Control of Ergot in Kentucky Bluegrass Seed Production Using Fungicides

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ABSTRACT

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Foliar applications of sterol demethylation inhibiting fungicides were evaluated for control of ergot (*Claviceps purpurea*) in a Kentucky bluegrass (*Poa pratensis*) seed production field near Post Falls, Idaho, during 1990 and 1991. Flusilazole, propiconazole, tebuconazole, and triadimefon were applied at several rates and plant growth stages. Single applications of all fungicides except triadimefon significantly reduced ergot compared with nontreated controls. Sclerotia and panicle exudate (honeydew) were reduced to zero, or near zero, with single applications of flusilazole alone or with flusilazole, propiconazole, and tebuconazole combined with a wetting agent. Application of fungicides at preanthesis controlled disease more than midanthesis or late-anthesis applications. Seed weight per panicle was reduced when high rates of flusilazole or fungicide–wetting agent combinations were used. Seed germination was not reduced by preanthesis applications except by the highest rates of flusilazole. Most fungicides applied at mid- and late anthesis reduced germination.

Additional keywords: junegrass, meadowgrass, *Sphacelia segetum*

Ergot, caused by *Claviceps purpurea* (Fr.:Fr.) Tul., is currently the most serious disease of Kentucky bluegrass (*Poa pratensis* L.) grown for seed in eastern Washington and northern Idaho. Seed yield reductions of 80–90% have been reported (14), although reductions are usually much less. Preharvest damage from ergot includes the replacement of plant ovaries with fungal tissue (sclerotia), the abortion of damaged ovaries, and the diversion of nutrients to infected florets. During harvest, panicle honeydew reduces equipment efficiency (4). Postharvest losses result from inoculum remaining after harvest which can infect subsequent crops, the cleaning of contaminated seed lots to remove sclerotia for phytosanitary certification, and the cleaning of contaminated screenings destined for livestock.

Chemical and cultural methods have been used to control ergot. Most chemical treatments are used to reduce primary inoculum (ascospores) produced by overwintering sclerotia. Sodium azide, urea, triadimefon, and triadimenol reduce primary inoculum when applied to seed or to sclerotia via soil applications (10,12,17). Incomplete contact of these chemicals with soilborne sclerotia (18) and failure to control primary inoculum originating from nearby crops or grass weeds result in unsatisfactory disease control.

Thermal sanitation (open-field burning) was reported to reduce ergot in grass seed fields, which was a primary reason that field burning became popular in the Pacific Northwest (11). The benefits of field burning are questionable due to the effects of smoke on air quality, and as an effective disease control practice. Alderman (2) observed no difference in the amount of ergot in burned and unburned grass seed fields in Oregon in 1988 and 1989.

The burial of sclerotia has been proposed to control ergot of cereals, because perithecial stroma cannot reach the soil surface to release ascospores (7). This practice obviously does not apply to perennial crops.

Resistance to ergot among Kentucky bluegrass cultivars is incomplete and not always available in desirable turf or seed production types. Presently, there are no cultivars highly resistant to ergot (15).

No highly effective procedures for control of ergot were reported until Cagas (8) reported significant control in bluegrass and other grasses with foliar applications of propiconazole. High levels of ergot control have been reported in buffelgrass (*Cenchrus ciliaris* L.) (9) and in Kentucky bluegrass (6) by using foliar applications of sterol demethylation inhibiting (SDI) fungicides. SDI fungicides are a modern class of systemic fungicides with broad spectrum fungicidal activity against many important pathogens at low rates of application (16). Protection by these compounds is active at the site of infection and can last through most of the flowering period when plants are susceptible to infection by ascospores or conidia of *C. purpurea*.

The purpose of this research was to determine the effectiveness of potential SDI fungicides when they were applied at different growth stages for control of ergot in Kentucky bluegrass seed production.

MATERIALS AND METHODS

Field plots were established in a commercial Kentucky bluegrass cv. Plush seed field near Post Falls, Idaho, in 1990 and 1991. The field was originally seeded in 1985 and had been burned after harvest for residue and disease management each year since 1986. Ergot has occurred in this field every year of seed production (Mark Lonam, Cenex Full Circle, *personal communication*).

Fungicides included for evaluation were flusilazole (Punch, 25 EC), propiconazole (Tilt, 3.6 EC), tebuconazole (Folicur, 3.6 F), and triadimefon (Bayleton, 50 WP) (Tables 1 and 2). In 1991, the wetting agent Penaturf (Chas. H. Lilly Co., Seattle, WA) was included in combination with the lowest rate of application for each fungicide (Table 2).

1990. Individual treatment plots were 3.0 × 6.1 m in a randomized complete-block, split-plot design (date = main plots) with five replications. Fungicides were applied at two rates of active ingredient per hectare on 2 June (preanthesis) and 18 June (late anthesis) (Table 1). Treatments were applied with a CO_2 pressurized boom sprayer at 275 kPa at a 262-L/ha carrier (water) rate. Each plot was rated for disease severity on 3 July by observing the amount of sclerotia and panicle exudate (honeydew), and by feeling panicles by hand for honeydew (tactile). Severity was rated on a 0–9 scale, with 9 representing the most severely diseased plants. On 5 July, approximately 100 panicles were harvested by hand from each plot. The mean weight of clean seed and sclerotia per panicle, the number of sclerotia per panicle, and 1,000-seed weight were determined. Four replicates of 200 seed from each treatment were tested for germination by the Association of Official Seed Analysts methods (3).

1991. Plot layout and experimental design were the same as in 1990. Fungicides were applied at two rates (except for flusilazole, which was applied at three rates) on 5 June (preanthesis) and 16 June (30% anthesis) (Table 2). Triadi-

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mefon was not included in the 1991 trials. Each treatment was replicated four times. Plots were rated for disease severity on 8 July by observing the amount of sclerotia and honeydew on panicles. On 12 July, 20 panicles were harvested by hand from each plot. Yield components and disease parameters were determined as in 1990. Data were analyzed by ANOVA and Fisher's LSD (1).

RESULTS AND DISCUSSION

Cool weather in northern Idaho during late spring of 1990 and 1991 favored ergot development. Consequently, disease was severe in nontreated field plots, and

Table 1. Field disease, yield component, and germination ratings for Kentucky bluegrass seed produced near Post Falls, Idaho, in 1990 following preanthesis and late-anthesis application of fungicides

Growth stage / Treatment	Rate (g. a.i./ha)	Disease ratings[a]			Yield (mg/panicle)		Wt./10^3 seed (mg)[c]	Germination (%)
		Sclerotia	Honeydew	Tactile	Seed[b]	Sclerotia		
Preanthesis								
Flusilazole	3,178	0.2	1.2	1.4	30.9	0.0	273	71.3
Flusilazole	6,430	0.0	1.0	1.2	33.4	0.0	300	69.1
Tebuconazole	252	5.6	7.4	7.8	48.7	7.5	318	85.3
Tebuconazole	504	4.2	5.6	6.0	56.8	1.3	305	83.0
Propiconazole	290	5.2	6.8	6.2	40.4	4.6	314	88.5
Propiconazole	580	3.4	4.6	4.0	53.2	5.9	300	82.1
Triadimefon	615	7.2	7.6	8.0	48.2	9.9	296	84.1
Triadimefon	1,230	6.8	7.6	7.8	45.3	11.5	302	80.3
Nontreated		7.8	7.8	8.6	43.2	11.7	289	83.0
LSD ($P < 0.05$)		1.1	1.0	1.2	NS[d]	4.3	NS	6.4
Late anthesis								
Flusilazole	3,178	5.0	6.6	6.2	50.9	6.5	309	64.4
Flusilazole	6,430	3.0	4.4	3.2	56.4	3.2	309	55.3
Tebuconazole	252	7.4	8.2	8.4	58.5	14.5	285	75.8
Tebuconazole	504	7.2	8.2	9.0	53.4	12.8	295	77.9
Propiconazole	290	7.2	8.2	8.2	62.2	16.1	295	74.0
Propiconazole	580	6.6	8.0	8.6	61.9	15.3	293	70.6
Triadimefon	615	6.4	7.8	8.4	54.7	14.1	300	79.0
Triadimefon	1,230	7.4	8.0	8.4	68.0	16.4	292	76.0
Nontreated		8.0	8.6	8.6	51.8	13.9	298	84.3
LSD ($P < 0.05$)		1.1	1.2	1.1	NS	4.4	NS	5.2

[a] Field disease rated 0 to 9; 0 = no disease.
[b] Yield in mg of clean seed/panicle.
[c] Based on 200 seed.
[d] Not significant.

Table 2. Field disease, yield component, and germination ratings for Kentucky bluegrass seed produced near Post Falls, Idaho, in 1991 following preanthesis and midanthesis application of fungicides

Growth stage / Treatment	Rate (g. a.i./ha)	Disease ratings[a]		Yield (mg/panicle)		Wt./10^3 seed (mg)[c]	Germination (%)
		Sclerotia	Honeydew	Seed[b]	Sclerotia		
Preanthesis							
Flusilazole	1,083	2.8	2.8	47.3	1.3	301	84.5
Flusilazole	2,167	0.5	0.4	21.2	0.1	270	86.0
Flusilazole	3,250	0.2	0.0	19.5	0.1	257	87.6
Flusilazole+Penaturf[d]	1,083	0.0	0.4	25.6	0.0	275	81.0
Tebuconazole	504	6.4	6.5	54.5	7.2	285	83.9
Tebuconazole	1,160	3.5	4.6	50.6	2.7	283	89.6
Tebuconazole+Penaturf	504	0.8	0.5	33.0	0.1	290	85.1
Propiconazole	580	6.4	7.1	40.7	7.9	305	90.8
Propiconazole	1,160	4.9	5.5	59.7	5.2	284	90.0
Propiconazole+Penaturf	580	1.9	1.6	26.2	0.8	276	87.3
Nontreated		8.6	8.0	53.7	16.0	305	83.3
LSD ($P \leq 0.05$)		1.2	1.1	15.9	3.9	NS[e]	6.5
Midanthesis							
Flusilazole	1,083	3.3	3.6	38.7	1.4	294	73.5
Flusilazole	2,167	2.1	2.3	29.6	0.4	275	75.3
Flusilazole	3,250	1.3	1.9	19.5	0.4	284	78.6
Flusilazole+Penaturf	1,083	1.4	2.3	28.8	0.5	276	73.6
Tebuconazole	504	6.9	6.8	54.8	10.4	302	83.1
Tebuconazole	1,160	6.3	6.0	50.4	7.6	310	79.5
Tebuconazole+Penaturf	504	3.1	2.6	18.8	1.4	263	67.6
Propiconazole	580	5.8	6.0	42.7	5.5	318	89.3
Propiconazole	1,160	3.8	4.3	44.4	1.2	297	80.9
Propiconazole+Penaturf	580	2.8	2.3	19.3	0.6	295	75.3
Nontreated		8.1	8.2	65.2	17.6	278	84.4
LSD ($P < 0.05$)		0.9	1.2	16.1	3.9	36	NS

[a] Field disease rated 0 to 9; 0 = no disease.
[b] Yield in mg of clean seed/panicle.
[c] Based on 200 seed.
[d] Wetting agent applied at 50 litres/ha.
[e] Not significant.

distinct differences in control were observed among treatments. In 1990 and 1991, all fungicides were less effective at controlling disease when applied after early anthesis (less than 10% of plants beginning anthesis) (Tables 1–3). Date of application was highly significant for disease expression, according to ANOVA (Table 3). Fungicides were most effective when applied just before anthesis, which is the beginning of the infection period for *C. purpurea* in grasses. This is likely due to the extended activity of the SDI fungicides that protect plants against both primary and secondary infections. This has been observed when using benomyl to control ergot in male-sterile barley (*Hordeum vulgare* L.) (18). In 1990, little control was observed when fungicides were applied postanthesis, except for flusilazole at 6,430 g/ha.

In 1990, seed yield per panicle was reduced more with preanthesis than with postanthesis fungicide applications. This was not observed in 1991 when fungicides were applied at pre- and midanthesis, and was likely due to the shorter interval between application dates. In 1990, flusilazole applied at 3,178 or 6,430 g/ha reduced germination at both application dates. No other treatment reduced germination when applied at preanthesis; however, many treatments reduced germination when applied at late anthesis.

In 1991, all fungicide treatments significantly reduced disease compared to nontreated plots (Table 2). Good control of ergot was observed with flusilazole at 1,083 g/ha, and intermediate control with tebuconazole and propiconazole, without affecting seed yield per panicle or germination. Date of application was not significant for seed or sclerotia weight per panicle, or for 1,000-seed weight (Table 3). Weight per 1,000 seed was not significantly affected by any treatment. Germination was not affected when fungicides were applied at preanthesis. However, germination was reduced by some treatments when applied at midanthesis (Table 2). Because triadimefon did not reduce germination or yield, or provide effective disease control in 1990, it was not included in 1991.

Disease control was significantly greater when fungicides were used in combination with a wetting agent than when they were used alone at the same rate. Brede and Williams (5) and Johnston et al (13) reported only moderate disease control when using wetting agents alone. In the present experiments, fungicide–wetting agent combinations resulted in greater disease control than did the same rate of fungicides or wetting agent alone. All fungicide–wetting agent combinations reduced seed weight per panicle. Brede and Williams (5) also reported some phytotoxicity with high application rates of wetting agents. More testing of combinations of fungicides with wetting agents is needed to insure the combination will not be phytotoxic or reduce yield.

Table 3. Results from ANOVA for field disease, yield components, and germination of Kentucky bluegrass seed grown in 1990 and 1991 analyzed as a split plot

Year / Factor	Disease		Yield		Wt./10^3 seed	Germination (%)
	Sclerotia	Honeydew	Seed	Sclerotia		
1990						
Fungicide	**[a]	**	NS	**	NS	**
Date	**	**	**	**	NS	**
Date × fungicide	**	**	NS	*	**	**
1991						
Fungicide	**	**	**	**	NS	**
Date	**	**	NS	NS	NS	**
Date × fungicide	**	**	NS	NS	NS	NS

[a] ** = Significant at $P \leq 0.01$; * = significant at $P \leq 0.05$; and NS = not significant.

The percentage of sclerotia in uncleaned seed ranged from 0% for the most efficacious treatments to 23% for nontreated plots. Foreign countries place import restrictions on Kentucky bluegrass seed purchased from the United States. Japan, for example, limits the weight of sclerotia in seed shipments to 0.05%. After commercial cleaning, seed from this field still contained over 5% sclerotia (M. Lonam, Cenex Full Circle, *personal communication*), which is 100 times greater than allowed by Japan. Consequently, it is not cost-effective to clean highly contaminated lots to meet stringent export restrictions.

Flusilazole and tebuconazole alone, and all three fungicides combined with a wetting agent, significantly reduced sclerotia and honeydew compared to nontreated plants (Table 2). Applications of propiconazole and tebuconazole significantly reduced sclerotia and resulted in increased (although not significantly) yield compared to nontreated plants. The primary benefit of controlling ergot in Kentucky bluegrass is to reduce seed losses that result from the harvesting of honeydew-covered panicles and the cleaning of seed lots that contain high amounts of sclerotia. Presently, SDI fungicides offer the best potential for chemical control of ergot in Kentucky bluegrass seed production.

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LITERATURE CITED

1. Abacus Concepts. 1989. SuperANOVA. Version 1.11. Abacus Concepts, Berkeley, CA.
2. Alderman, S. C. 1991. Assessment of ergot and blind seed diseases of grasses in the Willamette Valley of Oregon. Plant Dis. 75:1038-1041.
3. Association of Official Seed Analysts. 1988. Rules For Testing Seeds. Vol. 12. Association of Official Seed Analysts, Artcraft Printers, Bozeman, MT.
4. Brede, A. D., Sours, J., and Williams, R. 1990. Varietal differences in ergot resistance among Kentucky bluegrasses. Page 20 in: Proc. West. Reg. Coord. Comm.-11.
5. Brede, A. D., and Williams, R. R. 1991. Control of ergot in Kentucky bluegrass seed production, 1990. Fungic. Nematicide Tests. 46:310.
6. Brede, A. D., Williams, R. R., and Chastain, T. G. 1990. Control of ergot in Kentucky bluegrass seed production, 1989. Fungic. Nematicide Tests. 45:275.
7. Bretag, T. W., and Merriman, P. R. 1981. Effect of burial on survival of sclerotia and production of stromata by *Claviceps purpurea*. Trans. Br. Mycol. Soc. 77:658-660.
8. Cagas, B. 1986. Effectiveness of selected fungicides against the ergot (*Claviceps purpurea* (Fr.) Tul.) in grasses. Sb. UVTIZ Ochr. Rostl. 22:199-205.
9. Craig, J., and Hignight, K. W. 1991. Control of ergot in buffelgrass with triadimefon. Plant Dis. 75:627-629.
10. Hampton, J. G. 1984. Control measures for ergot in the Paspalum (*Paspalum dilatatum* Poir.) seed crop. J. Appl. Seed Prod. 2:32-35.
11. Hardison, J. R. 1976. Fire and flame for plant disease control. Annu. Rev. Phytopathol. 14:355-379.
12. Hardison, J. R. 1977. Chemical control of ergot in field plots of *Lolium perenne*. Plant Dis. Rep. 61:845-848.
13. Johnston, W. J., Schultz, T. R., and Golob, C. T. 1992. Control of ergot in Kentucky bluegrass seed production using wetting agents, 1991. Fungic. Nematicide Tests. 47:299.
14. Meyer, W. A. 1990. Bluegrasses. Page 71-72 in: Status Report Crop Advisory Committee Forage and Turf Grasses. K. H. Asay, chair. Crop Advis. Comm. Forage Turf Grasses.
15. Meyer, W. A., and Funk, C. R. 1989. Progress and benefits to humanity from breeding cool-season grasses for turf. Page 35 in: Contributions from Breeding Forage and Turf Grasses. Crop Sci. Soc. Am. Spec. Pub. 15.
16. Scheinpflug, H., and Kuck, K. H. 1987. Sterol biosynthesis inhibiting piperazine, pyridine, pyrimidine and azole fungicides. Pages 173-204 in: Modern Selective Fungicides. Horst Lyr, ed. Longman Scientific, London.
17. Shaw, S. 1984. Evaluation of triadimenol and other chemical seed treatments for the control of ergot (*Claviceps purpurea*) in contaminated seed. Proc. Br. Crop Prot. Conf. Pests Dis. 1:53-58.
18. Wood, G., and Coley-Smith, J. R. 1980. The effectiveness of fungicides used against *Claviceps purpurea* attacking male-sterile barley in field trials. Ann. Appl. Biol. 96:169-175.