Effect of Seed Treatment with Triadimenol on Severity of Take-All of Spring Wheat Caused by *Gaeumannomyces graminis* var. *tritici*

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ABSTRACT

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 Disease 70:749-751.

Triadimenol seed treatment of irrigated spring wheat delayed symptom development of take-all caused by *Gaeumannomyces graminis* var. *tritici*. Depending on the inoculum level and the dose of triadimenol on the seed, the onset of aboveground symptoms was delayed up to 53 days compared with the untreated control. Lesions on seminal roots and the subcrown internodes of 5-wk-old plants were reduced 50% by a triadimenol seed treatment at a rate of 0.22 ml a.i./kg of seed. Yield increases over the untreated controls were significant in both infested plots and a naturally infested commercial field.

Take-all of wheat caused by *Gaeumannomyces graminis* (Sacc.) Arx & Oliv. var. *tritici* Walker (*G. g.* var. *tritici*) is a severe disease when wheat (*Triticum aestivum* L.) is grown under high moisture conditions. In Montana, it is especially severe in irrigated spring wheat. Crop rotation as a control for take-all is not acceptable because alternative crops often result in reduced economic return. Although production of continuous wheat can lead to development of biological suppression of take-all, i.e., take-all decline (1), the short-term economic impact of such action can be severe on the wheat producer. As a result, there is great interest in other control measures for this disease.

Dolezal and Jones in 1981 (7) reported that seed treatment with triadimefon, a sterol-inhibiting systemic fungicide, was effective in controlling take-all of winter wheat in Arkansas. Several European reports have shown that fungicide treatment of soil by incorporating or drenching reduces take-all on winter wheat (2–5). In these tests, benomyl was the most effective compound when placed just below the seed as a soil drench.

The growing season for spring wheat in Montana is relatively short, usually lasting only 16 wk. If protection from *G. g.* var. *tritici* could be provided by seed treatment with triadimenol for 6–8 wk, as reported by Bockus (6), significant disease control and yield increase might be possible. The purpose of our study was to determine if a triadimenol seed treatment protects spring wheat from infection by *G. g.* var. *tritici* and to measure the efficacy of triadimenol under several levels of disease intensity. The experiments were done in soil with high moisture and pH, both conditions highly favorable to take-all (1).

MATERIALS AND METHODS

1983 Soil infestation tests. Ground oat kernels infested with *G. g.* var. *tritici* were used to infest plots on the Arthur Post Experiment Farm near Bozeman, MT. The study was designed as a 4 × 2 factorial with four levels of inoculum (5, 0.5, 0.05, and 0 g of infested oats per 3-m row) and two seed treatments (triadimenol at 0.29 ml a.i./kg and untreated). Single-row 3-m plots of the spring wheat cultivar Pondera at 56.5 kg/ha were planted in a split-plot design with inoculum rate as main plots and seed treatment as subplots and replicated four times. Inoculum that weighed less than 5 g was blended with autoclaved ground oat kernels so that each row received a total of 5 g of oats. Infested oats were added to the furrow at seeding.

Symptoms were assessed six times beginning 45 days after seeding and about every 10 days thereafter. A foliar disease scoring system of 0 = no symptoms to 5 = all plants showing severe stunting and chlorosis was used. Yield was determined only for the 0.5-g inoculum rate. The plot received 22 cm of precipitation during the growing season, but no irrigation was done.

1985 Soil infestation tests. A 4 × 4 factorial test was established at Bozeman with inoculum levels of 0, 0.5, 1, and 5 g of inoculum per 3 m and triadimenol levels of 0, 0.15, 0.22, and 0.29 ml a.i./kg of Pondera spring wheat seed. The experiment was seeded at the rate of 56.5 kg/ha in three-row 3-m plots on 30-cm centers in a randomized complete block design replicated four times. A second test was established at Bozeman and on the Southern Agricultural Research Center near Huntley, MT. A 2 × 2 factorial test with either 0 or 2 g of *G. g.* var. *tritici* inoculum per 3 m and seed treatment with either triadimenol (0.22 ml a.i./kg) or carboxin + thiram (Vitavax 200) (1.95 ml of formulation per kilogram) was established. The four-row plots, which were seeded with Pondera spring wheat at 56.5 kg/ha, were 3 m long, spaced 30 cm apart, and arranged in a randomized complete block design replicated four times.

Soil analysis from surface samples (0–15 cm) was as follows: At the Bozeman location, pH 7.0, 1.7% organic matter, 8 ppm nitrate-nitrogen, 7 ppm sodium bicarbonate-extractable phosphorus, and 188 ppm ammonium acetate-extractable potassium was present. At the Huntley location, pH 8.0, 1.8% organic matter, 16 ppm nitrate-nitrogen, 4 ppm phosphorus, and 620 ppm potassium was present. The Huntley plot received $NaNO_3$ broadcast at a rate of 102 kg/ha of nitrogen + 10 kg/ha of phosphorus applied with the seed as P_2O_5. Both sites received sprinkler irrigation or rain during the growing season totaling 27 cm at Huntley and 19 cm at Bozeman.

Plants were collected from the two outside rows 5 wk after seeding from the test at Huntley and from the second test at Bozeman. Roots were washed free of soil, and lesion severity was assessed on 15 plants per plot. The scale used was as follows: 0 = no symptoms on roots; 1 = one or two lesions on seminal roots; 2 = three to five lesions on seminal roots but no lesions on subcrown internode (SCI); 3 = six or more lesions on seminal roots, one or two lesions on SCI; 4 = numerous lesions on seminal roots, three to five lesions on SCI; and 5 = complete blackening of SCI.

Plants in all infested tests were scored for aboveground symptoms about every 14

This work was supported in part by grants from the Montana Wheat Research and Marketing Committee, Mobay Corporation, and Gustafson, Inc.

Contribution from the Montana Agricultural Experiment Station. Journal Series Paper J-1788.

Accepted for publication 28 February 1986 (submitted for electronic processing).

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days beginning 40 days after seeding using the 1983 foliar symptom scale. All plots were end-trimmed to 2.6 m before harvest.

Naturally infested plot. A commercial field near Toston, MT, naturally infested with *G. g.* var. *tritici* was selected for a large-scale test. Triadimenol was atomized onto Westbred 906R spring wheat seed at a rate of 0.22 ml a.i./kg. This treatment was compared with seed of the same cultivar treated with carboxin + thiram (1.95 ml of formulation per kilogram of seed). These two treatments were seeded in paired plots, each eight rows wide (15 cm between rows) by 1,200 m long and replicated twice. The test was planted with a commercial grain drill. Ten paired plots 1.2 × 10.7 m were harvested with a small-plot combine from each replicate. Grain weight was determined after drying to 8% moisture at 35 C. The data were analyzed using a standard paired *t* test.

RESULTS

Soil infestation tests. Disease developed in all infested plots, with symptom onset and severity greatly influenced by inoculum level. At the highest inoculum level (5 g/3 m), symptoms were evident both years within 6 wk of seeding in plots planted with untreated seed. In 1983, treatment with triadimenol delayed symptom onset by about 4 wk regardless of inoculum level (Table 1). In 1985, delay in symptom onset was related to fungicide dose and inoculum level, varying from no delay at the lowest fungicide/highest inoculum level treatment to more than a 7-wk delay at the two higher fungicide rates at the 1- and 0.5-g/row inoculum levels. The efficacy of the inoculum (8) was reduced with triadimenol (Table 2). While the highest fungicide rate provided the greatest decrease in inoculum efficacy, the response was nearly the same at the two lowest fungicide rates.

Triadimenol reduced the severity of symptoms on the seminal roots and the SCI during the first 5 wk of growth (Table 3) and delayed aboveground symptom development at Bozeman and Huntley. These reactions to triadimenol resulted in yields higher than those in the carboxin + thiram treatment or the infested-untreated check at both locations.

In 1983, where only one rate of triadimenol was used, the yield for the infested/triadimenol-treated plot was nearly the same as for the uninfested plot (Table 4). Yield was reduced nearly 47% where untreated seed was used in infested plots.

When several rates of triadimenol were used with several inoculum levels in 1985, significant ($P = 0.05$) yield increases (compared with the untreated/infested check) were observed for the 0.29- and 0.22-ml/kg rates but not the 0.15-ml rate at the highest inoculum level (5 g/3 m). At the 1-g inoculum level, all three fungicide rates provided significant yield increases, whereas at the lowest inoculum level, none of the fungicide rates significantly increased yields (Fig. 1).

In all of the above infested plots, the inoculum was placed in the furrow with the seed. To determine if seed treatment with triadimenol is effective against natural inoculum that would be randomly distributed in the soil, a large test was planted in a commercial field that had a history of moderate yield losses to take-all. In this test, triadimenol seed treatment increased yield 23% compared with carboxin + thiram seed treatment (2,892 vs. 2,354 kg/ha) (significant at $P = 0.05$).

DISCUSSION

Chemical seed treatments to control soilborne root rots of plants rarely have been successful because the roots emerge from the zone of protection soon after germination. With the development of systemically translocated compounds such as triadimenol (9), the possibility exists that protection can be extended to plant organs near and above the seed. Reports by Bockus (6) and Dolezal and Jones (7) showed that seed treatments with triadimenol and the related compound triadimefon delay infection by *G. g.* var. *tritici* long enough to provide

Table 1. Delay (days) in onset of foliar symptoms of take-all in Pondera spring wheat in plots planted with seed treated with triadimenol and infested with various levels of *Gaeumannomyces graminis* var. *tritici*

Test year	Fungicide dose (ml a.i./kg)	Delay[x] (days) in onset of foliar symptoms (inoculum rate, g/3 m)			
		5	1	0.5	0.05
1983	0.29	28	···	31	31
1985	0.15	0	13	25	···
	0.22	13	53	53	···
	0.29	13	53	53	···

[x] Symptom delay compared with symptom development in infested plots planted with untreated seed.

Table 2. Effect of triadimenol seed treatment of Pondera spring wheat on efficacy of take-all inoculum

Test year	Treatment	Fungicide dose (ml a.i./kg)	$R^a \times 10^3$ (inoculum rate, g/3 m)			
			5	1	0.5	0.05
1983	Untreated	···	52.0	···	2.4	0.2
	Triadimenol	0.29	22.0	···	1.0	0.1
1985	Untreated	···	56.0	7.4	4.2	···
	Triadimenol	0.15	44.0	5.2	2.8	···
	Triadimenol	0.22	42.0	4.4	2.4	···
	Triadimenol	0.29	36.0	3.8	2.2	···

[a] R = Efficacy of inoculum = $dx/dt\,(1/Q)$, where Q = amount of initial inoculum = g/3 m of row; dt = 98 days in 1983 and 91 days in 1985; dx = disease score on a basis of 0 = healthy to 1.0 = severe disease in 100% of the plants (8).

Table 3. Effect of seed treatment with triadimenol on yield of Pondera spring wheat in 1985 grown in soil infested with take-all oat kernel inoculum (2 g/3-m row)

Treatment	Fungicide dose	Disease score[y]		Yield (kg/ha)	
		Huntley	Bozeman	Huntley	Bozeman
Uninoculated check	···	0.1 a[z]	0.0 a	4,960 a	2,852 a
Inoculated check	···	2.6 c	2.0 c	3,793 c	962 c
Triadimenol	0.22 ml a.i./kg	1.3 b	0.6 b	4,259 b	1,729 b
Carboxin + thiram	1.95 ml form./kg	2.6 c	1.9 c	3,635 c	847 c

[y] Disease score: 0 = no symptoms on roots; 1 = one or two lesions on seminal roots; 2 = three to five lesions on seminal roots but no lesions on subcrown internode (SCI); 3 = six or more lesions on seminal roots, one or two lesions on SCI; 4 = six or more lesions on seminal roots, three to five lesions on SCI; and 5 = complete blackening of SCI.

[z] Means in a column followed by the same letter are not significantly different ($P = 0.05$) using the Student-Newman-Keuls multiple comparison test.

Table 4. Effect of triadimenol seed treatment on yield of Pondera spring wheat planted at Bozeman, MT, in soil infested with varying levels of take-all inoculum in 1983

Treatment	Fungicide dose (ml a.i./kg)	Inoculum rate (g/3 m)	Yield (kg/ha)
Untreated	···	0.5	1,444
Triadimenol	0.29	0.5	2,603
Untreated	···	0.0	2,716
LSD			744

yield increases in winter wheat. Because winter wheat is in the ground as long as 44 wk, a 6- to 8-wk delay in infection may not always be long enough to provide economically significant yield increases. With spring wheat, however, the growing time is greatly compressed such that a 6- to 8-wk delay in disease development should play an important role in reducing yield losses to *G. g.* var. *tritici*. Such was the case in our studies. We observed a delay in onset of aboveground symptoms and subsequent significant yield increases. Averaged across the three fungicide rates, yield increases were 35% for the low inoculum level (0.5 g/3 m), 188% for the intermediate level (1 g/3 m), and 433% for the highest level (5 g/3 m). Of the three triadimenol rates tested, the 0.22- and 0.29-ml a.i./kg rates provided the most consistent and significant yield increases. The 0.15-ml a.i./kg rate probably is too low to provide reliable control of *G. g.* var. *tritici*.

Control of *G. g.* var. *tritici* by seed treatment in the infested plots where the inoculum was near the seed is perhaps not too surprising. However, in naturally infested fields with randomly distributed inoculum, the possibility of protection provided by treated seed is probably reduced. In our field test, we could not visually detect reduction in the severity of take-all resulting from triadimenol seed treatment, because take-all occurred in randomly distributed patches 1–2 m in diameter (D. E. Mathre, *unpublished*). That triadimenol did provide protection is illustrated by the significant 23% yield increase associated with its use.

Triadimenol appeared to have its effect by inhibiting the development of lesions on the seminal roots and the SCI. Because it is not known to move downward in the plant (9), triadimenol probably provided protection by short-distance diffusion into the soil near the seed and/or by translocation upward into the SCI. That triadimenol could reduce the losses from take-all under neutral to alkaline soil conditions, which are highly favorable to take-all (1), suggests that its performance under conditions less favorable to disease development (i.e., soil pH < 7) might be even better.

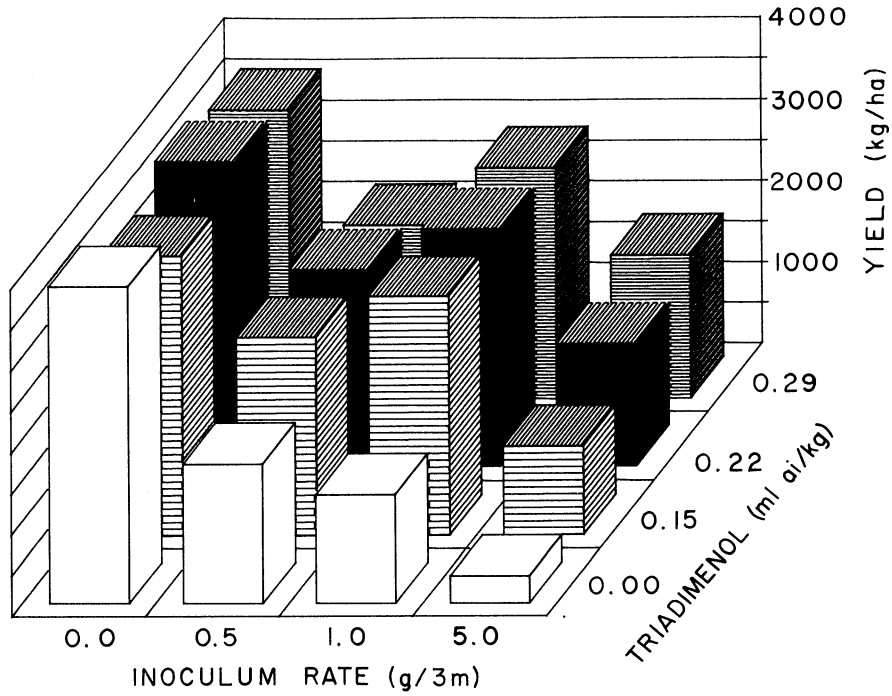

Fig 1. Effects of varying rates of triadimenol seed treatment of Pondera spring wheat at Bozeman, MT, in soil infested with varying levels of take-all inoculum in 1985.

Obviously, seed treatment with triadimenol is not the entire answer for controlling take-all. However, its use may allow a grower to maintain economic production of wheat during the time it takes for natural suppression, i.e., take-all decline, to develop. Furthermore, its use coupled with biological control techniques such as proposed by Weller and Cook (10) might be additive in nature, thus increasing the value of each.

ACKNOWLEDGMENTS

We wish to thank J. Jennings, B. E. Schaff, and W. E. Grey for technical assistance.

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