Occurrence of Gaeumannomyces Patch Disease in Maryland and Growth and Pathogenicity of the Causal Agent

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ABSTRACT

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Gaeumannomyces patch disease of turf caused by *Gaeumannomyces graminis* var. *avenae* (GGA) was observed on Penncross creeping bentgrass turf (*Agrostis palustris*) at four locations in Maryland in 1979 and 1980. Affected turf appeared as bronzed, reddish brown, or light yellow patches 15–60 cm in diameter. Dead or thinning turf was observed in the centers of these patches. Dark brown runner hyphae and simple hyphopodia were present on roots, crowns, and basal sheaths of diseased plants. Mean ascospore lengths for three GGA isolates ranged from 84.5 to 113.2 μm, within the range previously reported for GGA. Most perithecia were borne between leaf sheaths, but some isolates produced perithecia on roots in sandy soil. Pathogenicity tests showed that seedlings of all bentgrass species used as turf are very susceptible to the disease. Exeter colonial bentgrass (*A. tenuis*) seedlings, however, showed a significantly higher level of resistance to the disease than Astoria colonial and Penneagle creeping bentgrasses. The most rapid daily growth rate of four GGA isolates on potato-dextrose agar occurred at 25 C. No growth occurred at 35 C and growth declined significantly ($P = 0.05$) from the optimum temperature. The described symptomatology of the disease and supportive data provide the first documented record of Gaeumannomyces patch of bentgrass turf in the mid-Atlantic region of the United States.

Additional key words: *Agrostis canina*, Kingstown, Ophiobolus patch

Gaeumannomyces patch, formerly known as Ophiobolus patch, is a serious disease of bentgrass (*Agrostis* spp.) turf in many countries (2,6,9,12). The disease is caused by *Gaeumannomyces graminis* (Sacc.) Arx and Olivier var. *avenae* (E. M. Turner) Dennis (GGA) and some isolates of *G. graminis* var. *tritici* Walker (6,7,13,17,19). The pathogen most commonly attacks bentgrasses (*Agrostis* spp.), although *Poa annua* L. and *P. pratensis* L. have also been reported susceptible to the disease (9,14). The disease was first reported in Holland in 1937 on bentgrass turf (10). The widespread presence of the disease in England and other parts of the British Isles was confirmed by Smith (12) in 1951, and in 1961, Gould et al (6) documented the disease in western Washington state. In the United States, the disease was believed to be restricted to the Pacific Northwest; however, in the middle to late 1970s Jackson (8) discovered the disease in Rhode Island and Massachusetts. In 1979 and 1980, the disease was observed on bentgrass turf at four locations in Maryland (5). Gaeumannomyces patch is currently considered a serious disease problem of bentgrass turf in Australia, Europe, and some regions in the United States (9).

We report the symptomatology and distribution of the disease in Maryland. Ascospore measurements, growth rates, virulence among isolates, and relative susceptibility of several bentgrass hosts are also presented. Preliminary results of some of these studies were reported previously (5).

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MATERIALS AND METHODS

Growth curve. All isolates of GGA used in these studies were obtained from field-infected *Agrostis palustris* Huds. 'Penncross' plants. Isolates were obtained from Beltsville, MD (silty loam soil, pH 6.5–7.0), Upper Marlboro, MD (Marlboro Country Club, sandy loam soil, pH 6.9–7.4), Oakland, MD (Village Inn Golf Course, sandy loam soil, pH 6.2–6.5), and Sloccum, RI (Tuckahoe Sod Farm, soil data not collected).

The isolates tested for growth rate and optimum growth temperature were Beltsville, Marlboro, Oakland, and Sloccum. The isolates were grown on Difco potato-dextrose agar (PDA). A 3-mm disk of mycelium and agar was removed with a cork borer from the edge of an actively growing colony. One disk was placed in the center of a sterile petri dish (100 × 15 mm) containing about 20 ml of PDA and dishes were incubated without light at 15, 20, 25, 30, and 35 C ± 1 C. Petri dishes were sealed with parafilm to prevent contamination and allow gas exchange with the atmosphere. Colony diameters were measured every 24 hr for 5 days. The experimental design was a completely randomized block with four replicates per treatment.

Pathogenicity tests. Seed of Penncross and Penneagle creeping bentgrass (*A. palustris*), Exeter and Astoria colonial bentgrass (*A. tenuis* Sibth.), and Kingstown velvet bentgrass (*A. canina* L.) were germinated on moistened paper towels. Twenty 0.5-cm-tall seedlings of each cultivar were planted in each of 10 5-cm plastic pots containing a sandy soil amended with inoculum (1% by weight) of the appropriate isolate. Each GGA isolate was grown in 250-ml flasks on a moistened autoclaved mixture of tall fescue (*Festuca arundinacea* Schreb.) seed and bran (1:1, v/v) for 30 days at 22–24 C. Twenty seedlings of each cultivar planted in 10 pots containing only soil served as uninoculated controls. Soil pH was 6.8, organic matter content was 1.2% and the mechanical analysis was 93% sand, 2% silt, and 5% clay.

The pots, arranged randomly on plastic trays, were maintained under a fluorescent light (155 μE m^{-2} sec^{-1}) in a growth chamber at 25 C for 49 days. The position of the trays was shifted at weekly intervals to minimize chamber effects. The seedlings were irrigated with tap water as needed and were not clipped. Seedling mortality was recorded weekly for 7 wk. During the first four rating periods, all dead seedlings were microscopically examined at ×40 magnification for the presence of diagnostic dark brown runner hyphae of GGA. On the final three rating dates, only randomly selected dead seedlings were examined. Reisolation of GGA was also performed from randomly selected dying seedlings. Data were analyzed as a completely randomized design.

Ascospore measurements. About 80 days after inoculation, perithecia were obtained from diseased plants and crushed under glass coverslips in water on microscope slides to liberate ascospores. One hundred ascospores from at least three but usually five or more mature perithecia were obtained from seedlings of each host-isolate combination. Location of perithecia was noted and

ascospores were measured at ×400 magnification. Data were analyzed by analysis of variance and significant means were separated using the Bayes LSD (BLSD) multiple comparison test (15).

RESULTS

Symptoms and signs. Affected patches of turf ranged from 15 to 60 cm in diameter, but numerous patches coalesced to encompass large areas of turf. Plants on the outer peripheries of the patches appeared bronze and dead plants with rotted roots remained in the centers of the patches at Beltsville and Oakland. At Easton, affected turf was reddish brown, whereas at Upper Marlboro, patches were light yellow. At the latter two locations, only partial thinning of the turf and not death of all plants in the centers of the patches was observed. At the Beltsville site, where weeds were not controlled, dandelions (*Taraxacum officinale* Weber) colonized the dead centers of diseased patches. Typical dark brown runner hyphae and simple hyphopodia were observed on roots, crowns, and basal sheath tissues of diseased plants. Mature perithecia were abundant on naturally infected plants in autumn of 1981 at Oakland.

Distribution. Disease symptoms and signs of the pathogen were observed in 1979, 1980, 1981, and 1982 on Penncross creeping bentgrass, *A. palustris*, in Beltsville; in 1980, 1981, and 1982 in Easton and Oakland; and in 1980 and 1981 in Upper Marlboro, MD. Beltsville and Upper Marlboro are in central Maryland, whereas Oakland is in mountainous western Maryland and Easton is on the Delmarva Peninsula. At each location, the disease appeared in the spring after a fall seeding. During the first year, only a few patches were observed but the number of patches and severity of the disease intensified in subsequent years.

At Beltsville, the soil had been fumigated with methyl bromide before seeding in 1978. Soil fumigation is known to enhance take-all of wheat (*Triticum aestivum* L.) incited by *G. graminis* var. *tritici* (GGT) (18). The affected areas were seeded in 1975 at Easton and Upper Marlboro. Intense symptoms of the disease occurred in the spring of 1981 and 1982 at Easton, and according to the greenskeeper, in the spring of 1979 at Upper Marlboro. In 1980 and 1981, however, symptoms were extremely mild at Upper Marlboro. Disease symptoms appeared during the spring and fall in Oakland but normally appeared only in the spring at the other three sites. The Oakland site, seeded in 1979, is typically cooler and more moist than other areas where the disease is known to occur in Maryland.

Growth curve. There were no significant ($P = 0.05$) differences in fungal growth rate among the four isolates so the data were pooled. No colony growth occurred at 35 C and the temperature optimum for colony growth of the isolates was 25 C (Fig. 1). Significant ($P = 0.05$) differences were obtained for 15, 20, 25, 30, and 35 C.

Pathogenicity and virulence tests. Runner hyphae and aggregations of hyaline infection mats of mycelium and simple hyphopodia were observed on tissues of most of the morbid inoculated seedlings, whereas runner hyphae and hyphopodia were not observed on dead uninoculated control seedlings. GGA was normally isolated from randomly sampled morbid seedlings from each host-isolate combination but never from dead or dying uninoculated control seedlings.

Penncross data were eliminated from the statistical analyses because of high mortality among uninoculated Penncross seedlings. Seedling mortality data for 12 host-isolate combinations are presented in Table 1. In general, the Marlboro isolate was the most virulent, particularly to Kingstown velvet and Exeter colonial bentgrasses. Lowest host-isolate seedling mortality rates were observed among the Kingstown-Beltsville, Exeter-Beltsville, Astoria-Oakland, and Exeter-Oakland combinations.

To better elucidate the relative host susceptibilities to GGA and relative virulence levels among isolates, seedling mortality data among hosts were combined regardless of isolate and seedling mortality data among isolates were combined regardless of host (Figs. 2 and 3). Initially, Penneagle appeared to be the most susceptible cultivar to GGA (Fig. 2). In general, mortality levels of Astoria, Exeter, and Kingstown seedlings remained significantly ($P = 0.05$) below Penneagle during the first 5 wk after inoculation. By the seventh week, however, Exeter seedling mortality levels remained significantly ($P = 0.05$) below Penneagle and Astoria but not Kingstown.

The relative virulence of the three GGA isolates, as measured by mortality of seedlings regardless of host cultivar, showed initially that the Beltsville isolate appeared to be the most virulent (Fig. 3). The mortality level of seedlings inoculated with the Marlboro isolate, however,

Table 1. Mean number of dead seedlings of four bentgrass cultivars 49 days after inoculation with *Gaeumannomyces graminis* var. *avenae*

Bentgrass cultivar	Isolate			Uninoculated control[x]
	Beltsville	Marlboro	Oakland	
Penneagle	11.9 bcde[y]	14.6 ab	12.9 bcd	4.9 h
Kingstown	9.5 ef	16.4 a	12.5 bcd	3.0 hi
Astoria	13.6 abc	13.8 abc	10.9 cdef	5.3 gh
Exeter	10.6 def	14.3 ab	8.0 fg	1.9 i

[x] An uninoculated treatment for each cultivar was used.

[y] Means followed by different letters are statistically different at the 5% level according to the BLSD multiple comparison test. There were 20 seedlings per pot and values are a mean of 200 observations per treatment.

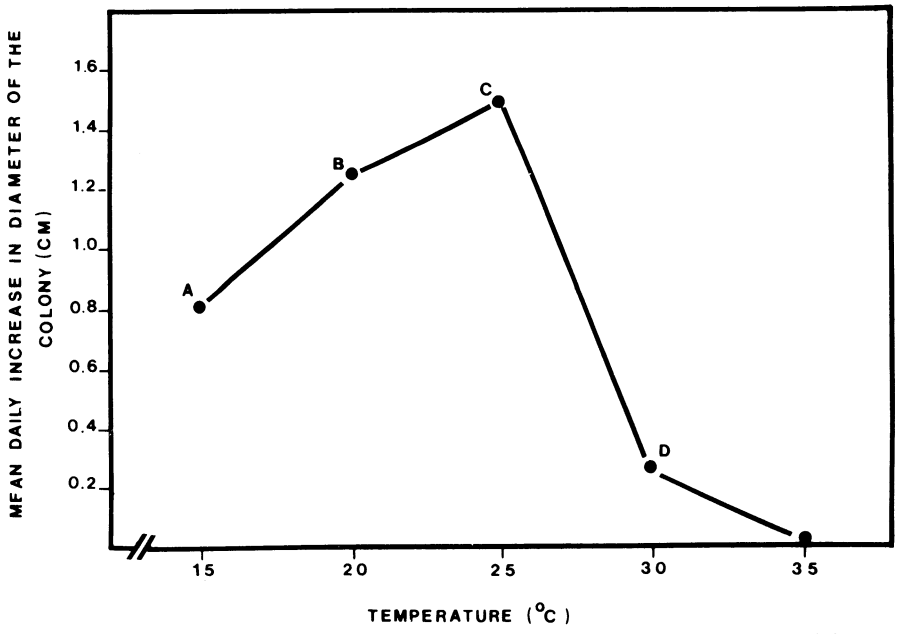

Fig. 1. Mean daily increase in colony diameter of four isolates of *Gaeumannomyces graminis* var. *avenae* grown on PDA at five temperatures. Each point represents the mean of four replications of four isolates measured over a 5-day period. Points marked by uncommon letters are significantly different at the 5% level according to the BLSD multiple comparison test. There was no growth at 35 C.

increased to levels significantly ($P = 0.05$) comparable to the Beltsville isolate during the third through the fifth week after inoculation. By the sixth week of the study, mortality levels among Marlboro-inoculated seedlings significantly surpassed mortality levels of the Beltsville- and Oakland-inoculated seedlings. Seedling mortality rate appeared to plateau about 6 wk after inoculation (Figs. 2 and 3).

Ascospores and perithecia. Ascospore measurements and location of perithecia formation of three GGA isolates on five bentgrass hosts are recorded in Table 2. No perithecia were produced by the Beltsville isolate on Penneagle or Kingstown seedlings. Most perithecia were produced between leaf sheaths (Fig. 4). Perithecia were also borne on roots of several host-isolate combinations, eg, Penneagle-Oakland and Penncross- and Penneagle-Marlboro. The largest numbers of perithecia were produced by the Oakland and Beltsville isolates.

There was considerable variation in length of ascospores among host-isolate combinations (Table 2). Ascospore lengths ranged from 64.7 to 144.7 μm, whereas mean ascospore lengths among isolates ranged from 84.5 to 113.2 μm. Mean ascospore lengths of the Marlboro isolate were longer (107.6 μm) than the Oakland (97.7 μm) and Beltsville (91.7 μm) isolates. Host-isolate combinations exhibiting mean ascospore lengths above 100 μm were Penncross-Oakland and Penncross-, Penneagle-, Exeter- and Astoria-Marlboro. Shortest mean ascospore lengths were obtained from perithecia of the Exeter-Beltsville combination.

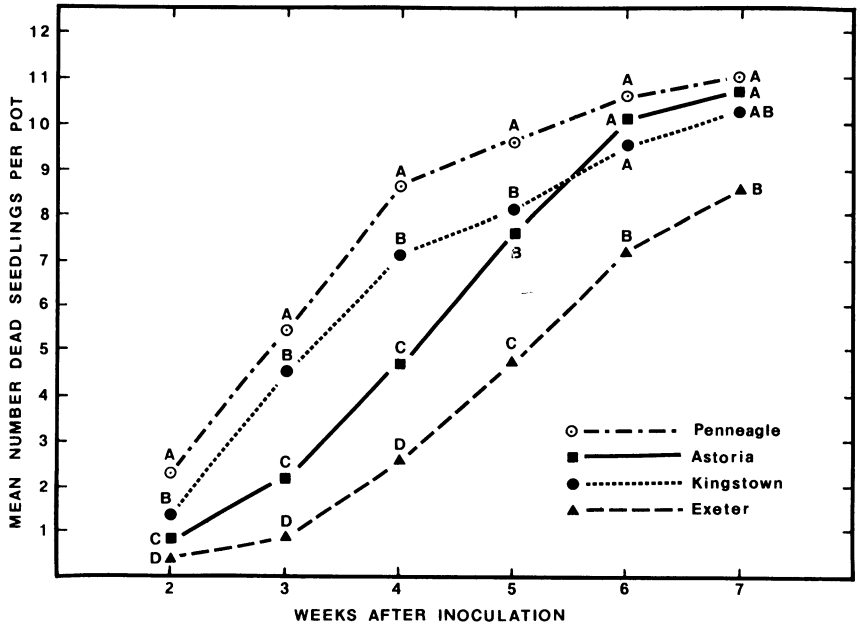

Fig. 2. Susceptibility as measured by seedling mortality of four bentgrass cultivars to *Gaeumannomyces graminis* var. *avenae*. Each point represents the mean number of dead seedlings in pots, separately inoculated with three isolates of *G. graminis* var. *avenae*. There were 10 replications per host-isolate combination, 20 seedlings per pot, and all data for each host were averaged over six recording dates regardless of isolate. Points marked by a common letter on each of the six sampling dates are not significantly different at the 5% level according to the BLSD multiple comparison test.

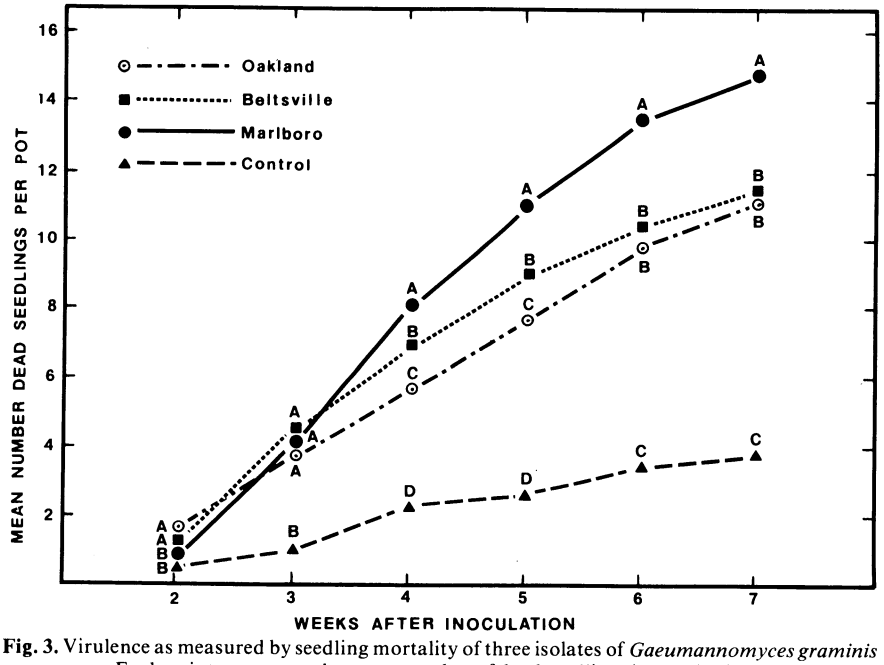

Fig. 3. Virulence as measured by seedling mortality of three isolates of *Gaeumannomyces graminis* var. *avenae*. Each point represents the mean number of dead seedlings in pots individually planted with one of four bentgrass cultivars. There were 10 replications per host-isolate combination and 20 seedlings per pot. Data for each host were averaged over all isolates. For each sampling date, points marked by a common letter are not significantly different at the 5% level according to the BLSD multiple comparison test.

DISCUSSION

Gaeumannomyces patch was observed on bentgrass turf grown in three environmentally diverse regions within Maryland. The bronze coloration of affected turf at the advancing margins of affected patches with plants dying or dead in the centers conforms to the symptomatology of Gaeumannomyces patch in England, western Washington, and New England (6,9,12). Reddish brown and light yellow patches with living or slightly thinning turf in the centers observed at Easton and Upper Marlboro have not been reported previously as symptoms for this disease. The severity of Gaeumannomyces patch declines progressively over a period of 2–4 yr, and disease symptoms eventually disappear (1,3,22). It is conceivable that the pathological condition at these two sites, which first appeared in 1976, represents the decline phase of the disease. Broadleaf weeds colonizing dead areas within patches as noted at the Beltsville site were also observed by Gould et al (6) and Smith (13).

The varieties of *G. graminis* are principally separated on the basis of hyphopodial characteristics, ascospore length, and host range (18). Turner (16) was first to observe varietal differences among *G. graminis* isolates attacking wheat and oats, and the isolates were separated on the basis of host preference and ascospore length. Turner (16) reported that isolates attacking oats had mean ascospore lengths ranging from 101 to 107 μm, whereas wheat-attacking isolates had mean ascospore lengths ranging from 79 to 86 μm. The type specimen of *G. graminis* from wheat as described by von Arx and Olivier (17) had ascospores ranging from 65 to 95 μm. The mean ascospore length range of the three isolates of GGA from Maryland was 68.1–138.2 μm, within the range for GGA isolates from *Agrostis* spp. reported by other researchers. Reported lengths of *G. graminis* ascospores from *Agrostis* spp. hosts include the following: *A. tenuis*

88–124 μm (6), 75–138 μm (13), and 107–130 μm (11); *A. canina* 85–123 μm (13); *A. palustris* 79.8–151.2 μm (9); and *A. stolonifera*, synonym *A. palustris*, 89–120 μm (13).

Most of the ascospores from the various host-isolate combinations possessed a length range below 75 μm (Table 2) and therefore below the shortest length of the ascospore length range reported on bentgrasses by other researchers (6,9, 11,13). However, GGA isolates with ascospore lengths below 75 μm have been reported from other hosts. Dennis (4) reported a GGA isolate attacking wheat to possess ascospore lengths in the range of 65–106 μm. Holden and Ashby (7) reported that a GGT isolate from turf affected with Ophiobolus patch had an ascospore length intermediate to GGT and GGA. Although wide variations in the range of ascospore lengths among GGA isolates exist, *G. graminis* isolates pathogenic to *Agrostis* spp. and with mean ascospore lengths greater than 90 μm probably belong to GGA. Perithecia were observed between leaf sheaths of naturally infected bentgrass plants from Oakland in late autumn 1981. Smith (12) also noted that mature perithecia are produced in nature between leaf sheaths of bentgrass plants late in the year. Weste (20) found that *G. graminis* hyphae require light for induction of perithecia, and this would explain why perithecia are generally produced above the soil line and between leaf sheaths. The Oakland and Marlboro isolates, however, produced mature perithecia on roots of several hosts (Table 2). Willetts (21) also observed formation of GGA perithecia on oat roots in sand culture.

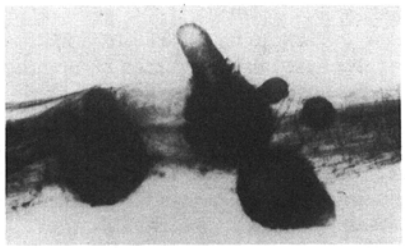

Fig. 4. Perithecia produced by *Gaeumannomyces graminis* var. *avenae* between leaf sheaths of bentgrass.

All four GGA isolates exhibited significantly ($P = 0.05$) more rapid growth at 25 C than at 15, 20, or 30 C (Fig. 1). These results are more or less in agreement with Willetts (21), who reported that six isolates of GGA exhibited optimum growth between 21.5 and 24.0 C. Other varieties of *G. graminis*, however, show maximum growth at temperatures other than 25 C (18,20).

These studies have shown that seedlings of all three species of bentgrass used as turf are very susceptible to Gaeumannomyces patch. Although Exeter colonial bentgrass was slightly resistant to the disease, the cultivar may still be judged as being quite susceptible to Gaeumannomyces patch. There are no other known studies that have compared the susceptibilities of contemporary bentgrass cultivars to this disease. Field observations reported by Smith (14) have also indicated that all bentgrass species used as turf are susceptible to this disease, particularly New Zealand and Oregon colonial bentgrass.

Data revealed that the virulence among GGA isolates varied significantly ($P = 0.05$) (Fig. 3). The Marlboro isolate was shown to be the most virulent; however, this isolate was obtained from a putting green turf showing mild symptoms of the disease.

The disease symptoms and signs of the pathogen described in this report, coupled with supporting pathogenicity tests and acquisition of ascospore lengths in the range of GGA, confirm the presence of Gaeumannomyces patch in Maryland. This constitutes the second report of this disease in the eastern United States and is the first report of its occurrence in the mid-Atlantic states. The sudden appearance of this disease in the eastern United States is difficult to explain. Jackson (8,9) suggested that the pathogen may have caused mild chronic disease symptoms in the eastern United States but that the disease had been either misdiagnosed or dismissed as a cultural problem. These recent and often severe outbreaks of the disease, according to Jackson (9), may also be attributed to the introduction or development of more virulent biotypes of the pathogen or a decline of naturally occurring microorganisms that antagonize and suppress GGA. It is also possible that the widespread use of sterilized and/or high sand content greens mixes in the last 10 yr has contributed to the increased frequency of the disease.

Table 2. Location of perithecia and mean ascospore lengths of three isolates of *Gaeumannomyces graminis* var. *avenae* produced on four bentgrass cultivars

Bentgrass cultivar	Isolate ascospore measurements (μm)		
	Oakland	**Marlboro**	**Beltsville**
Penncross			
Location of perithecia	Between leaf sheaths	On roots, adjacent to crown	Between leaf sheaths
Range	78.3–132.8	86.8–136.2	71.5–107.2
Mean[a]	101.2	113.2	97.2
Penneagle			
Location of perithecia	On roots	On roots, adjacent to crown	Produced no perithecia
Range	71.5–119.1	71.5–144.7	
Mean[a]	95.4	102.3	
Kingstown			
Location of perithecia	Between leaf sheaths	Between leaf sheaths	Produced no perithecia
Range	69.8–125.9	73.2–134.5	
Mean[a]	99.0	99.0	
Exeter			
Location of perithecia	Between leaf sheaths	Between leaf sheaths	Between leaf sheaths
Range	64.7–108.9	68.1–134.5	69.8–107.2
Mean[a]	94.8	108.6	84.5
Overall mean range	70.1–122.9	74.9–138.2	68.1–111.2
Overall mean	97.7[b]	107.6[c]	91.7[d]

[a] Mean length of 100 ascospores from at least three perithecia from each host isolate combination.
[b] Mean length of 500 ascospores from 39 perithecia of the Oakland isolate.
[c] Mean length of 500 ascospores from 25 perithecia of the Marlboro isolate.
[d] Mean length of 300 ascospores from 12 perithecia of the Beltsville isolate.

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