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

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Introduction

Although severity of ergot caused by the fungus *Claviceps purpurea* (Fr.:Fr.) Tul., in Kentucky bluegrass in the Pacific Northwest has varied from year to year, ergot continues to be one of the most economically important diseases of Kentucky bluegrass grown for seed (Alderman et al. 1998). The pathogen survives as sclerotia in the soil, and germinates to produce ascospores that infect the grass flowers. Plants become resistant to infection shortly after pollination. Seven to 10 days after infection, "honey dew", plant sap containing fungal spores called conidia, oozes from infected ovaries. Conidia can cause new infections. Later, the ovary of the infected flower is replaced with fungal material that ultimately grows into a sclerotium or ergot body. Seed loss occurs when infected seeds are replaced by the pathogen sclerotia. Good seed is also lost during the recleaning process to remove sclerotia to meet seed certification standards. At high ergot levels, grass seed becomes contaminated with ergot sclerotia during harvest, and if not removed during seed cleaning, the seed can be rejected or heavily discounted in value. Management of ergot is based on reducing the primary inoculum source (ascospores from sclerotia) through deep plowing (Bretag and Merriman 1981), residue burning (Johnston et al. 1996), and spraying fungicide to inhibit ascospore production (Wood and Coley-Smith 1980), and by preventing infections through fungicide applications (Schultz et al. 1993; Hamm et al. 2007, 2008; Walenta et al. 2010; Wu et al. 2010). Reduction of the primary inoculum source has proven to reduce infection, but not enough to provide adequate control. Deep plowing to bury sclerotia can be effective in annual crops, but cannot be conducted in established perennial Kentucky bluegrass fields or in areas where exposed soils are prone to wind erosion. High temperatures during residue burning were found to kill ergot bodies near the soil surface (Johnston et al. 1996) but high residue levels are required to provide sufficient heat, preventing the removal of straw. In addition, discussions surrounding the banning of field burning continue to surface every year due to the increasing environmental and human health concerns over the resulting smoke. Spraying fungicides has not adequately inhibited ascospore production in commercial fields (Wood and Coley-Smith 1980), probably due to poor coverage and timing of application, or the use of materials that were not effective.

Spraying fungicide at flowering can provide protection to flowers at a susceptible stage and has significantly reduced ergot infection, but efficacy varies with the timing of the application (Schultz et al. 1993; Hamm et al. 2007, 2008; Walenta et al. 2010; Wu et al. 2010). Multiple applications must often be made over a relatively short time period and still an inadequate level of control is obtained.

Environmental conditions trigger the germination of sclerotia and the release of ascospores. However, the relationship between weather conditions and ergot germination and ascospore release, and that of grass flowering and pollination of Kentucky bluegrass, is not well understood. Previous studies on the release of ascospores revealed that high levels of ergot infection were associated with high numbers of ascospores during the flowering period. Fungicide sprays may be unnecessary if ascospores are absent during the flowering period of Kentucky bluegrass, or could be more focused to protect seed heads if grass flowering and ascospore production are occurring simultaneously. Different environmental conditions from one

year to the next might explain the varying levels of ergot from one year to the next. Simply put, some years of low infection may be the result of peak ascospore production occurring outside of the peak flowering, due to different environmental conditions.

Efforts have been made to time the fungicide sprays according to availability of ascospores and grass flowering. Special spore traps are necessary and constant monitoring of data is required. This knowledge would be very helpful to determine when to begin fungicide applications and how many fungicide applications are needed to provide the most effective disease control. The cost of control could be reduced through fewer fungicide applications, as would the potential environmental impact. The data gained from field and laboratory studies could eventually be used for developing a model to estimate the risk of ergot to aid in decision making.

The objectives of this study were: 1) to monitor ascospore and pollen production in Kentucky bluegrass fields; and 2) to compare efficacy of Quilt® fungicide applied at different times to control ergot infection in Kentucky bluegrass.

Materials and Methods

Two trials were established in commercial Kentucky bluegrass fields with a history of ergot in central Oregon: field A was planted in 2003 with variety SR2100 and was irrigated by a wheel line irrigation system; field B was planted in 2007 with a seed mixture of one-third SR2100, one-third 'Merit', and one-third 'Atlantis', and watered by a pivot irrigation system. Both fields were fertilized and irrigated according to growers' standard, but no fungicide was applied in the plot areas.

Starting from April 1 until harvest, weather conditions were monitored in each field using weather stations with sensors for soil temperature, soil moisture, air temperature, air relative humidity, and wind speed and direction. Growth stage of Kentucky bluegrass in the fields was recorded weekly. A 7-day recording volumetric spore trap (one BS02178 from Burkard Scientific Ltd., UK, and another from Burkard Manufacturing Co Ltd, UK) was setup in each field in mid-May where air is forcibly moved across a sticky tape on a daily basis and ascospores are "trapped" on the tape. Each week tapes were replaced and brought back to the laboratory. Tapes were stained to make ascospores and grass pollens more visible and then ascospores and pollen grains were counted and numbers trapped on a daily basis were calculated.

Four treatments were included in the field trial: Quilt sprayed at 14 oz/acre 1) on June 8; 2) on June 15; 3) both on June 8 and 15; and 4) unsprayed control. The treatments were arranged according to a randomized complete block design with four replicates. Each plot was 20 ft by 30 ft. At harvest, 200 panicles were randomly collected from the center 10-ft by 10-ft area of each plot for determining ergot infection levels. Infested seeds per panicle were counted first. After air drying, shredding and cleaning, total dry seed weight per 200 panicles, number of ergot per 1,000 seeds, weight of total ergot bodies among the 1,000 seeds, and weight of the remaining healthy seeds were determined.

Results and Discussion

Weather Conditions, Ascospores, and Grass Pollens Trapped

According to AgriMet Historical Archive Weather Data, the temperature from winter 2009 to spring 2010 remained low long enough for breaking the dormancy of ergot sclerotia in Madras

(Mitchell and Cooke 1968). The spring of 2010 was wet in general with 24 precipitation days between March 25 and May 19, and the longest no-rain duration of 6 days. Because soil and air temperatures followed a similar pattern in both fields, only data from field B were presented (Figure 1). The daily maximum soil temperature exceeded 59^{o}F from May 14 to May 17 and from June 5 to 13. The daily minimum soil temperatures were often below 45^{o}F during the nights prior to May 7, then increased thereafter except for a dip to 41^{o}F on May 23. According to the literature, the soil temperature in both fields would be optimal for germination of sclerotia of *C. purpurea* (Mitchell and Cooke 1968). Ascospores were detected starting on May 22 in field B, with one peak of spores approximately May 26 and a second peak from June 4 to June 9 (Figure 2). Grass pollen grains were detected beginning in early June with peak pollen trapped approximately June 7 to June 12 in the same field. In field A, release of grass pollens followed a very similar trend as that in field B, but the numbers of ascospores trapped were much lower in field A than in field B (Figure 2). Daily maximum air temperature was generally higher than 68^{o}F after June 4 except for 1 day with daily maximum air temperature below 59^{o}F (Figure 1). Temperatures are presumed optimal for infection of grass flowers by the ergot pathogen (Montes-Garcia et al. 2009).

Final Disease Level of Ergot

In field A, incidence of panicles with ergot bodies showed no significant difference between unsprayed check plots (17.4%) and plots sprayed on June 8 (21.6%) or June 15 (10.8%, Figure 3). The plots sprayed twice had significantly lower incidence (6.4%) than that in the check plots. Incidence in plots sprayed twice was significantly lower than in plots sprayed once on June 8, but did not significantly differ from those sprayed once on June 15. In field B, the incidence of diseased panicles was significantly lower in plots sprayed on June 8 (45.9%) or June 15 (44.3%) than that in the unsprayed check plots (63.6%) (Figure 3). The difference between plots sprayed once on different dates was not statistically significant. In addition, incidence of panicles with ergot bodies in plots with two Quilt spays (26.4%) was significantly lower than that in plots with one Quilt spray (Figure 3). The average number of ergot bodies on each panicle followed a similar pattern to the incidence of diseased panicles, and the fewest ergot bodies per panicle were observed in plots sprayed twice in both fields (data not shown).

Using the variance-to-mean ratio as an index measuring the degree of aggregation (a value >1 suggests aggregation), diseased panicles in a plot were found highly aggregated, or clustered (data not shown), similar to results seen previously (Montes-Garcia et al. 2009). These results once again demonstrated that the ergot sclerotia were aggregated on individual panicles, or when one ergot body was found on a panicle, more ergot bodies were likely to be found on the same panicle. The mechanisms causing this aggregation remain unclear. Possible explanations are localized dispersal of ascospores, secondary spread from infected flowers, or other factors.

The incidence of ergot bodies in seed after cleaning was numerically lowest in plots sprayed twice in both fields (data not shown). In field A, incidence of ergot bodies in the plots with one fungicide application on June 8 was not significantly different from that in the untreated plot, but had significantly higher incidence of ergot infection than plots with one spray on June 15 or two sprays, and the later two treatments did not differ significantly from each other. In field B, incidence of ergot bodies was reduced in all treatments over the untreated plots. However, the levels were not significantly different between treatments with one or two applications of Quilt. The numbers were different from those counted prior to shred, probably due to the loss of ergot bodies during seed cleaning.

The first Quilt spray on June 8, which was aimed to protect the early blooming flowers from ascospore infection, was delayed due to a conflict with the grower's schedule but still significantly reduced the infection in field B. Many ascospores were trapped in that field and final disease incidence in unsprayed plots was high. However, the first fungicide application did not reduce infection in field A, where few ascospores were trapped and final disease incidence in unsprayed plots was low. In a previous study (Montes-Garcia et al. 2009) and this study, Quilt applied at the end of the grass flowering period, when ascospores of the pathogen were absent, significantly reduced final disease levels. The susceptibility of wheat and barley flowers to ergot pathogen declines quickly after pollination, but some flowers remain susceptible to infection for a short period of a few days (Cunfer et al. 1975, Dalington and Mathre 1976). A likely explanation for this would be that the honeydew, which is essentially plant sap containing conidia of the pathogen, may have contributed to secondary spread of ergot, and Quilt applied at the end of flowering period could reduce this spread. This hypothesis needs to be investigated further with well designed experiments in the future.

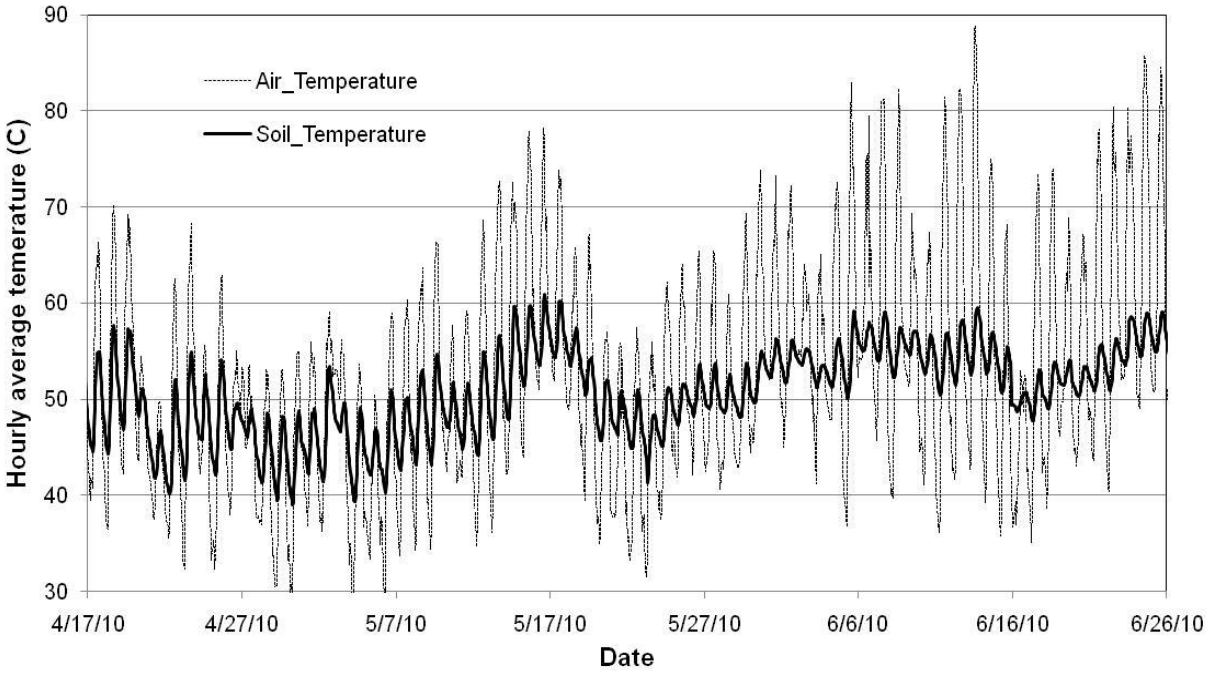

Figure 1. Hourly soil and air temperature averages, each calculated based on data from two sensors in field B. Central Oregon Agricultural Research Center, Madras, OR, 2010.

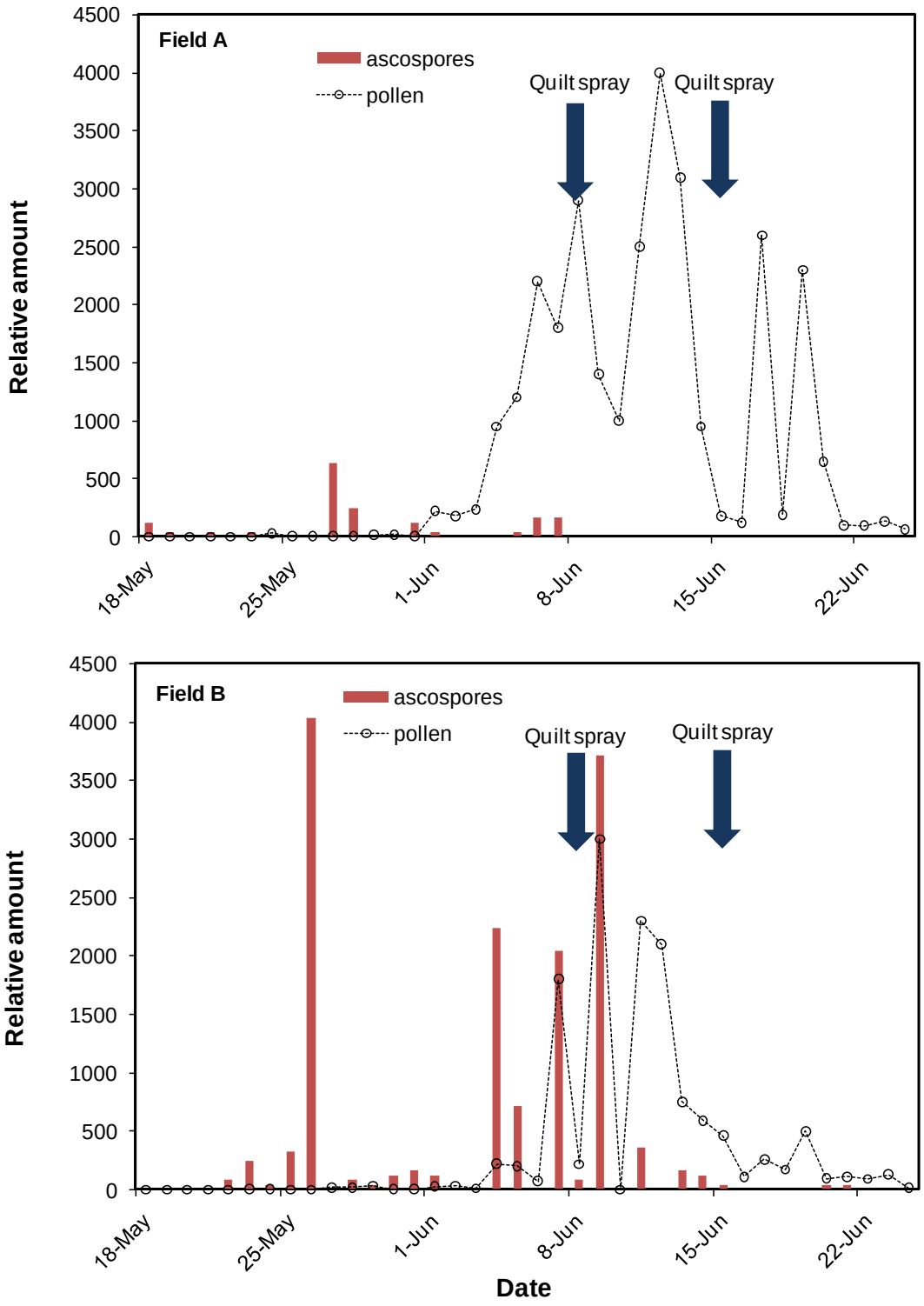

Figure 2. Relative amounts of ascospores and grass pollens trapped by spore traps and dates of Quilt applications for controlling ergot in Kentucky bluegrass, Central Oregon Agricultural Research Center, Madras, OR, 2010.

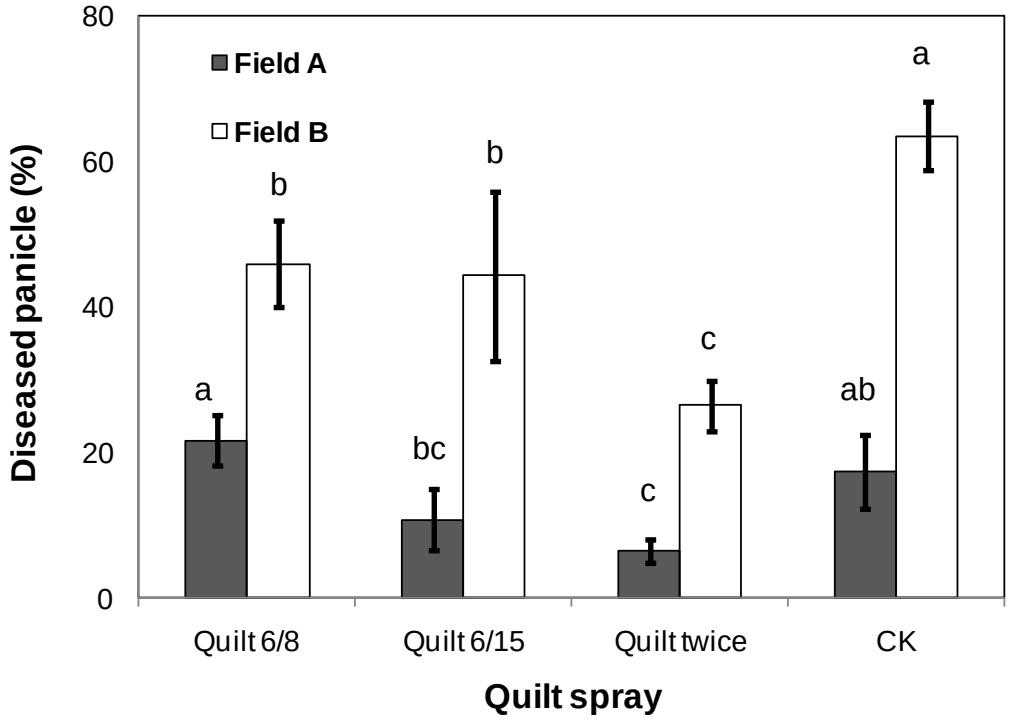

Figure 3. Incidence of panicles with one or more ergot bodies in plots with different Quilt treatments in the two field trials, Central Oregon Agricultural Research Center, Madras, OR, 2010.

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