Effects of Weather Conditions on Ergot in Kentucky Bluegrass in Central Oregon

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Introduction

With warm and dry summers and relatively cool winters, central Oregon has become one of the prime producers for Kentucky bluegrass (*Poa pratensis*) seed, with sales totaling $10.1 million in 2007. Ergot caused by the fungus *Claviceps purpurea* (Fr.:Fr.) Tul. is an important disease infecting Kentucky bluegrass. The pathogen survives in the soil as sclerotia (also known as ergot bodies), which are resistant to extreme weather, and germinate and form perithecia to produce ascospores that infect the grass flowers before they are pollinated. Initially an infected flower produces "honey dew", a droplet of sweet plant sap containing fungal spores called conidia, which can also cause new infections. Later, the ovary of the infected flower is replaced with fungal material that ultimately grows into a sclerotium. Seed loss occurs when infected seeds are replaced by sclerotia. Good seed is also lost during the cleaning and recleaning process that is required for seed with high infection rates, to ensure that the percentage of ergot bodies is below seed certification standards. Previous work reported the detection of this disease in 36, 44, and 62 percent of Kentucky bluegrass fields in central Oregon, with total loss around $113,294, $13,929, and $39,040, in 1991, 1992, and 1993, respectively (Alderman et al. 1998).
Previous studies have shown that properly timed fungicide sprays during the flowering season could significantly reduce this disease (Schultz et al. 1993). However, the relationship between weather conditions and ascospore release or pollination of Kentucky bluegrass has not been well understood. Fungicide sprays may be unnecessary if ascospores are not present during the flowering period of Kentucky bluegrass. The objectives of this study were to investigate 1) the relationship between weather conditions and ascospore release; 2) the relationship between ascospore presence during flowering and infection of Kentucky bluegrass by the pathogen; and 3) compare the efficacy of applying Quilt® (0.62 lb azoxystrobin/gal + 1.04 lb propiconazole/gal) at different growth stages for controlling ergot.

Methods and Materials

1. Two trials were set up in commercial Kentucky bluegrass fields with the same variety VNS KBG with a history of ergot: one in the northern and the other in the southern range of the North Unit Irrigation District (NUID) in central Oregon.
2. Starting from June 10, weather conditions were monitored in both commercial fields using Campbell Scientific weather stations with sensors for soil temperature, soil moisture, air temperature, air relative humidity, leaf wetness, and wind speed and direction. Growth stage of Kentucky bluegrass in the fields was also recorded weekly.
3. A spore trap (model BS02178, Burkard Scientific Ltd., UK) was set up in the north field in mid-June. Ascospore numbers were counted on a daily basis and the beginning and ending dates of pollen release were also determined by counting pollen grains collected on the spore trap.
4. Four fungicide treatments were included—Quilt sprayed at 14 oz/acre: 1) at the onset of anthesis; 2) 5 days after the onset of anthesis; 3) 12 days after the onset of anthesis; and 4) unsprayed control. The treatments were arranged according to a randomized complete block design with four replicates. Plot size was 30 by 40 ft.
5. Two hundred panicles were randomly taken from the center 16 by 16-ft area of each plot prior to harvest to determine ergot infection.
6. Infested flowers (based on the formation of sclerotia) per panicle were counted, and total dry weight of seeds and sclerotia from 200 panicles, dry weight per 1,000 sclerotia, and dry weight of the healthy seeds were determined after air drying.

Results and Discussion

Weather Conditions and Ascospores Trapped

Soil temperature followed a similar pattern in the two fields (Fig. 1). The daily maximum soil temperature was higher in field no. 1 than field no. 2 from June 12 to June 18, lower on June 19 to 21, and they were very similar on other days during the study period. The daily minimum soil temperatures in the two fields were very similar for most nights, and ranged from 9 to 12^{o}C (48 to 54^{o}F) during the study period, except for a dip below 9^{o}C (48^{o}F) on June 22 for both fields. According to the literature, the soil temperature in both fields would be optimal for germination

of sclerotia of *Claviceps purpurea*. However, due to a delay in our set up, no data were available before June 12 and no ascospores were ever observed.

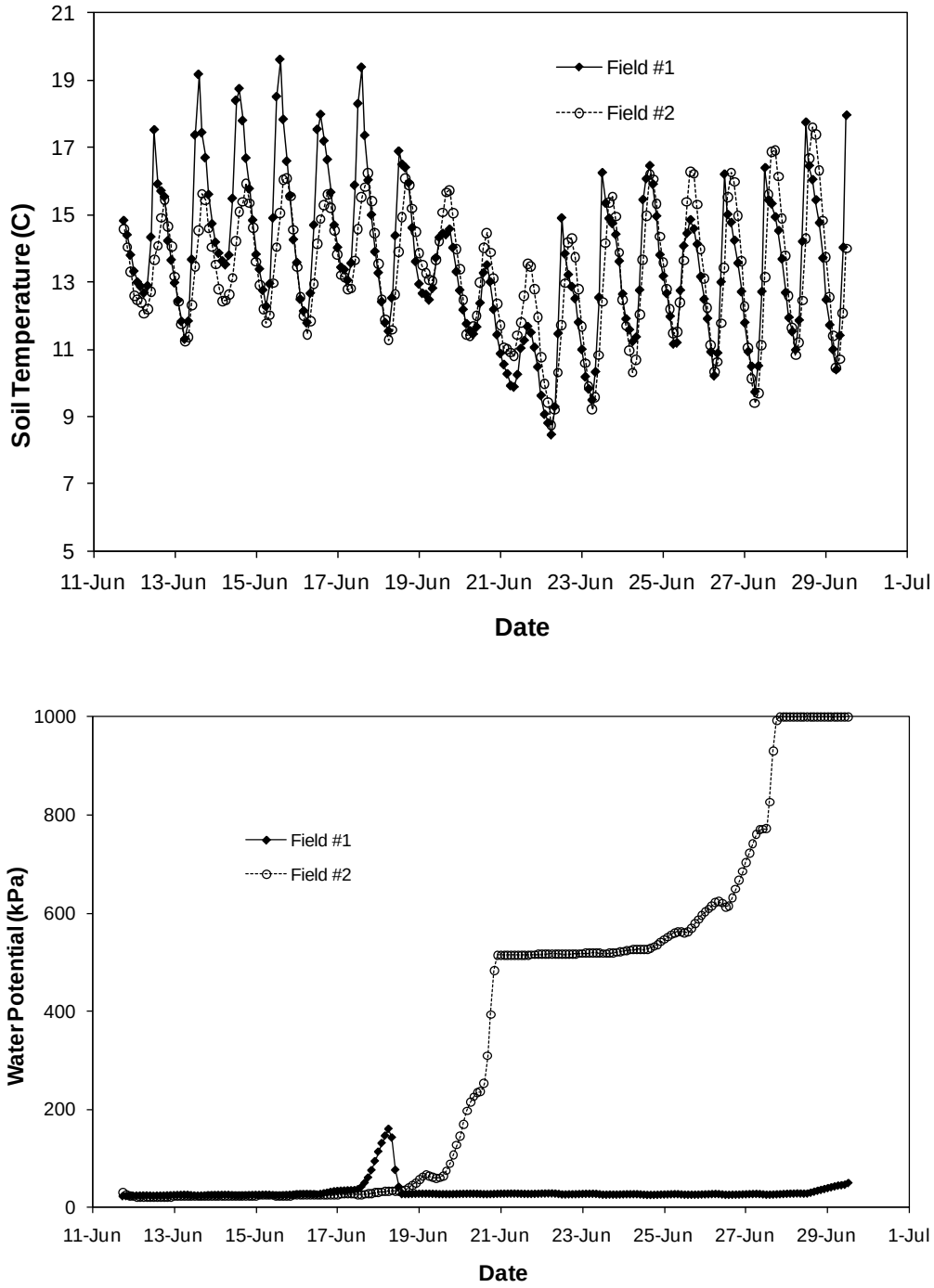

Figure 1. Soil temperature (top) and soil moisture (bottom) measured in the two trial fields during the experiment period. The data were the average of two sensors buried 1-2 inches deep in the soil in each field, Madras, 2009.

Final Disease Incidence of Ergot

In field no. 2 (south part of NUID), the disease level was extremely low, ergot was not found in 14 out of 16 total plots, and incidence of diseased panicles was less than 5 percent in the other 2 plots. In field no. 1 (north part of NUID), incidence of diseased panicles ranged from 0 to 46.5 percent among plots, and percent seeds infected ranged from 0 to 1.24 percent (Table 1). The incidence of diseased panicles was lower in plots sprayed on June 2 or June 7 than in plots sprayed on June 14 or in the unsprayed control (Fig. 2), but the difference was not statistically significant. The standard error was as high as 7.2 percent among unsprayed plots, and 9.9 percent among plots sprayed at the late stage of flowering (Fig. 2).

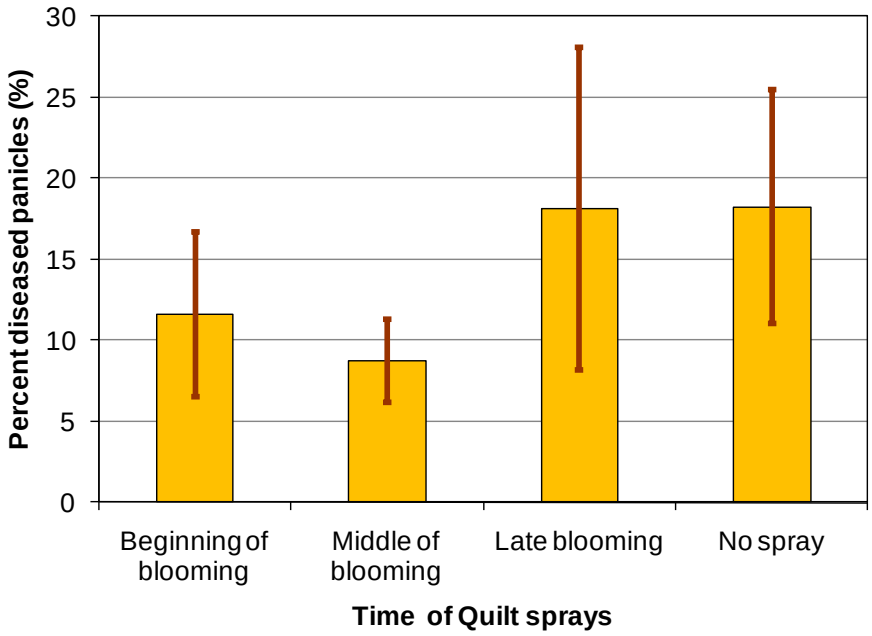

Figure 2. Percentage of Kentucky bluegrass panicles with ergot in plots sprayed with Quilt (0.62 lb azoxystrobin/gal + 1.04 lb propiconazole/gal, at 14 oz/acre) at different times. Each data point is a mean of four replicates and vertical bars represent standard errors. Madras, 2009.

When the data were analyzed further, it was found that the variance-to-mean ratio of the number of diseased panicles was as high as 23.2, suggesting a highly aggregated distribution of diseased panicles (Table 1). More interesting, sclerotia on each panicle also showed a high aggregation pattern in that the expected number of diseased panicles would be much higher if those sclerotia

were distributed randomly. A Chi-square test showed that the probability of a random distribution of infected seeds is less than 10^{-34}.

This is the first year of this project, and due to a delay in setting up the spore trap and weather stations, weather data were not collected before June 11, ascospores were not collected before June 12, and no ascospore release was observed. Therefore, disease level could not be related to ascospore release and flowering, nor could a relationship be determined between ascospore release and weather data. However, from the disease level in plots sprayed at different times, we can deduce that most infections occurred before June 14 because plots sprayed on June 16 showed no difference in disease incidence compared to unsprayed controls.

Table 1. Distribution of diseased Kentucky bluegrass panicles and seeds in plots of field no. 1, Madras, 2009.

Rep	Treat[1]	Seed incidence	Observed diseased panicles	Expected diseased panicles
1	A	0.000%	0	0
1	B	0.021%	3	4
1	C	0.078%	15	18
1	D	0.037%	7	8
2	A	0.099%	15	22
2	B	0.297%	25	60
2	C	0.227%	33	47
2	D	0.140%	25	31
3	A	0.432%	47	81
3	B	0.161%	25	35
3	C	1.240%	93	155
3	D	1.083%	75	146
4	A	0.581%	31	101
4	B	0.119%	17	26
4	C	0.016%	4	3
4	D	0.614%	39	105
Variance to mean ratio			23.2	
P-χ2			<0.00001	

[1]Quilt sprayed at 14 oz/acre: A) at the onset of anthesis; B) 5 days after the onset of anthesis; C) 12 days after the onset of anthesis; and D) unsprayed control.

The results from this year demonstrated that the diseased panicles and individual sclerotia were highly aggregated. The mechanisms causing this aggregation were unclear. At this time we cannot exclude other possible causes such as aggregated micro-environmental conditions that favor germination of sclerotia and infection by ascospores. However, aggregation around the primary inoculum sources (either sporulating perithecia or conidium-containing honeydew) was the most likely explanation, especially for the aggregation of sclerotia on each panicle. If the highly aggregated diseased seeds resulted from secondary spread of the disease via honeydew, it is good indirect evidence of the role of honeydew (conidia) in the disease epidemics. This is an aspect worthy of further investigation in the future.

References

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Schultz, T.R., W.J. Johnston, C.T. Golob, and J.D. Maguire. 1993. Control of Ergot in Kentucky bluegrass seed production using fungicides. Plant Disease 77:685-687.