence 14 days after sowing fungicide treated and untreated wheat seed in soil infested with *Rhizoctonia solani* anastomosis group 4 (AG-4) at three temperatures

	Temperature (C)		
Treatment	**15**	**25**	**35**
Triadimenol	94.4 aA[z]	93.1 aA	88.8 aB
Difenoconazole	89.1 bcA	58.1 bB	65.9 bB
UBI1886	82.8 cA	52.5 bB	48.1 cB
Nusan + Nuzone	83.1 cA	48.8 bcB	45.0 cB
Imazalil	68.8 dA	39.7 cdB	35.9 dB
Untreated check	62.2 dA	35.0 dB	31.8 dB

[z] Treatment means followed by the same letter (lowercase for columns and uppercase for rows) do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

RESULTS

In vitro toxicity test. As indicated by ANOVA, there was no interaction between study by chemical or study by chemical rate so data from all repeated tests were merged. Each of the fungicides tested inhibited growth of *R. solani* (Table 1). When the effects of chemical concentration on fungal growth were analyzed by regression, R^2 values were significant at $P = 0.05$ and ranged from 0.76 to 0.96. In most cases, the data were best described by nonlinear equations. There was little inhibition of radial growth at rates of 0.01–0.1 μg, but at higher rates, differences among products were apparent, and inhibition of growth by triadimenol was greater than the other products, with the exception of UBI1886 at 10 μg/ml.

Effects of temperature and disease on seedling emergence. At each of the five temperatures tested, significantly fewer plants emerged from infested soil than noninfested soil (Fig. 1). In infested soils, as temperatures increased from 15 to 35 C, emergence decreased ($R^2 = 0.50$, $P = 0.05$). In noninfested soil, temperature had no significant effect on emergence ($R^2 = 0.09$).

Seed treatment and temperature effects on disease. At each temperature, the emergence of seed treated with triadimenol was significantly greater than all other treatments (Table 2). The ranking of chemical treatments with regard to seedling emergence was the same at each of the temperatures tested. Seedlings from seed treated with triadimenol had the highest percent emergence, followed by difenoconazole, UBI1886, Nusan + Nuzone, imazalil, and the untreated check. All treatments had significantly lower emergence at 25 and 35 than at 15 C, except the triadimenol treatment, where emergence at 25 C equaled emergence at 15 C.

In addition to emergence, dry matter production was also affected by the different treatments. With all treatments except triadimenol, total dry weight was significantly greater at 15 than at 25 or 35 C (Table 3). For the seed treated with triadimenol, temperature had no effect on total dry weight produced.

Duration of triadimenol activity. Untreated seeds were infected by *R. solani* as early as 48 hr after placement in the soil, and differences between

treated and untreated seed in percent infection were significant ($P = 0.05$) at every sampling date (Fig. 2). The amount of infection in the untreated seed exceeded 80% at 5 days compared with approximately 5% for the treated seed. The treated seed continued to have a much lower incidence of infection compared with the check up to and including day 25.

Infection of coleoptiles from treated seed was also significantly less than coleoptiles from untreated seed (Fig. 3). On all but two sampling dates, the percentage of coleoptiles from untreated seed was significantly greater than that on coleoptiles from treated seed. When coleoptile infection over time was analyzed by regression, the coefficient of determination was low and nonsignificant, $R^2 = 0.09$, for the untreated control. However, infection of coleoptiles from treated seed increased significantly over time ($R^2 = 0.57$, $P \leq 0.05$).

DISCUSSION

Growth of *R. solani* in vitro was severely inhibited by triadimenol at ≥1 μg/ml of active ingredient. This concentration is lower than those reported in grain or tissue by Thielert et al (18). They applied a 25% a.i. dry formulation to seed and found that 11.4 and 4.98% of labeled triadimenol was present after 21 days in seed and shoots, respectively. On a unit basis, this is equivalent to 41.6 and 18.7 μg/g for the seed and shoots, respectively. Assuming a similar rate of uptake and translocation, the seed and shoot in our study would be expected to contain up to 25.6 and 11.5 μg of the active ingredient after 21 days. These quantities are well above the amounts found to be inhibitory to *R. solani* in vitro.

Temperature greatly affected the activity of the AG-4 isolate used in our study. The R^2 values for plant emergence in infested soil at the various temperatures was significant, and as temperature increased, emergence decreased. This demonstrates why it is beneficial for growers to plant later in the year when temperatures are cooler. Not all *Rhizoctonia* isolates, however, behave the same way with regard to temperature. The various anastomosis groups differ greatly in activity and virulence, depending on temperature (3). At cool temperatures, isolates of AG-3 are found to be more virulent on potatoes than at higher temperatures (3). Higher temperatures, however, had the opposite effect on isolates of AG-5 and AG-8, and these were more virulent on potatoes at warmer temperatures (3).

The reaction of various isolates of *R. solani* to fungicides has been found to vary within and among the different anastomosis groups (2). Of all of the fungicides tested in our study, triadimenol appears to have the greatest activity against *R. solani*. It was equally effective against our isolate at low and high temperatures, even though the isolate used in these studies caused more disease at higher temperatures. Plants emerging from seed treated with the fungicides were usually able to survive postemergence infections, leading us to conclude that the fungal isolate used was most damaging to wheat preemergence. In these studies, triadimenol was most

Table 3. Total dry matter (g) produced per row of plants surviving in soil infested with *Rhizoctonia solani* anastomosis group 4 (AG-4) after 21 days at three temperatures

	Temperature (C)		
Treatment	**15**	**25**	**35**
Triadimenol	0.23 abA[z]	0.22 aA	0.26 aA
Difenoconazole	0.23 aA	0.14 bB	0.20 bA
UBI1886	0.21 abA	0.11 cB	0.13 cB
Nusan + Nuzone	0.21 bA	0.10 cB	0.13 cB
Imazalil	0.17 cA	0.09 cB	0.10 cB
Untreated check	0.16 cA	0.08 cB	0.09 cB

[z]Treatment means followed by the same letter (lowercase for columns and uppercase for rows) do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

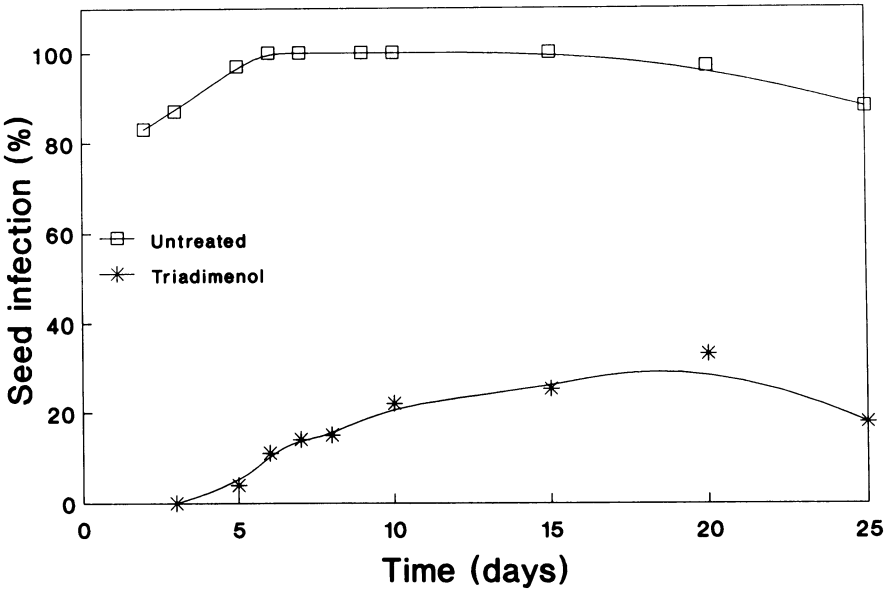

Fig. 2. Infection of treated or untreated wheat seed by *Rhizoctonia solani* AG-4 over time.

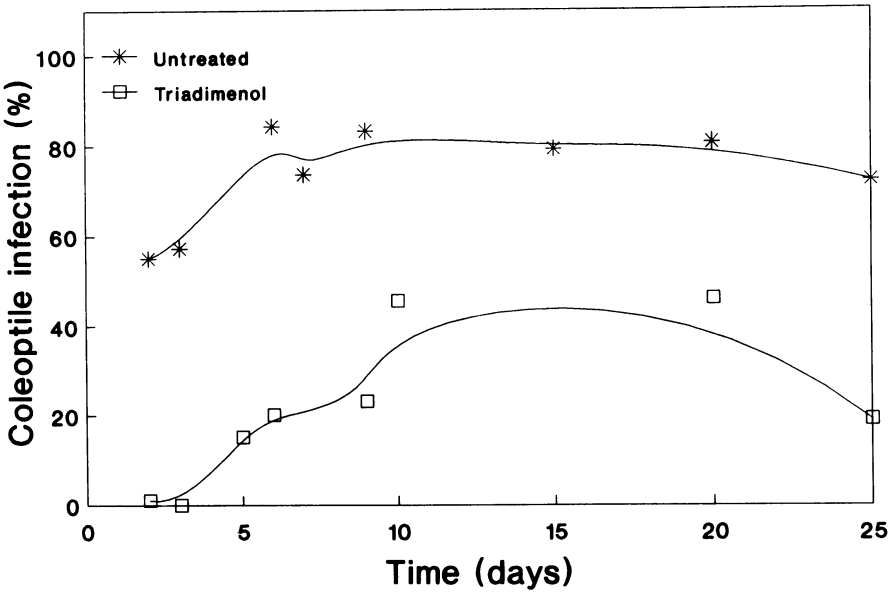

Fig. 3. Effects of seed treatment and time on infection of wheat coleoptiles by *Rhizoctonia solani* AG-4.

effective in producing higher plant populations and dry matter in infected seedlings (Tables 2 and 3). With the exception of imazalil, which failed to increase emergence or dry weight in any of the tests, the other fungicide treatments also improved emergence but not dry weight. Smiley et al (15) also tested a number of compounds to control Rhizoctonia root rot of wheat. Working with AG-8 of *R. solani* and *R. oryzae* Ryker & Gooch, they found all of the treatments were ineffective or unreliable for controlling the disease in the field. Flutolonil and tolclofos-methyl, however, were both toxic to *R. solani* AG-8 and *R. oryzae* in vitro (14).

In the duration study, triadimenol was effective in controlling infection by *R. solani* for up to 25 days. Other researchers have also reported triadimenol seed treatment confers protection against pathogenic fungi for several weeks. Bockus (1) showed triadimenol could be effective against *Gaeumannomyces graminis* (Sacc.) Arx & D. Oliver var. *tritici* J. Walker for up to 8 wk, and Frank et al (4) showed effective control of *Erysiphe graminis* DC. f. sp. *tritici* Ém. Marchal up to growth stage 10.5 on wheat on the Feekes scale (5). In our study, approximately 20% of the seed treated with triadimenol were infected with *R. solani* after 25 days. This level of infection may have been the result of inadequate coverage of the fungicide on the seed. However, the method of fungicide application we used was similar to commercial weight/volume methods. When such methods are employed, variation in application rate of ±20% is considered acceptable by the seed industry (K. Rushing, Gustafson, *personal communication*). The use of more advanced applicators with an accuracy of ±5% will allow producers to gain the full benefit of seed treatments. Also, as time passes, the triadimenol present on the seed may be metabolized by the plant, thus lowering the concentration of active ingredient present needed to control infection. Residue levels of triadimenol do decrease over time and wheat may be grazed for forage 6 wk after planting.

Providing producers with a product effective in reducing seedling diseases will enable them to plant earlier with reduced concern over potential stand loss. Triadimenol is effective against *R. solani* AG-4 and can be used to improve plant populations and should be considered as a management tool for reducing disease in early-planted wheat.

LITERATURE CITED

1. Bockus, W. W. 1983. Effects of fall infection by *Gaeumannomyces graminis* var. *tritici* and triadimenol seed treatment on severity of take-all in winter wheat. Phytopathology 73:540-543.
2. Carling, D. E., Helm, D. J., and Leiner, R. H. 1990. In vitro sensitivity of *Rhizoctonia solani* and other multinucleate and binucleate *Rhizoctonia* to selected fungicides. Plant Dis. 74:860-863.
3. Carling, D. E., and Leiner, R. H. 1990. Effect of temperature on virulence of *Rhizoctonia solani* and other *Rhizoctonia* on potato. Phytopathology 80:930-934.
4. Frank, J. A., and Ayers, J. E. 1986. Effect of triadimenol seed treatment on powdery mildew epidemics on winter wheat. Phytopathology 76:254-257.
5. Large, E. C. 1954. Growth stages in cereals: Illustrations of the Feekes scale. Plant Pathol. 3:128-129.
6. McDonald, H. J., and Rovira, A. D. 1985. Development of inoculation techniques for *Rhizoctonia solani* and its application to screening cereal cultivars for resistance. Pages 174-176 in: Ecology and Management of Soilborne Plant Pathogens. C. A. Parker, A. D. Rovira, K. J. Moore, P. T. W. Wong, and J. F. Kollmorgen, eds. American Phytopathological Society, St. Paul, MN.
7. Neale, S. M. 1985. *Rhizoctonia* in South Australian wheat fields. Pages 54-56 in: Ecology and Management of Soilborne Plant Pathogens. C. A. Parker, A. D. Rovira, K. J. Moore, P. T. W. Wong, and J. F. Kollmorgen, eds. American Phytopathological Society, St. Paul, MN.
8. Pumphrey, F. V., Wilkins, D. E., Hare, D. C., and Smiley, R. W. 1987. Influence of tillage and nitrogen fertilizer on Rhizoctonia root rot (bare patch) of winter wheat. Plant Dis. 71:125-127.
9. Rovira, A. D. 1986. Influence of crop rotation and tillage on Rhizoctonia bare patch of wheat. Pytopathology 76:669-673.
10. Rovira, A. D., Ogoshi, A., and McDonald, H. J. 1986. Characterization of isolates of *Rhizoctonia solani* from cereal roots in South Australia and New South Wales. Phytopathology 76:1245-1248.
11. Rovira, A. D., and Venn, N. R. 1985. Effect of rotations and tillage on take-all and Rhizoctonia root rot in wheat. Pages 255-258 in: Ecology and Management of Soilborne Plant Pathogens. C. A. Parker, A. D. Rovira, K. J. Moore, P. T. W. Wong, and J. F. Kollmorgen, eds. American Phytopathological Society, St. Paul, MN.
12. Rush, C. M., and Mathieson, J. T. 1990. Effects of common root rot on winter wheat forage production. Plant Dis. 74:982-985.
13. Samuel, G., and Garrett, S. D. 1932. *Rhizoctonia solani* on cereals in South Australia. Phytopathology 22:827-836.
14. Smiley, R. W., Uddin, W., Ott, S., and Rhinhart, K. E. L. 1990. Influence of flutolanil and tolclofos-methyl on root and culm diseases of winter wheat. Plant Dis. 74:788-791.
15. Smiley, R. W., Wilkins, D. E., and Klepper, E. L. 1990. Impact of fungicide seed treatments on Rhizoctonia root rot, take-all, eyespot, and growth of winter wheat. Plant Dis. 74:782-787.
16. Specht, L. P., and Rush, C. M. 1988. Fungi associated with root and foot rot of winter wheat and populations of *Cochliobolus sativus* in the Texas Panhandle. Plant Dis. 72:959-963.
17. Sterne, R. E., and Jones, J. P. 1978. Sharp eyespot of wheat in Arkansas caused by *Rhizoctonia solani*. Plant Dis. Rep. 62:56-60.
18. Thielert W., Steffens, W., Fuhr, F., Kuck, K. H., and Scheinpflug, H. 1988. Uptake of triadimenol through wheat caryopris after application by seed treatment. Pestic. Sci. 22:93-105.
19. Weller, D. M., Cook, R. J., MacNish, G., Bassett, E. N., Powelson, R. L., and Petersen, R. R. 1986. Rhizoctonia root rot of small grains favored by reduced tillage in the Pacific Northwest. Plant Dis. 70:70-73.
20. Wiese, M. V. 1987. Compendium of Wheat Diseases. 2nd ed. American Phytopathological Society, St. Paul, MN. 112 pp.
21. Winter, S. R., and Thompson, E. K. 1987. Grazing duration effects on wheat growth and grain yield. Agron. J. 79:110-114.