Factors Affecting Control of Take-All of Spring Wheat by Seed Treatment with Sterol Biosynthesis-Inhibiting Fungicides

CELSA GARCIA and D. E. MATHRE, Department of Plant Pathology, Montana State University, Bozeman 59717

ABSTRACT

Garcia, C., and Mathre, D. E. 1987. Factors affecting control of take-all of spring wheat by seed treatment with sterol biosynthesis-inhibiting fungicides. Plant Disease 71:743-746.

Eight sterol biosynthesis-inhibiting fungicides, nuarimol, imazalil, prochloraz, triadimenol, bitertanol, propiconazol, etaconazole, and diniconazole, were tested in vitro against *Gaeumannomyces graminis* var. *tritici* (*G. g.* var. *tritici*). All were strongly inhibitory at 10 μM, and imazalil and prochloraz were effective at 0.01 μM. Field tests of five of these compounds applied as seed treatments on Pondera spring wheat when inoculum was placed with the seed indicated a difference in efficacy in reducing symptom severity and increasing grain yield. Triadimenol and prochloraz were the most effective at rates above 0.3 g a.i./kg. Studies in the field and greenhouse on inoculum location vs. efficacy of triadimenol seed treatment showed that this compound is most effective in reducing infection when the inoculum is below the seed.

Take-all, caused by *Gaeumannomyces graminis* (Sacc.) Arx & Oliv. var. *tritici* Walker (*G. g.* var. *tritici*), has been reported from most regions where wheat is grown (1). This disease can be controlled with crop rotation or continuous production of wheat; the latter involves a biological suppression known as take-all decline (1).

Additional control measures for take-all, such as fungicidal seed treatments, are needed. Gorska-Paczapko (14) found benomyl to be the best in vitro mycelial growth inhibitor (1 μg a.i./ml) among several systemically translocated fungicides tested against *G. g.* var. *tritici*. It also had activity against *G. g.* var. *tritici* in greenhouse tests when applied as a seed dressing (2 g a.i./kg of seed). Pren and McIntosh (17), however, did not observe activity of benomyl against take-all in naturally infested fields. Ballinger and Kollmorgen (2) found that benomyl significantly reduced the disease in the greenhouse but not in the field. In contrast, Bateman (3–8) and Bateman and Nicholls (9) found benomyl to suppress take-all when applied as a soil drench in the greenhouse and in the field.

Dolezal and Jones (12) reported that seed treatment with triadimefon significantly reduced yield losses of winter wheat in fields artificially infested with *G. g.* var. *tritici*. Similarly, triadimenol protected winter wheat seedlings in the greenhouse for 8 wk and increased yield by 38% in the field (10). Mathre et al (16) also found that triadimenol reduced infection and increased yield of spring wheat in naturally and artificially infested fields.

The purpose of this study was to evaluate eight sterol biosynthesis-inhibiting fungicides (18) for their potential as seed treatments in controlling take-all on spring wheat. We also evaluated several factors affecting the performance of seed treatment in controlling take-all.

MATERIALS AND METHODS

In vitro mycelial growth tests. Eight sterol biosynthesis-inhibiting chemicals were tested for their effects on mycelial growth of *G. g.* var. *tritici*, including nuarimol (mol wt 315), imazalil (mol wt 297), prochloraz (mol wt 376), triadimenol (mol wt 296), bitertanol (mol wt 337), propiconazol (mol wt 329), etaconazole (mol wt 315), and diniconazole (mol wt 326). Each fungicide was tested at 100, 10, 1, 0.1, and 0.01 μM of active ingredient in potato-dextrose agar (PDA) or Czapek agar (CA). Two experiments were conducted with PDA, and one was conducted with CA. Triadimenol was not included in the CA test. CA was used because it does not contain sterols, which if present, might interfere with the tests. Dilutions of the fungicides were prepared in sterile water except for bitertanol, which was diluted with 95% ethanol. The solution/suspension was added to molten agar medium at 50 C, mixed, and poured into four petri plates (100 × 15 mm). After solidification, each plate was inoculated with an 11-mm disk of *G. g.* var. *tritici* mycelium. After incubation at room temperature, mycelial growth was measured on the eighth day. Data (total accumulated growth) were subjected to a factorial analysis of variance (ANOVA), with fungicides and concentrations as factors. Fungicides were compared only within the same concentration.

Inoculum preparation. Oat kernels were autoclaved. The next day, one petri plate culture of *G. g.* var. *tritici* was chopped into small pieces and mixed with each jar containing 250 g of oats. The cultures were incubated at room temperature for 3 wk, air-dried for 1 wk, then fragmented briefly in a Waring Blendor.

Planting. Two field experiments were planted 3 May 1985 on the A. H. Post Research Farm near Bozeman. Plots were four rows wide × 3 m long and seeded 2.5 cm deep with 6 g of Pondera spring wheat per row. *G. g.* var. *tritici* inoculum was added with the seed except as noted; the treatments with no inoculum received autoclaved oats in an amount equal to the highest inoculum rate, whereas treatments with less than the maximum rate were supplemented with autoclaved oats so that each row received the same quantity of oat kernels.

Evaluation of fungicides. Based on the results of the in vitro tests, five fungicides were selected for testing as seed treatments at the following doses: triadimenol at 0.16, 0.31, and 0.47 g a.i./kg; propiconazol at 0.01, 0.02, and 0.04 g a.i./kg; diniconazole at 0.11 and 0.22 g a.i./kg; prochloraz at 0.20 and 0.40 g a.i./kg; and imazalil at 0.05 and 0.10 g a.i./kg. Inoculum rates used were 0, 1, 2, and 5 g of inoculum per row. This experiment was planted with four replicates in a split-plot design with inoculum rates as the main plots and fungicides as subplots.

Six weeks after planting, five plants from each of the border rows were pulled, the roots were washed free of soil, and lesion severity was assessed by the following scale: 1 = no symptoms, leaves green; 2 = discoloration of the roots only, leaves green; 3 = discoloration in the root and crown tissues, leaves green; 4 = discoloration of the entire crown, roots heavily discolored, leaves somewhat chlorotic; and 5 = complete discoloration of the crown, severe rotting of roots, plants nearly dead. From this, a disease index (DI) was calculated that equaled the sum of the number of plants in each category times the infection score divided by the total number of plants. All plots were also scored for aboveground

Contribution from the Montana Agricultural Experiment Station. Journal Series Paper J-1923.

Accepted for publication 2 April 1987 (submitted for electronic processing).

symptoms 8 and 12 wk after planting. A scale of 1–5 was used where 1 = healthy plants and 5 = severely stunted plants with a very low population of plants remaining. Grain yield was evaluated from the two inner rows. ANOVAs were performed to examine the effects of inoculum rates and fungicides. If statistical differences were indicated by ANOVA, comparisons among fungicides were made within each inoculum rate.

Because lesion severity was scored with discrete values, nonnormality of data and nonhomogeneity of variances were suspected. For this reason, these data were analyzed by the CATMOD (Categorical Data Modeling) procedure of the SAS (Statistical Analysis System) program. The raw scores were converted to number of plants in each response category and analyzed using these counts.

Effect of inoculum placement. In the field tests described previously, the *G. g.* var. *tritici* inoculum was close to the seed because it was added to the furrow at the time of seeding. In a second experiment, inoculum was placed in the furrow with the seed (0, 2, or 4 g/3 m of row) or broadcast on the soil surface (90 or 180 kg/ha) and then rototilled in to a depth of 0–10 cm. Seed treatments consisted of triadimenol at 0.31 g a.i./kg of seed, propiconazol at 0.02 g a.i./kg of seed, and an untreated check. This test was established as a split-plot design with four replicates, with inoculum rates as the main plots and fungicides as subplots. Six weeks after planting, five plants from each of the border rows were pulled, roots were washed free of soil, and lesion severity was assessed as described previously. All plots were scored for aboveground symptoms 8 and 12 wk after planting. Percent white heads was determined in 1-m sections from each of the two inner rows. Data on lesion severity were analyzed by the SAS-CATMOD procedure.

Table 1. Effects of sterol biosynthesis-inhibiting fungicides applied as seed treatments to Pondera spring wheat on severity of take-all disease after 6 wk in the field

Fungicide	Dose (g a.i./kg)	Root disease index[a] for each inoculum rate (g/3 m)			
		0	1	2	5
Triadimenol	0.16	1.0[b]	2.1	2.3	3.2
	0.31	1.5	1.9	1.8	2.7
	0.47	1.0	1.6	1.9	2.3
Propiconazol	0.01	1.4	2.0	3.5	3.8
	0.02	1.3	2.1	3.2	3.8
	0.04	1.0	2.7	3.2	3.8
Diniconazole	0.11	1.9	2.5	3.3	3.8
	0.22	1.3	1.5	2.7	3.8
Prochloraz	0.20	1.3	2.6	2.6	3.5
	0.40	1.2	2.5	2.8	3.4
Imazalil	0.05	1.1	2.3	3.0	3.3
	0.10	1.2	2.5	2.4	3.4
Untreated	···	1.3	2.5	3.4	3.5

[a] DI = [(no. of plants) × (infection score)]/total no. of plants; infection score: 1 = no visible symptoms to 5 = plants nearly dead.
[b] LSD = 0.6 ($P = 0.05$) within columns.

Table 2. Effects of sterol biosynthesis-inhibiting fungicides applied as seed treatments to Pondera spring wheat on severity of take-all after 8 and 12 wk in the field

Fungicide	Dose (g a.i./kg)	Foliar disease index[a] for each inoculum rate (g/3 m)							
		8 wk				12 wk			
		0	1	2	5	0	1	2	5
Triadimenol	0.16	1.0[b]	1.1	1.7	1.8	1.0	2.2	2.0	3.2
	0.31	1.0	1.1	1.1	1.4	1.0	1.0	1.2	2.5
	0.47	1.0	1.1	1.0	1.1	1.0	1.5	1.7	2.2
Propiconazol	0.01	1.0	1.4	2.4	4.1	1.0	2.7	3.5	4.7
	0.02	1.1	1.5	2.4	4.0	1.0	3.0	3.2	4.5
	0.04	1.1	1.9	2.2	4.1	1.2	3.0	3.2	4.7
Diniconazole	0.11	1.0	1.2	2.4	3.9	1.0	2.0	3.0	4.5
	0.22	1.2	1.4	2.0	3.9	1.0	2.0	3.0	4.7
Prochloraz	0.20	1.0	1.3	1.8	3.6	1.0	2.7	2.5	4.5
	0.40	1.0	1.4	1.9	3.2	1.0	2.2	2.7	3.7
Imazalil	0.05	1.2	1.7	2.4	3.7	1.7	2.5	3.2	4.2
	0.10	1.3	2.1	2.6	3.6	1.2	3.0	3.2	3.7
Untreated	···	1.0	1.2	2.2	3.9	1.2	2.7	3.2	4.7

[a] Scale of 1–5, where 1 = no visible symptoms and 5 = severely stunted plants and very low population of plants remaining in the plot.
[b] LSD = 0.6 ($P = 0.05$) within columns.

Greenhouse tests on inoculum location. To determine more precisely whether the location of inoculum in relation to a triadimenol-treated seed affects the activity of this fungicide, tests were set up in the greenhouse with inoculum location as a variable. A growth medium of fen peat moss, fine sand, and Bozeman silt loam (1:1:1, v/v) was used to fill 16.5-cm-deep × 3-cm-wide tapered plastic containers. Fragmented *G. g.* var. *tritici*-oat kernel inoculum was mixed with the growth medium at rates of 0.1, 1, and 5% (w/w). The inoculum was placed 1) from 1 cm below the seed to the bottom on the container, 2) 1 cm above the seed in a band 2 cm thick, or 3) throughout the medium. The experiment was conducted as a split-plot design with five replicates, using fungicide doses of 0, 0.16, 0.31, or 0.64 g a.i./kg seed as main plots, and inoculum rate and position as subplots. Each container received four seeds of Pondera spring wheat planted 7 cm deep. This experiment was conducted twice.

The containers were irrigated as needed with one-half-strength Hoagland's solution. Plants were harvested after 5 wk for the first experiment and after 6 wk for the second. Soil was washed from the roots, and infection was assessed with the scale described previously. Raw scores of infection severity of both experiments were analyzed by the SAS-CATMOD procedure.

RESULTS

All of the fungicides tested at 100 and 10 μM inhibited mycelial growth on PDA. At lower concentrations, some fungicides were more effective than others in reducing mycelial growth. At 1 μM, diniconazole, prochloraz, etaconazole, and triadimenol were all equally effective, reducing growth by at least 85%; bitertanol was the least inhibitory but still reduced growth by 54% at 1 μM. At the lowest concentration tested, 0.01 μM, prochloraz and imazalil inhibited growth by at least 71%, whereas the remaining compounds were minimally inhibitory.

When the fungicides were tested in CA, their effects were generally more severe than when tested in PDA. No mycelial growth on CA occurred at 100 μM with any of the fungicides, but some growth was observed at 10 μM with several of the fungicides. Bitertanol was again the least toxic of the eight fungicides. Nuarimol, prochloraz, propiconazol, etaconazole, and diniconazole were strongly inhibitory at concentrations of 1 μM but less so at lower concentrations. Contrary to the test on PDA, imazalil and prochloraz were slightly inhibitory at 0.1 and 0.01 μM.

Evaluation of fungicides in the field. The main factors, inoculum rate and fungicides, and the interaction of inoculum rate × fungicides were significant for all parameters evaluated in this

experiment. When the fungicide doses were compared within each inoculum rate (Table 1), the DI on seedlings varied from 1 to 1.9 for all uninoculated treatments. With 1 g of inoculum per row, the DI of the plants in the untreated plots was 2.5. Even at this low level of infection, fungicides at the highest rates tested differed in their ability to protect the plants. The best control was provided by diniconazole at 0.22 g a.i./kg and by triadimenol at 0.47 g a.i./kg. The DI for other compounds and rates did not differ significantly from that of the untreated check. When the inoculum rate was doubled to 2 g/row, the DI of the plants in the untreated plots increased to 3.4. The best control was achieved with triadimenol. Diniconazole, imazalil, and prochloraz provided intermediate control. At 5 g of inoculum per row, the DI of plants in the untreated plots was 3.5 and triadimenol was the only fungicide that significantly lowered the DI.

Foliar symptoms on more mature plants clearly showed the superiority of triadimenol in reducing the effects of take-all (Table 2). By the eighth week, few foliar symptoms were observed at the inoculum rate of 1 g/row. At 2 g/row, there was clear evidence of diseased plants, and only the two highest doses of triadimenol prevented symptoms from developing at this time. At the rate of 5 g/row, most plots showed severe effects of the disease but plants in triadimenol plots showed greatly reduced symptoms. Four weeks later, disease severity had increased considerably, and no seed treatment prevented all disease symptoms. Triadimenol, prochloraz, and imazalil significantly reduced symptom severity at the highest rate of inoculation.

Grain yield was also adversely affected by the amount of inoculum (Table 3). Inoculation with 1 and 2 g of inoculum resulted in grain yield reductions of 33 and 63%, respectively, in the absence of fungicides. The two highest doses of triadimenol maintained yields at levels comparable to the untreated-uninoculated check. None of the other fungicides provided this level of protection. When the inoculum pressure was very high (5 g), triadimenol did not prevent yield reduction completely. At the highest dose of triadimenol, there was a 40% yield reduction compared with the uninoculated check; however, the yield was three times higher than that in the untreated check.

Effect of inoculum placement. The level of infection in plots in which the inoculum was rototilled into the soil was not as high as when the inoculum was placed close to the seed. By the sixth week in the untreated plots, 4 g of inoculum in the furrow produced a significantly higher DI than that of the uninoculated check. Although 4 g of inoculum per 3 m of row represents only 43 kg of inoculum per hectare, the infection and damage produced by this amount of inoculum in the seed furrow was almost twice as high as that produced by the highest broadcast-rototilled inoculum rate of 180 kg/ha. Propiconazol and triadimenol did not have a significant effect on DI where the inoculum was broadcast.

By the eighth week, the effect of root infection was still not visually reflected in the aerial portions of the plants. By 12 wk, however, the effects of the infection were more evident (Table 4). The broadcast inoculum treatments did not produce a high level of infection; thus, triadimenol did not show the same significant protective effect as it did when the inoculum was placed in the row. This same effect was observed in grain yield (Table 5).

Greenhouse tests on inoculum location. The fungicide × inoculum rate and inoculum rate × inoculum location interactions were not significant. The interaction of fungicide × inoculum position, however, was significant in the first experiment but not in the second. From Table 6, it is obvious that, at least for the highest rates of inoculum, there is an interaction between fungicide and inoculum location. When the inoculum was placed above the seed or throughout the growth medium, the DI for the fungicide treatments was very similar to that of the untreated check. When the inoculum was placed below the seed, there was a marked reduction in infection for all doses of triadimenol at the 1 and 5% inoculum rates.

When compared with each other, the three doses of triadimenol were different from the untreated check, but no differences were observed among doses. The average DI for plants in the untreated check across inoculum rates was 3 8. For 0.16, 0.32, and 0.64 g a.i./kg of triadimenol, the DIs were 3.0, 2.6, and 2.4, respectively. Rates of inoculum also led to statistically different DIs. The DI for all the uninoculated checks was 1.0. For 0.1, 1, and 5% inoculum, the average DIs were 1.6, 3.3, and 4.0, respectively. Only the 0.1% rate was not significantly different from the uninoculated check. Locations of inoculum were also different. The average DI was 2.7 when inoculum was placed above the seed, 1.9 when inoculum was placed below the seed, and 3.8 when inoculum was distributed throughout the container.

DISCUSSION

All of the fungicides tested were effective inhibitors of in vitro growth of *G. g.* var. *tritici*. Prochloraz and imazalil inhibited growth by more than 50% at a concentration as low as 0.01 μM. The

Table 3. Effects of sterol biosynthesis-inhibiting fungicides applied as seed treatments to Pondera spring wheat on grain yield of plants grown in field plots artificially infested with *Gaeumannomyces graminis* var. *tritici*

Fungicide	Dose (g a.i./kg)	Grain yield (g/m) for each inoculum rate (g/3 m)			
		0	1	2	5
Triadimenol	0.16	116[a]	75	72	35
	0.31	105	89	111	35
	0.47	115	93	90	60
Propiconazol	0.01	112	52	45	6
	0.02	105	69	43	14
	0.04	101	56	49	18
Diniconazole	0.11	111	83	54	10
	0.22	116	77	50	13
Prochloraz	0.20	117	66	64	20
	0.40	111	64	53	47
Imazalil	0.05	108	65	57	20
	0.10	97	68	51	15
Untreated	···	102	68	37	21

[a] LSD = 20 ($P = 0.05$) within columns.

Table 4. Effects of inoculum level and placement on severity of take-all of 12-wk-old Pondera spring wheat in the field

Inoculum placement	Inoculum rate[a]	Disease index[b]		
		Triadimenol	Propiconazol	Untreated
In-furrow	0	1.5[c]	1.0	1.0
	2	2.0	2.7	3.5
	4	3.0	4.0	3.7
Broadcast	90	1.2	1.7	1.5
	180	1.7	2.0	2.0

[a] 0 = Uninfested oat kernels placed in the furrow together with the seed; 2 and 4 = grams of infested oat kernels placed in the furrow together with the seed; and 90 and 180 = kilograms per hectare of infested oat kernels incorporated into the upper 10 cm of soil.
[b] Scale of 1–5, where 1 = no visible symptoms and 5 = severely stunted plants and very low population of plants remaining in the plot.
[c] LSD = 0.5 ($P = 0.05$) within columns.

Table 5. Interactions of seed treatment, inoculum rate, and placement of *Gaumannomyces graminis* var. *tritici* on grain yield of Pondera spring wheat

Inoculum placement	Inoculum rate[a]	Grain yield (g/m) Triadimenol (0.32 g a.i./kg)	Propiconazol (0.02 g a.i./kg)	Untreated
In-furrow	0	110[b]	107	99
	2	69	51	39
	4	61	22	38
Broadcast	90	96	95	91
	180	100	81	76

[a] 0 = Uninfested oat kernels placed in the furrow together with the seed; 2 and 4 = grams of infested oat kernels placed in the furrow together with the seed; and 90 and 180 = kilograms per hectare of infested oat kernels incorporated into the upper 10 cm of soil.
[b] LSD = 20 ($P = 0.05$) within columns.

Table 6. Effects of triadimenol seed treatment on severity of take-all of seedlings of Pondera spring wheat in the greenhouse as affected by inoculum placement

Inoculum rate[a] (%)	Inoculum placement[b]	Disease index[c] for each triadimenol dose (g/a.i./kg) 0	0.16	0.32	0.64
0.0	...	1.0	1.0	1.0	1.0
0.1	Above	1.5	1.4	1.2	1.4
0.1	Below	1.6	1.0	1.0	1.0
0.1	Around	3.3	2.9	1.7	1.5
1.0	Above	3.9	3.4	3.7	3.4
1.0	Below	4.4	1.5	1.0	1.1
1.0	Around	4.7	4.2	4.5	3.8
5.0	Above	4.8	4.7	4.2	4.2
5.0	Below	5.0	3.1	1.8	1.0
5.0	Around	5.0	4.7	4.5	4.7

[a] Percentage of infested oat kernels in growth mixture (w/w).
[b] Placement in relation to the seed.
[c] DI = [(no. of plants) (infection score)]/total no. of plants, on a scale of 1–5, where an infection score of 1 = no infection; 5 = plants nearly dead. Values are the average of two experiments.

level of sensitivity of *G. g.* var. *tritici* is very similar to that exhibited by other fungi to the C-14 dimethylation inhibitors. The minimum concentrations that inhibit growth of *Penicillium italicum* are 0.01–0.05 μg/ml, equivalent to about 0.1 μM (15,19). Larger amounts of imazalil were needed to inhibit growth of *Aspergillus nidulans* (19). Bitertanol was the least inhibitory of all the fungicides to *G. g.* var. *tritici*.

Although all of the fungicides were toxic to mycelium of *G. g.* var. *tritici*, only triadimenol greatly influenced disease development in the field. The fungicides diniconazole, imazalil, and prochloraz provided intermediate protection of young plants at moderate levels of inoculum, but under high inoculum pressure, only triadimenol was effective.

Inoculum rate greatly influenced infection severity, with losses in grain yield as high as 80%. Under these circumstances, only triadimenol prevented high levels of infection and thus reduced yield losses. From this study, it was not possible to determine why the other fungicides, which were toxic in vitro, failed to influence disease severity. Not all fungicides used, however, were formulated as seed treatments, and some may not have moved into the plant to provide systemic protection. Steffens et al (21) have shown that ^{14}C-labeled triadimenol applied as a seed treatment to wheat and barley diffuses into the soil surrounding the seed, forming a zone of protection near the seed. It also is translocated into the developing shoot, but most of it accumulates in the leaf tips. Therefore, it is likely that triadimenol protects the plant by preventing or delaying the growth of runner hyphae from the roots past the seed into the subcrown internode and other portions of the crown.

Greenhouse results indicated that triadimenol provides efficient protection to the lower stem of the plant above the seed but only provides limited protection to the roots. When the inoculum was placed below the seed, infection did not advance past the point of seed attachment. Although *G. g.* var. *tritici* can infect any part of the plant, Fellows (13) stated that invasion of crown tissue occurs mainly through systemic infection from the roots.

This property of triadimenol of protecting crown tissue from invasion, and the capacity of wheat and barley to produce adventitious roots as long as crown tissues are not damaged, make triadimenol a very promising alternative for the control of take-all on spring wheat, particularly when combined with biological control and use of chloride (11) and/or ammonium fertilizer (20).

ACKNOWLEDGMENTS

This work was supported in part by grants from Mobay and Gustafson, Inc., and a fellowship to C. Garcia provided by the government of Mexico.

LITERATURE CITED

1. Asher, M. J. C., and Shipton, P. J. 1981. Biology and Control of Take-All. Academic Press, New York. 538 pp.
2. Ballinger, D. J., and Kollmorgen, J. F. 1986. Glasshouse and field evaluation of benomyl and triadimefon applied at seeding to control take-all in wheat. Plant Pathol. 35:61-66.
3. Bateman, G. L. 1980. Prospects for fungicidal control of take-all of wheat. Ann. Appl. Biol. 96:275-282.
4. Bateman, G. L. 1981. Effects of soil application of benomyl against take-all (*Gaeumannomyces graminis*) and footrot diseases of wheat. J. Plant Dis. Prot. 88:249-255.
5. Bateman G. L. 1982. Formulations of soil-applied fungicides for controlling take-all (*Gaeumannomyces graminis* var. *tritici*) in experiments with pot-grown wheat. J. Plant Dis. Prot. 89:480-486.
6. Bateman, G. L. 1984. Effects of surfactants on the performances of soil-applied fungicides against take-all (*Gaeumannomyces graminis* var. *tritici*) in wheat. J. Plant Dis. Prot. 91:345-353.
7. Bateman, G. L. 1984. Soil-applied fungicides for controlling take-all in field experiments with winter wheat. Ann. Appl. Biol. 104:459-465.
8. Bateman, G. L. 1985. The effects of distribution of two soil-incorporated fungicides on control of take-all (*Gaeumannomyces graminis* var. *tritici*) in wheat. J. Plant Dis. Prot. 92:194-203.
9. Bateman, G. L, and Nicholls, P. H. 1982. Experiments on soil drenching with fungicides against take-all in wheat. Ann. Appl. Biol. 100:197-303.
10. 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.
11. Christensen, N. W.. Taylor, R. G., Jackson, T. L., and Mitchell B. L. 1981. Chloride effects on water potentials and yield of winter wheat infected with take-all root rot. Agron. J. 73:1053-1058.
12. Dolezal, W. E., and Jones, J. P. 1981. Fungicide promising for take-all in wheat. Agrichem. Age 25:34.
13. Fellows, H. 1938. Interrelations of take-all lesions on the crown, culms, and roots of wheat plants. Phytopathology 28:191-195.
14. Gorska-Poczopko, J. 1971. Studies on chemical control of *Ophiobolus graminis* Sacc. I. Testing systemic fungicides against *Ophiobolus graminis* Sacc. Acta Phytopathol. Acad. Sci. Hung. 6:393-398.
15. Kerkenaar, A., van Rossum, J. M., Versluis, G. G., and Marsman. J. W. 1984. Effect of fenpropimorph and imazalil on sterol biosynthesis in *Penicillium italicum*. Pestic. Sic. 15:177-187.
16. Mathre, D. E., Johnston, R. H., and Engel, R. 1986. Effect of seed treatment with triadimenol on severity of take-all of spring wheat caused by Gaeumannomyces graminis var. tritici. Plant Dis. 70:749-751.
17. Pren, R. D., and McIntosh, A. H. 1975. Effects of benomyl and other fungicides on take-all, eyspot and sharp eyespot diseases of winter wheat. Plant Pathol. 24:67-71.
18. Siegel, M. R. 1981. Sterol-inhibiting fungicides: Effects on sterol biosynthesis and sites of action. Plant Dis. 65:986-989.
19. Siegel, M. R., and Ragsdale, N. N. 1978. Antifungal mode of action of imazalil. Pestic. Biochem. Physiol. 9:48-56.
20. Smiley, R. W.. and Cook, R. J. 1973. Relationship between take-all of wheat and rhizosphere pH in soils fertilized with ammonium vs. nitrate-nitrogen. Phytopathology 63:882-890.
21. Steffens. W., Fuhr, F., Kraus, P., and Scheinpflug. H. 1982. Uptake and distribution of Baytan in spring barley and spring wheat after seed treatment. Pflanzenschutz-Nachrichten 35:171-188.