Report of Accomplishments from Research Funded by the Washington Turfgrass Seed Commission Research (2013):

Title: Controlling Ergot in Kentucky Blue Grass

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Abstract:

Ergot is a major seed replacement disease of perennial ryegrass (PRG) and Kentucky Bluegrass (KBG) grown for seed in the U.S. Pacific Northwest. The disease causes yield loss, reduced seed vigor, and difficulties during harvest, cleaning, and seed lot certification. The objectives of this research were to: 1) follow the spread of ergot from a source of inoculum in experimental plots to determine the relative importance of primary and secondary spread and identify spatial patterns of ergot in commercial fields, potentially leading to new cultural and/or chemical management tactics; 2) evaluate the impact of elevation and environment on ergot spore production and disease severity to develop models aimed at predicting spore production and improve the timing of fungicide applications; 3) investigate the potential of soil-applied fungicides to prevent or reduce ergot sclerotia germination and spore production in the field; and 4) quantify the number of sclerotia remaining in the field after harvest, which may provide inoculum in subsequent years and be a potential target for control efforts. Data suggests that ergot spores can travel at least 200 ft from a point source regardless of prevailing wind direction. Although ergot was widespread in PRG fields, clusters of high disease severity were observed in all three fields. For KBG, disease incidence was lower but clusters of high disease severity were observed in one infected field. Spore production began during the last week of April in all locations, consistent with data from previous years showing that airborne ascospores are present in these areas from the beginning of May until mid-June. Fungicide assays in the lab and experimental plots indicate that the soil-applied fungicides pyraclobstrobin, picoxystrobin, azoxystrobin+propiconazole, azoxystrobin, and fluopyram+prothioconazole can reduce the germination of ergot sclerotia, which would prevent the release of ascospores during anthesis and disrupt the ergot disease cycle.

Objective(s):

- **Objective 1.** Follow the spread of ergot from a source of inoculum in experimental plots to determine the relative importance of primary and secondary spread and identify spatial patterns of ergot in commercial fields, potentially leading to new cultural and/or chemical management tactics. Knowledge about the spatial patterns of ergot can be used to improve methods of disease sampling and assessment, identify potential sources of inoculum to target for control, and develop more effective management tactics.
- **Objective 2.** Evaluate the impact of elevation and environment on ergot spore production and disease severity to develop models aimed at predicting ergot spore production and improve the timing of fungicide applications.
- **Objective 3.** Assess the potential of soil-applied fungicides to prevent or reduce ergot sclerotia germination and spore production in the field.
- **Objective 4.** Quantify the number of sclerotia remaining in the field after harvest which may produce inoculum in subsequent years and be a potential target for control efforts.

Procedures:

- **Objective 1.** Two 0.25 acre plots each of KBG (Midnight) and PRG (Top Hat II) were planted in August 2012 at the Hermiston Agricultural Research Station (HAREC), where there is typically a low level of naturally occurring *C. purpurea* ascospores. Each plot was divided into 256 subplots 17 ft^2 in size and spaced 2 ft apart. A point source of inoculum, consisting of 25 germinated sclerotia, was established at the center of each plot when between 10 and 25% of plants were beginning to flower. Slides coated with spore trap grease were placed above each point source and every 9 to 12 feet away in four directions to capture spores produced at the point source. Slides were also placed on each side of the plot to capture any spores originating from outside of the plots. The point source was removed after 4 days and the presence/absence of honeydew was assessed four days later. Ten seed heads were collected from each microplot ten days later. Spores captured on coated slides were stained and counted. Disease incidence and severity was determined by counting the number of ergot sclerotia in each of the ten seed heads.

  Three 125 acre, center-pivot irrigated commercial PRG fields in Umatilla County, OR were surveyed in 2013. Three 125 acre, center-pivot irrigated commercial KBG fields located in Benton County, WA and seven commercial KBG fields located in Union County, OR were also surveyed. The KBG fields in Union County were of various shapes and sizes. Ergot was assessed approximately one week prior to harvest at sample points located along irrigation wheel tracks. Sample points consisted of quadrats 11 ft^2 in size spaced 33 to 98 ft apart. Seed heads were arbitrarily collected from each quadrat for evaluation in the lab. The number of sclerotia was counted in each seed head to determine incidence and severity at each quadrat. Quadrats were mapped using a GPS unit and disease severity in each field was visualized using heat maps, which display relative disease severity as colors. Spatial autocorrelation, or the correlation of disease severity observed in one quadrat compared to nearby quadrats, was

determined using the commonly used spatial autocorrelation index Moran's I. The index of aggregation, I_a, was calculated using the SADIE method to determine the amount of ergot disease clustering in each field. SADIE (Spatial Analysis by Distance IndicEs) conducts spatial analysis of ecological data and is particularly useful for data that is in the form of counts. SADIE I_a values greater than 1 represent non-random clustering of ergot and values less than 1 represent a regular spatial pattern of disease. T he SADIE index v was also calculated, where $v_i > 1.5$ indicates above average clustering (areas of high disease severity), $v_i < -1.5$ values indicate below average gaps (areas of low disease severity), and v_i values between -1.5 and 1.5 indicate a random distribution of disease severity.

- **Objective 2.** Burkard 7-day volumetric spore traps were used to monitor the number of *C. purpurea* ascospores and grass pollen in the air over time in two PRG fields and five KBG fields. The two PRG fields were 125 acres each and located in Umatilla County, OR at elevations between 700 and 800 ft. Three of the KBG fields were located in Benton County, WA at elevations between 900 and 1,250 ft. Two KBG fields were situated in Union County, OR at an elevation of approximately 2,700 ft. Data loggers were placed in each field and recorded air temperature, soil temperature, relative humidity, and dew point on an hourly basis. Spore trap samples were stained and the numbers of spores were counted. Correlation and regression analysis is being used to identify environmental variables that are associated with spore production for use in a predictive model.
- **Objective 3.** Fresh **s**clerotia from PRG and KBG were obtained from seed cleaning facilities in August. A total of four replicate petri plates, each containing 25 sclerotia, were used for each combination of sclerotia type and fungicide treatment and arranged in a randomized complete block (RCB) design. Sclerotia were preconditioned in moist sterile soil at 41°F for six weeks to simulate the winter chilling period required for sclerotia germination. Treatments included the four most efficacious fungicide products as determined in the previous experiment, all of which contained two active ingredients each. When available, the individual active ingredients of these four combination products were also included (Tables 3 and 4). Early (0 days at 61°F), late (14 days at 61°F), and dual applications (0 and 14 days at 61°F) were made for the four fungicide combinations. Early applications were performed for fungicide treatments consisting of a single active ingredient. A single spray of approximately 750 µl (equivalent to 100 gal/acre) was used for each treatment application. The experiment was repeated once. The numbers of germinating sclerotia and capitula (fruiting bodies) were recorded at 2, 3, 4, 5, 6, and 7 weeks in the first trial and at approximately 2, 3, 4, 5, 6, and 8 weeks in the trial repeat. Sclerotia and capitula ratios were calculated by dividing each observation by the mean of the control.

Soil-applied fungicide trials were also conducted in replicated plots located at the HAREC. A 0.10 acre plot was planted with 2-year old KBG cultivar 'Midnight' in August 2012. Rows were divided into plots 3.3 ft long and spaced 3.3 ft apart. Plots were infested with sclerotia from either KBG (100 mg, equivalent to approximately 100 sclerotia) or PRG (100 count) in October 2012. Each treatment was replicated four times and treatments were

arranged in a RCB design. A total of 11 fungicide treatments and a water-treated control were included (Table 5). The fungicide treatments included all four combination chemistries tested in the petri plate assays and their individual components, with the exception of fluxapyroxad which was not available from the manufacturer. Treatments were applied to plots on April 9, 2013 prior to anthesis using a backpack sprayer in a total volume equivalent to approximately 400 g/acre of water. The number of germinating capitula in each treatment was counted every 4 to 6 days and counts were converted to area under capitula production curves (AUCPC), which is analogous to an area under disease progress curve (AUDPC). The mean and maximum number of capitula observed for each treatment during the course of the experiment was also calculated.

- **Objective 4.** Vacuum samples were collected in the PRG fields in Umatilla County, OR and the KBG fields in Union County, OR described in Objective 2. A commercial vacuum sweeper was used to collect sclerotia and seed from 20 plots in each of the three PRG fields. Each PRG plot was 108 ft^2 in size and sampled after postharvest residue was cut and baled. The seven KBG fields were sampled using a handheld commercial vacuum. A total of 20 KBG plots, 11 ft^2 in size, were sampled from each field before propane flaming of postharvest residue. Seed heads were also collected from larger PRG plots (4,300 ft^2) that were swept in 2012 to determine if vacuum collection of sclerotia has the potential to control ergot the following year. Ten seed heads were taken at 30 ft intervals along five transects in each large plot to determine ergot incidence and severity.

Accomplishments:

- **Objective 1.** Spores were not observed on any perimeter slides and only 8 spores were captured on slides placed directly above the inoculum source over a period of four days. The small surface area of the slides and the passive nature of the spore capture method likely limited the ability to capture spores. Nevertheless, honeydew was observed in 91 and 96% of the Kentucky bluegrass microplots. Only one plant with honeydew was observed in PRG plots. Ergot sclerotia were present in 60 and 37% of KBG subplots but disease gradients or patterns were not observed (Fig. 1). Sclerotia were not observed in PRG subplots at any point in the season. However, infection and sclerotia were observed in other KBG experimental plots located in the area. The infested plots at HAREC were the closest and only known sources of ergot inoculum, suggesting that ergot spores can travel a minimum of 200 ft regardless of predominant wind direction.

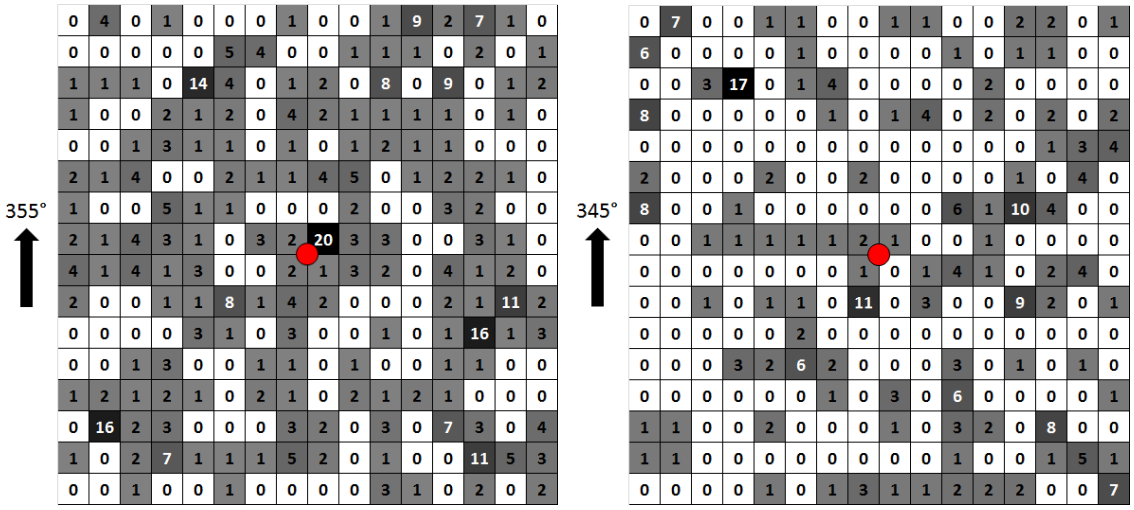

Fig. 1. The number of ergot sclerotia in two Kentucky bluegrass plots after establishing a temporary source of inoculum in the center of the plots (red dot) during early anthesis. Plots were divided into a total of 256 microplots each 17 f^2 in size and spaced 2 ft apart. Ten panicles were collected from each microplot on June 19. Arrows indicate the orientation of the plots, with magnetic North = 0°.

A total of 1,405 and 2,480 quadrats were examined in the three PRG fields and ten KBG fields, respectively. All three PRG fields and four out of ten KBG fields exhibited ergot sclerotia during pre-harvest sampling (Table 1). Significant autocorrelation was observed in all three PRG fields and in three of four infected KBG fields, indicating that ergot severity among neighboring quadrats were similar and the disease was not randomly distributed throughout the field. Significant clusters of high disease severity were observed in all three PRG fields and one KBG field (Table 2, Figs. 2 and 3). Infected wild rye (*Secale cereale*) was observed in roadsides and borders near PRG fields and was flowering as early as May 10 and through the summer and may serve as sources of inoculum.

Table 1. The number of quadrats examined, ergot incidence and the mean number of infected heads and ergot severity in three fields of perennial ryegrass (PRG) and ten fields of Kentucky bluegrass (KBG) grown for seed

				Ergot		
Grass	**County**	**Cultivar**	**No. quadrats**	**Incidence[a]**	**Mean infected heads[b]**	**Mean severity[c]**
PRG	**Umatilla Co., OR**	**Top Hat II**	471	0.79	2.63	4.48
		Pavilion	476	0.63	1.51	2.27
		Provocative	458	0.86	2.92	4.86

KBG	**Benton Co., WA**	**Skye/Prafin**	610	0.00	0.00	0.00
		Cadet	578	0.89	3.22	6.02
		Midnight	579	0.23	0.34	0.43
	Union Co., OR	**Wildhorse**	130	0	0.00	0.00
		Midnight	137	0	0.00	0.00
		Baron	98	0	0.00	0.00
		Right	136	0	0.00	0.00
		K10	41	0.15	0.15	0.15
		Merit	72	0	0.00	0.00
		Baron	99	0.14	0.20	Nd

[a] Proportion of quadrats with at least one infected seed head.

[b] Mean number of infected heads per 10 seed heads.

[c] Mean number of ergot bodies per 10 seed heads.

Table 2. Indices of spatial autocorrelation (Moran's I), aggregation (I_a), patch clusters (v_i) and gap clusters (v_j) in perennial ryegrass (PRG) and Kentucky bluegrass (KBG) grown for seed[a]

Grass	Cultivar	Moran's I (*P*-value)	I_a	v_j	v_i
PRG	**Top Hat II**	$P < 0.0001$	3.809*	-3.825*	3.648*
	Pavilion	$P < 0.0001$	1.505*	-1.415*	1.469*
	Provocative	$P < 0.0001$	2.225*	-2.010*	2.083*
KBG	**Cadet**	$P < 0.0001$	1.388*	-1.320*	1.331*
	Midnight	$P = 0.0034$	1.175	-1.173	1.154
	K10	$P = 0.8551$	0.762	-0.745	0.807
	Baron	$P = 0.0019$	1.321	-1.288	1.491**

[a] A significant ($P \leq 0.05$) Moran's I autocorrelation index indicates that disease severity is highly correlated among nearby quadrats. Index of aggregation I_a values greater than 1 represent clustering and values less than 1 represent a regular spatial pattern. Above average clusters (areas of high disease severity) or below average gaps (areas of low disease severity) of ergot were determined using the index *v*, where $v_i > 1.5$ indicates above average clustering, $v_i < -1.5$ values indicate below average gaps, and *v* values between -1.5 and 1.5 indicate a random spatial distribution.

* Significant at $P \leq 0.05$

** Significant at $P = 0.545$

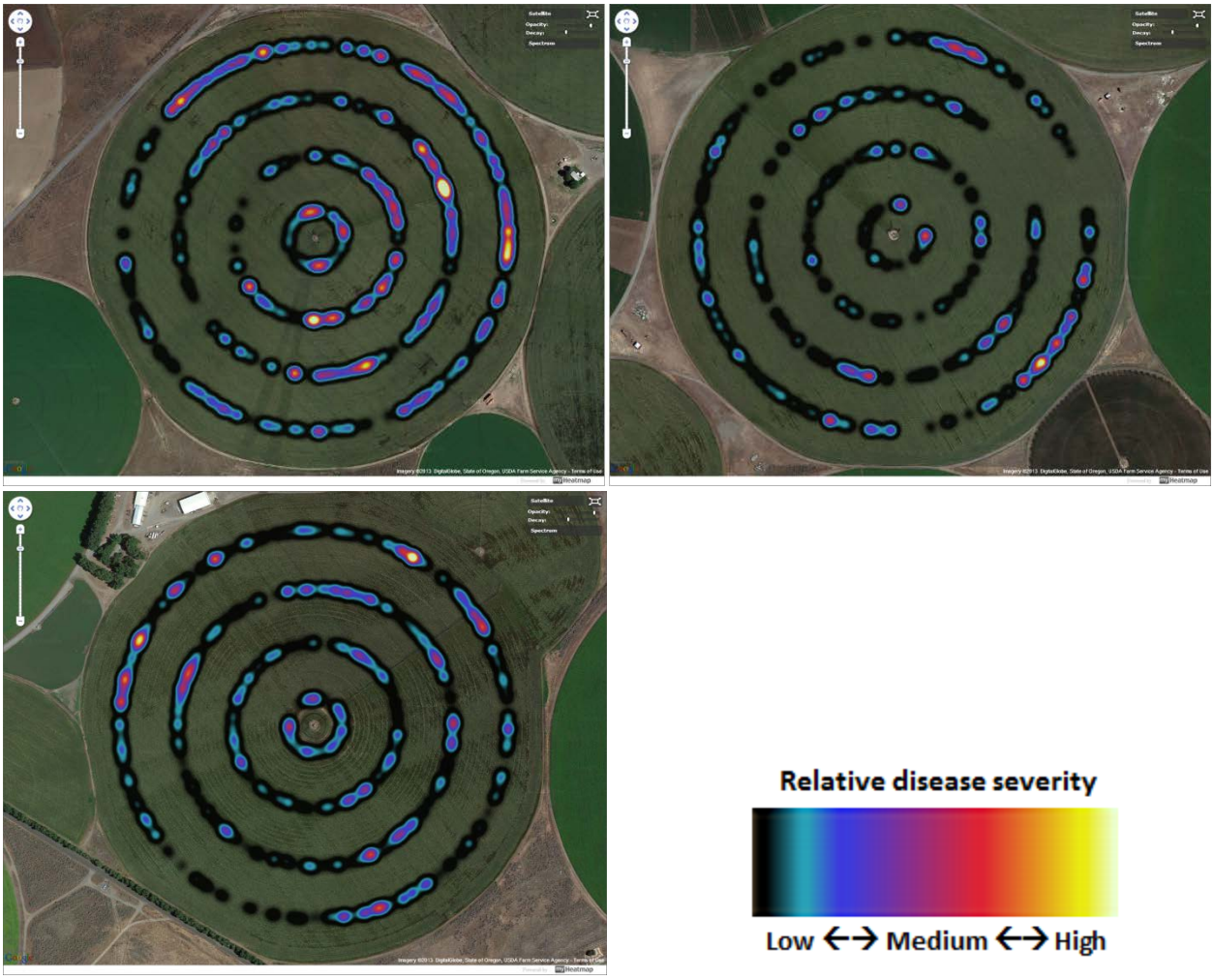

Fig. 2. Spatial distribution of ergot severity in perennial ryegrass fields of Top Hat II (upper left), Pavilion (upper right), and Provocative (lower left) displayed as colors on a heat map. The heat map legend is in lower right.

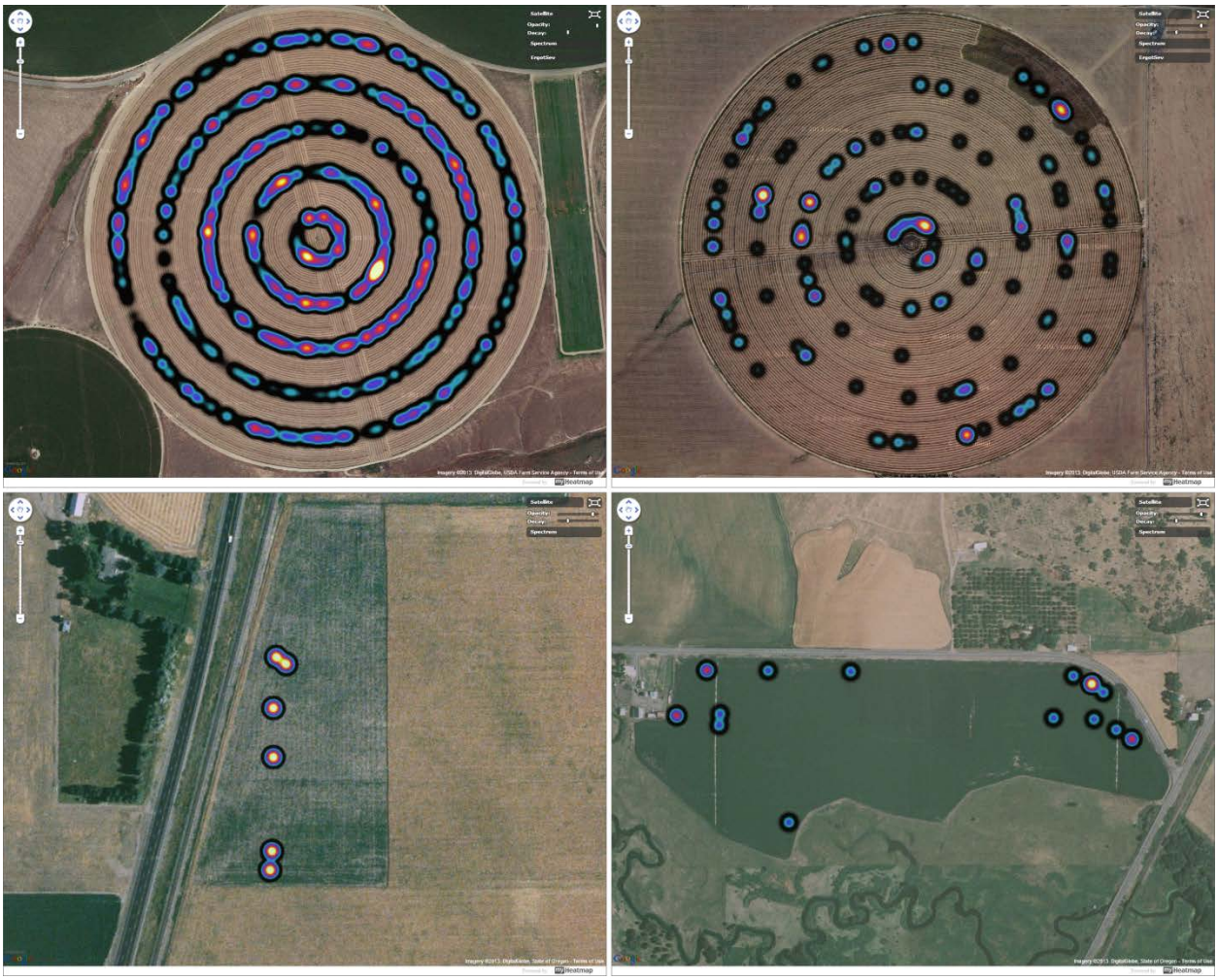

Fig. 3. Spatial distribution of ergot severity in Kentucky bluegrass fields of Cadet (upper left), Midnight (upper right), K10 (lower left), and Baron (lower right) displayed as colors on a heat map. A heat map legend can be found in Fig. 2.

- **Objective 2.** Large numbers of spores (between 56,000 and 114,000) were captured in PRG fields between April 24 and June 19. Fewer spores (178 and 215) were detected in KBG fields in Union County, OR between April 24 and June 23 and even fewer spores (between 7 and 50) were captured in KBG fields in Benton County, WA between April 4 and June 20. The numbers of spores captured by individual spore traps were not necessarily associated with ergot incidence or severity observed in fields. Spore production began between April 24 and April 30 in all three areas. Peak spore production, defined as ≥10 spores captured/day, ceased by June 3 in KBG fields in Union County, OR and ended by June 17 in PRG fields in Umatilla County, OR. Model development is currently in progress.

- **Objective 3.** Early applications of pyraclostrobin, picoxystrobin + cyproconazole, picoxystrobin, azoxystrobin + propiconazole, azoxystrobin, and propiconazole significantly reduced capitula production in KBG in both laboratory trials (Table 3). Late applications of

azoxystrobin + propiconazole and dual (early + late) applications of picoxystrobin + cyproconazole, pyraclostrobin + fluxapyroxad, azoxystrobin + propiconazole, and fluopyram + prothioconazole also significantly reduced capitula production in KBG in both trials. Early, late, and dual applications of picoxystrobin + cyproconazole and azoxystrobin + propiconazole significantly reduced capitula production in PRG in both trials (Table 4). Dual applications of fluopyram + prothioconazole significantly reduced capitula production in PRG, as did early applications of pyraclostrobin, cyproconazole, and azoxystrobin.

Capitula were not observed in any of the field plots infested with ergot sclerotia collected from Kentucky bluegrass. However, capitula were observed in all field plots infested with ergot sclerotia collected from perennial ryegrass. The effect of soil-applied fungicide treatment significantly reduced AUCPC values and the mean and maximum number of capitula produced by ergot sclerotia collected from perennial ryegrass. Pyraclobstrobin, picoxystrobin, azoxystrobin + propiconazole, azoxystrobin, and fluopyram + prothioconazole, all significantly reduced AUCPC values and the mean and maximum number of capitula compared to the water-treated control (Table 5 and Fig. 4). Soil applications of pyraclostrobin + fluxapyroxad, picoxystrobin + cyproconazole, cyproconazole, propiconazole, fluopyram, or prothioconazole did not significantly reduce capitula production. Although not all fungicides significantly reduced capitula production compared to the control treatment, significant differences between fungicides were not observed. These results demonstrate that soil-applied fungicides have the potential to reduce the germination of ergot sclerotia

Table 3. Effect of fungicides on KBG ergot germination ratios in petri plate assays[1]

		Sclerotia ratio[2]		Capitula ratio[2]	
Fungicide	**Timing**	**Trial 1**	**Trial 2**	**Trial 1**	**Trial 2**
Nontreated control	**NA**	1.00	1.00	1.00	1.00
Pyraclostrobin + fluxapyroxad	**Early**	0.26[3]	0.17	0.25[3]	0.08
	Late	0.53	0.67	0.40	0.42
	Early+Late	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Pyraclostrobin	**Early**	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Fluxapyroxad	**Early**	1.00	1.00	1.07	0.83
Picoxystrobin + cyproconazole	**Early**	0.00[3]	0.00[3]	0.00[3]	0.00[3]
	Late	0.21[3]	0.34	0.14[3]	0.25
	Early+Late	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Picoxystrobin	**Early**	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Cyproconazole	**Early**	0.00[3]	0.34	0.00[3]	0.25
Azoxystrobin + propiconazole	**Early**	0.05[3]	0.00[3]	0.04[3]	0.00[3]
	Late	0.05[3]	0.00[3]	0.04[3]	0.00[3]
	Early+Late	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Azoxystrobin	**Early**	0.00[3]	0.00[3]	0.00[3]	0.00[3]
Propiconazole	**Early**	0.05[3]	0.00[3]	0.04[3]	0.00[3]
Fluopyram + prothioconazole	**Early**	0.32[3]	0.17	0.25[3]	0.08
	Late	0.26[3]	0.50	0.18[3]	0.50
	Early+Late	0.16[3]	0.00[3]	0.11[3]	0.00[3]
Fluopyram	**Early**	0.53	0.17	0.50	0.08
Prothioconazole	**Early**	0.63	0.34	0.64	0.50

[1] Early (0 days at 61°F), late (14 days at 61°F) and dual applications (0 and 14 days at 61°F).

[2] Sclerotia and capitula ratios were calculated by dividing each observation by the mean of the control

[3] Significantly different ($P < 0.05$) than the non-treated control using Poisson regression.

Table 4. Effect of fungicides on PRG ergot germination ratios in petri plate assays[1]

		Sclerotia ratio[2]		Capitula ratio[2]	
Fungicide	**Timing**	**Trial 1**	**Trial 2**	**Trial 1**	**Trial 2**
Nontreated control	**NA**	1.00	1.00	1.00	1.00
Pyraclostrobin + fluxapyroxad	**Early**	0.63[3]	0.83	0.59[3]	0.64
	Late	0.87	1.09	0.66	1.00
	Early+Late	0.78	0.66	0.71	0.52
Pyraclostrobin	**Early**	0.68	0.46[3]	0.57[3]	0.37[3]
Fluxapyroxad	**Early**	1.06	0.92	1.13	0.93
Picoxystrobin + cyproconazole	**Early**	0.27[3]	0.20[3]	0.22[3]	0.15[3]
	Late	0.62[3]	0.54	0.50[3]	0.34[3]
	Early+Late	0.15[3]	0.06[3]	0.09[3]	0.03[3]
Picoxystrobin	**Early**	0.66[3]	0.74	0.58[3]	0.69
Cyproconazole	**Early**	0.03[3]	0.29[3]	0.02[3]	0.16[3]
Azoxystrobin + propiconazole	**Early**	0.35[3]	0.31[3]	0.23[3]	0.36[3]
	Late	0.46[3]	0.17[3]	0.31[3]	0.12
	Early+Late	0.27[3]	0.29[3]	0.15[3]	0.20[3]
Azoxystrobin	**Early**	0.55[3]	0.37[3]	0.43[3]	0.34[3]
Propiconazole	**Early**	0.49[3]	0.69	0.42[3]	0.52
Fluopyram + prothioconazole	**Early**	0.66[3]	0.74	0.45[3]	0.53
	Late	0.59[3]	0.83	0.40[3]	0.80
	Early+Late	0.38[3]	0.60	0.21[3]	0.41[3]
Fluopyram	**Early**	0.71	0.91	0.57[3]	0.87
Prothioconazole	**Early**	0.57[3]	0.66	0.47[3]	0.54

[1] Timing of applications included early (0 days at 61°F), late (14 days at 61°F), and dual applications (0 and 14 days at 61°F).

[2] Sclerotia and capitula ratios were calculated by dividing each observation by the mean of the control

[3] Significantly different ($P < 0.05$) than the non-treated control using Poisson regression.

Table 5. Mean area under capitula production curve (AUCPC) values, mean number of capitula, and maximum number of capitula observed in experimental plots infested with ergot sclerotia from perennial ryegrass in October 2012 and treated with soil-applied fungicides in April 2013[a]

Fungicide (product and rate)	AUCPC	Mean capitula	Maximum capitula
Water-treated control (NA[b])	3025 a	69.8 a	133.3 a
Pyraclostrobin + fluxapyroxad (Priaxor at 6 oz/acre)	1955 ab	45.3 ab	102.8 ab
Pyraclostrobin (Headline at 12 oz/acre)	1466 b	34.3 b	63.0 b
Picoxystrobin + cyproconazole (DuPont DPX-PZX74 at 13.7 oz/acre)	1792 ab	41.8 ab	77.3 ab
Picoxystrobin (DuPont DPX-YT669 at 18 oz/acre)	1187 b	27.2 b	61.0 b
Cyproconazole (Alto at 5.5 oz/acre)	1569 ab	36.4 ab	76.3 ab
Azoxystrobin + propiconazole (Quilt Xcel at 14 oz/acre)	1299 b	29.9 b	60.5 b
Azoxystrobin (Abound at 15.5 oz/acre)	1129 b	26.0 b	52.8 b
Propiconazole (Tilt at 8 oz/acre)	1601 ab	37.6 ab	81.3 ab
Fluopyram + prothioconazole (Propulse at 14 oz/acre)	1369 b	31.3 b	69.3 b
Fluopyram (Luna at 5.5 oz/acre)	2092 ab	48.2 ab	87.0 ab
Prothioconazole (Proline at 5.7 oz/acre)	1682 ab	38.3 ab	76.3 ab

[a] Values followed by the same letter are not significantly different using Tukey's test ($P < 0.05$).
[b] NA: Not applicable

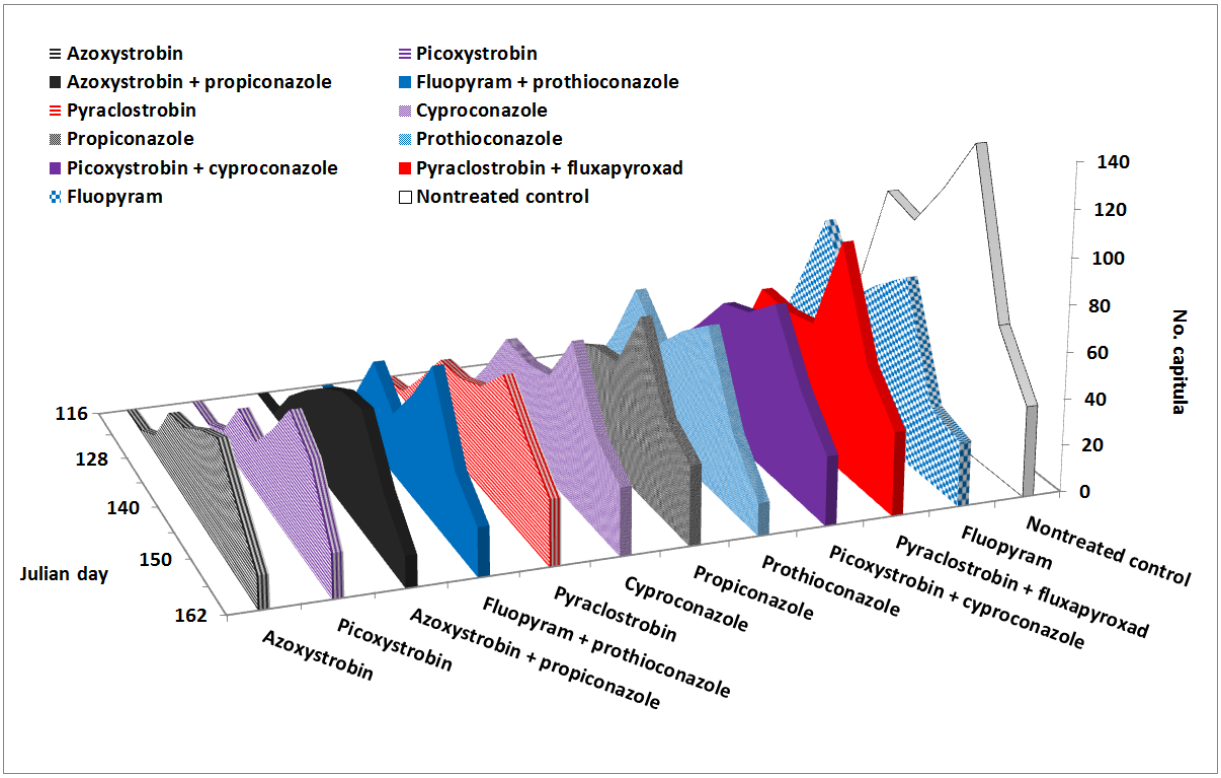

Fig. 4. Number of capitula observed between April 26 and June 11, 2013 in experimental plots infested with ergot sclerotia from perennial ryegrass in the fall and treated with soil-applied fungicides on April 9, 2013.

- **Objective 4.** Vacuum samples collected in August 2013 were pre-cleaned at HAREC. Final processing and analyses are currently being conducted by the seed conditioning lab located at the USDA-ARS National Forage Seed Production Research Center. Data should be available by the end of 2013/beginning of 2014. Unfortunately, most of the seed head samples collected from the larger PRG plots were destroyed by rodents and reliable data could not be obtained.

Impacts:

KBG plots located upwind of the infested plots at HAREC, the only known sources of ergot inoculum, showed signs of ergot infection, suggesting that ergot spores can travel at least 200 ft regardless of prevailing wind direction. Ergot incidence and severity increased in the second year in 2 out of 3 PRG fields and significant clustering of disease severity was observed in all three fields. Significant clustering of high disease severity was observed in one KBG field. The significant clustering observed suggests that ergot severity is not randomly distributed in the field and patches of high disease severity and large numbers of sclerotia occur. Such clustering may be due to secondary spread within and among neighboring plants via honeydew and/or concentrated sources of ascospores from sclerotia located within or outside of the field. Results obtained in 2012 suggest that sclerotia left in fields after harvest are a potential major source of inoculum. Reducing the number of sclerotia and/or sclerotia germination would decrease the

amount of ascospores available during anthesis. However, the contribution of infected weeds, volunteers, or native plants to ergot epidemics via insect transmission also requires further investigation. Infected wild rye was observed flowering in areas surrounding PRG fields as early as May 10, at least 2 weeks before anthesis of PRG. If infected early, wild rye may serve as a source of honeydew for later flowering PRG fields. Additional research is required to determine the impact of infected wild rye and insect vectors with regards to secondary spread of honeydew.

Spore production began during the last week of April in all three locations. The number of spores captured in KBG fields stopped or dropped to negligible levels (<3/day) by the first and third week of June in Benton County, WA and Union County, OR, respectively. Spore levels dropped in PRG fields in Umatilla County, OR to ≤7/day by June 19. These results are consistent with spore trapping data collected in 2008 and 2012 and suggest that airborne ergot ascospores are generally present in these areas from the beginning of May until mid-June. These data will be used to develop a predictive model for ergot ascospore production and help direct and time fungicide applications.

Experiments conducted using petri plate assays in the lab and experimental plots located at HAREC indicate that soil-applied fungicides have the potential to reduce the germination of ergot sclerotia, prevent the release of ascospores during anthesis, and disrupt the ergot disease cycle. Such soil-applied fungicides may be used to reduce the buildup of ergot sclerotia in established fields or to reduce the amount of ergot in neighboring fields that are potential inoculum sources for newly planted fields. Continuation of studies in 2014 will provide additional information required to further develop conclusions and new management strategies.

Relation to Other Research:

The plant pathology program at HAREC has the responsibility of providing information related to identification and management of all diseases found in high value irrigated crops grown in the region. Grass seed is an important component to the crop base of this region and due to the losses this disease causes, a wide range of support from different sources has been obtained (see overall budget listed in proposal), including grower groups/commissions.

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