ERGOT ESCAPE POTENTIAL OF COMMERCIAL CULTIVARS OF PERENNIAL RYEGRASS

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Introduction

Ergot, caused by the fungal pathogen *Claviceps purpurea,* is an important disease of cool-season grass seed crops. It results in yield reduction and hinders seed certification efforts. Toxic alkaloids contained in contaminated bales may impact livestock health. This disease is a persistent problem in many perennial ryegrass (PRG) and Kentucky bluegrass (KBG) growing regions in the Pacific Northwest (Alderman et al., 1996; Alderman et al., 1998). The fungus has a very wide host range in North America, including important grains grown for human and livestock consumption, as well as forage, turf, and weedy grasses (Alderman et al., 2004).

The pathogen infects the unfertilized flowers of grasses and grains and transforms seed into fungal structures called sclerotia. Sclerotia overwinter and germinate in the spring to produce fruiting bodies called capitula, which in turn release millions of airborne ascospores. In addition to ascospores, asexual spores called conidia are produced in large numbers and mix with plant sap exuded from infected ovaries to form a substance referred to as honeydew. Honeydew can serve as secondary inoculum if splash- or insect-dispersed to uninfected flowers during the growing season.

Ascospore release typically coincides with grass flowering (anthesis), which is the only period of host susceptibility. However, Kentucky bluegrass fields for seed production in eastern Oregon have been able to escape the disease in some years because most of the ascospores are released prior to flowering, or because the cultivar may flower before or after peak ascospore release (Menzies and Turkington, 2015).

Limited information exists on the disease escape mechanism in commercial PRG and KBG cultivars grown for seed. In this study, we sought to evaluate PRG cultivars for their potential to escape infection by ergot. We hypothesized that cultivars that flower before or after peak ergot spore production, or those with shortened periods of anthesis, would escape infection and have less disease than do those cultivars that flower when ergot spores are present at high levels.

Materials and Methods

This study was conducted at the Hermiston Agricultural Experiment and Research Center, Hermiston, OR, for 2 consecutive years (April to July of 2014 and 2015). Twelve PRG cultivars ('Applaud II', 'IG2', 'Integra II', 'PST-2M20', 'Silver Dollar', 'Quickstart II', 'Top Hat 2', 'Derby Extreme', 'Esquire', 'Fiesta 4', 'SR 4600', and 'Karma') were evaluated. Cultivars were selected based on turfgrass breeders' recommendations of cultivars that differed in their initiation of anthesis, length of anthesis, and possible ergot resistance level.

Plots (30 feet x 4 feet) were arranged as a randomized complete block design with four replicates. For the 2014 study, experimental plots were established in September 2013, followed by artificial infestation in October 2013. For the 2015 trial, cultivars were planted in September 2014 and infested in October 2014. Plots were infested with 200 sclerotia collected from the seed cleaning facilities that sorted PRG lots harvested in 2013 and 2014, respectively.

A Burkard 7-day recording volumetric spore sampler was used to determine the timing of ascospore release in the PRG plots. The spore sampler was placed in the middle of the plots from April to June in both 2014 and 2015, with the air intake orifice located approximately 2 feet above the soil surface. Spore trap tapes were collected weekly, and each tape was cut into daily segments and stained. The number of *C. purpurea* ascospores was determined microscopically for each hour and then totaled to establish daily counts.

Crop phenology was assessed weekly between May and June in both years to determine the timing and duration of anthesis for each PRG cultivar. Crop phenology was measured using the Feekes scale, whereby the appearance of stigmas and/or anthers is considered the beginning of flowering (stage 10.51) (Cook and Veseth, 1991). Flowering was considered to be completed when at least 90% of the plot reached Feekes stage 11.1 (ripening). The timing and duration of anthesis for each cultivar was recorded to determine when the cultivar is susceptible to infection or has the potential to escape infection.

Once honeydew production was initiated, disease incidence was recorded for 2 weeks, based on the percentage of infected flowers out of 40 flower spikes collected randomly from each plot. Upon maturity, disease incidence was calculated based on the number of seed heads containing ergot sclerotia out of 100 randomly collected seed heads. Disease severity was calculated based on the number of sclerotia present in each infected seed head. Seed heads were swathed and harvested to determine seed yield and the number of sclerotia per 100-gram sample in each treatment. Based on statistical analysis, the 2 years' data could be pooled and statistically analyzed using ANOVA, with multiple comparisons made using Tukey's test.

Results and Discussion

Combined analysis indicated that there was a significant difference in the time of anthesis initiation (start date), anthesis termination (stop date), and anthesis duration among the cultivars evaluated in both years (Table 1). The cultivar 'Quickstart II' initiated flowering earlier in the season in both years than did other cultivars, and its length of anthesis period was significantly extended (Table 1). On average, the flowering period of 'Quickstart II' began on the 127th day of the year (May 7), which coincided with peak ascospore production (1,410 spores). As a consequence, the highest disease incidence was observed in 'Quickstart II' (Table 1). Conversely, the two varieties 'PST-2M20' and 'Derby Extreme' exhibited significantly less disease incidence ($P < 0.0001$), based on the frequency of honeydew production (Table 1). Flowering in 'Derby Extreme' began later in the season (late May), was significantly shorter in duration, and coincided with the least ascospore production (528 spores), likely resulting in disease escape.

A significant negative correlation existed between ergot infection and anthesis initiation date ($P = 0.0021$; $r = -0.3$) (Figure 1), thereby indicating the potential for disease escape in cultivars that tend to initiate flowering later in the season. Other host plant resistance mechanisms (host fertilization status, postpollination stigmatic constriction, etc.) may have played roles in the observed disease incidence, yet were outside the scope of this study. We will continue to evaluate these cultivars in 2016 to gather more data under both field and greenhouse conditions.

Conclusions

It is important to consider the difference between escape and resistance throughout the breeding process (i.e., when selecting new grass cultivars; see Figure 2). Identification of anthesis traits that enable disease escape may aid plant breeders in the development of cultivars that avoid ergot infection. A parallel study evaluating disease escape potential in commercial KBG cultivars was conducted in 2015 at the Central Oregon Agricultural Research Center in Madras, OR. This study will be repeated in 2016. Additional KBG plots were planted in La Grande, OR for evaluation in 2016. Further studies are needed to understand the other mechanisms of host plant resistance not explored in the current study.

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Table 1. Day of the year corresponding to anthesis timing, total spores during anthesis, ergot incidence, and ergot severity for 12 perennial ryegrass cultivars evaluated during 2014–2015.[1]

Cultivar	Anthesis initiation[2]	Anthesis termination[2]	Anthesis duration	Total spores during anthesis	Incidence[3]	Severity[3]
	(date)	(date)	(days)			
Applaud II	131.8 cd	163.1 cd	31.2 abc	972.7 ab	34.3 bcd	4.2
1G2	137.1 abc	166.5 c	29.3 abc	873.0 ab	15.6 ef	2.6
Integra II	132.9 bcd	168.3 bc	35.3 a	1,116.7 ab	20.4 def	4.5
PST-2M20	138.1 abc	174.0 a	35.9 a	805.6 ab	5.4 f	4.5
Silver Dollar	135.1 abcd	164.6 cd	29.5 abc	914.8 ab	41.7 ab	9.9
Quickstart II	126.5 d	160.7 d	34.2 ab	1,410.2 a	65.0 a	7.5
Top Hat 2	140.9 ab	163.7 cd	22.7 c	584.1 b	51.1 ab	2.1
Derby Extreme	142.4 a	172.3 ab	29.9 abc	528.4 b	5.4 f	1.9
Esquire	140.8 abc	168.7 abc	27.8 abc	637.8 b	34.2 bcde	6.3
Fiesta 4	137.6 abc	166.9 bc	29.2 abc	600.2 b	34.3 bcd	4.1
SR 4600	140.7 abc	163.6 cd	22.8 c	571.1 b	47.5 ab	5.6
Karma	141.9 ab	167.5 bc	22.7 c	537.4 b	25.3 cde	2.8
F-value (*P*-value)	5.92 (<0.0001)	5.21 (<0.0001)	8.90 (<0.0001)	2.66 (0.0035)	17.74 (<0.0001)	NS[4]

[1]Means followed by the same letters are not statistically different using Tukey's comparison.
[2]Dates are based on perpetual calendar days or day of the year (e.g., 126 = May 6; 174 = June 23).
[3]Disease incidence (number of infected seed heads) and severity (number of sclerotia) were determined from a random sample of 100 seed heads collected from each plot at harvest.
[4]NS = not significant

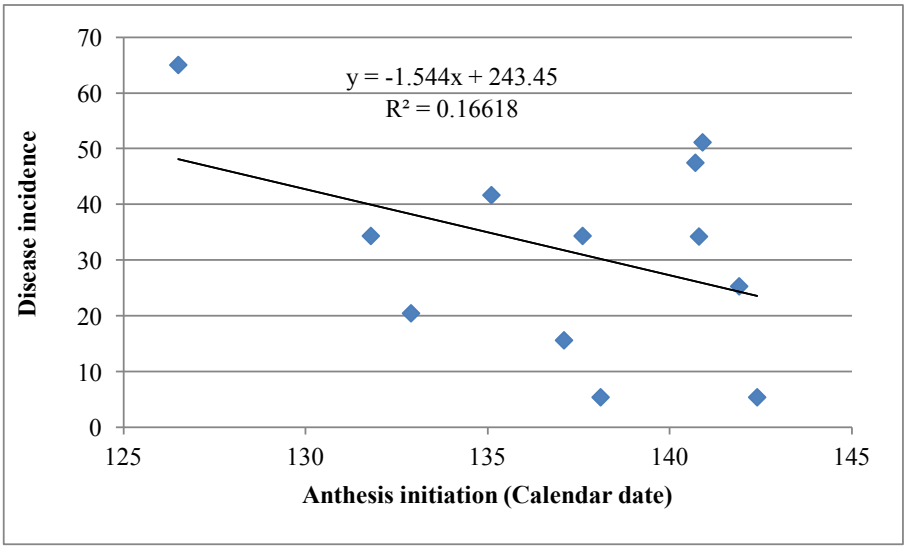

Figure 1. Correlation between observed ergot incidence and anthesis initiation date (r = -0.3; P = 0.0021) in 12 perennial ryegrass cultivars evaluated during 2014 and 2015.

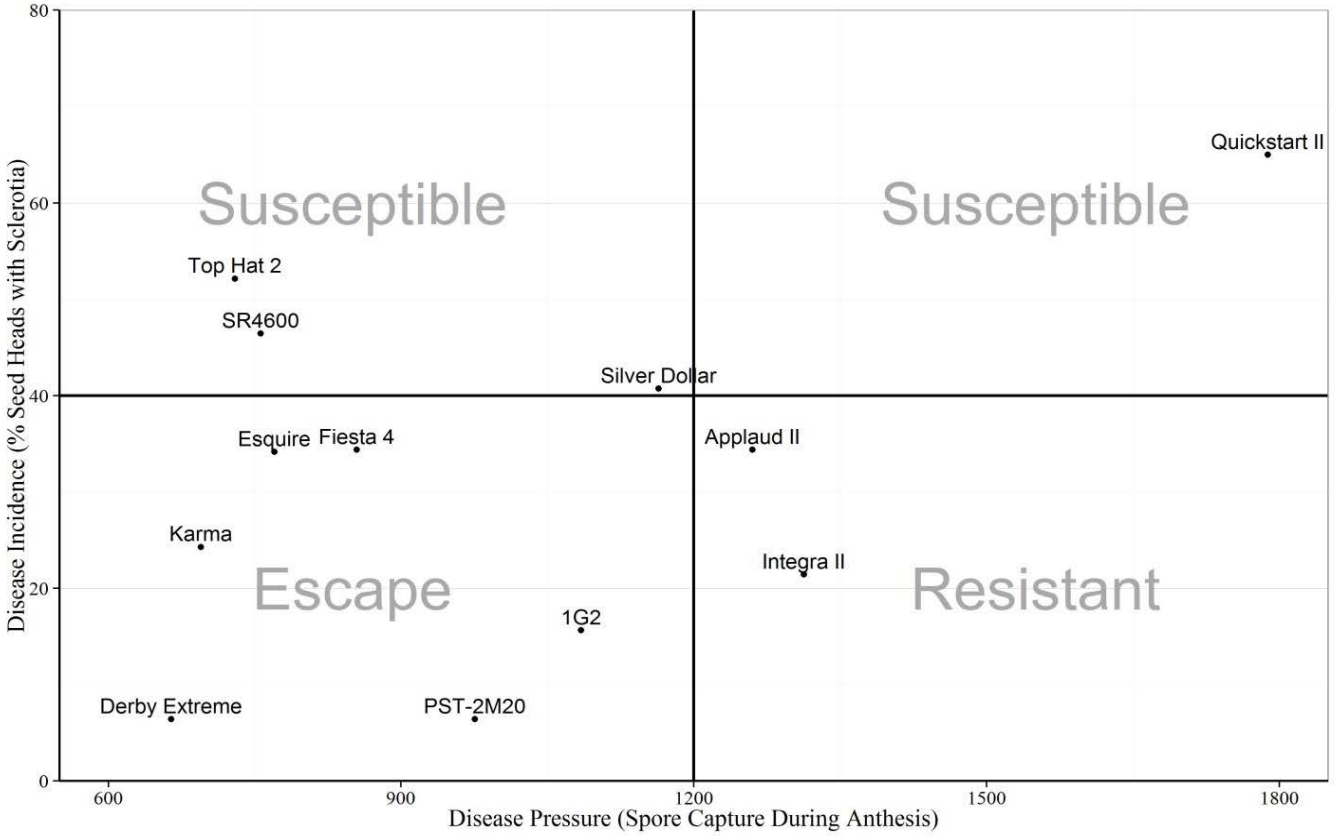

Figure 2. Response of commercial perennial ryegrass cultivars (disease incidence) to disease pressure (number of ascospores present during anthesis), indicating potential disease escape, susceptibility, or resistance.