Ophiosphaerella herpotricha, a Cause of Spring Dead Spot of Bermudagrass in Kansas

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ABSTRACT

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Ophiosphaerella herpotricha was consistently isolated from stolons and roots of bermudagrass (*Cynodon dactylon*) affected with spring dead spot (SDS) in Kansas. The fungus was sterile in culture but occasionally produced fertile pseudothecia on inoculated plants. Optimal growth rates on potato-dextrose agar occurred between 20 and 25 C. In greenhouse tests, inoculation with *O. herpotricha* significantly increased root discoloration ratings and, in most experiments, decreased root weights of bermudagrass. The fungus colonized root tissue at soil temperatures of 15 and 25 C. In field tests, symptoms of SDS were reproduced on 18 clones or cultivars 1-2 yr after inoculation with *O. herpotricha*.

Additional keywords: *Leptosphaeria korrae*, *Gaeumannomyces incrustans*, *Gaeumannomyces graminis* var. *graminis*

Spring dead spot (SDS) is a serious root disease of bermudagrass (*Cynodon dactylon* (L.) Pers.) along the northern range of its adaptation in the United States (1,3) and in Australia (12). SDS results in the formation of circular or arc-shaped patches of dead turf in early spring as bermudagrass breaks dormancy. During the summer, bermudagrass recolonizes areas where the turfgrass was killed by SDS, but dead patches often reappear in the same location the following spring. The disease tends to be associated with cold winters and with management factors that reduce hardiness, delay fall dormancy, or promote early spring growth (7).

In 1965, Smith (11) reported *Ophiobolus herpotrichus* (Fr.) Sacc. as a cause of SDS in Australia. The fungus was later determined to be misidentified and was renamed *Leptosphaeria korrae* Walker & Smith (18). A second, more common fungus also associated with SDS in Australia was named *L. narmari* Walker & Smith (18). *L. korrae* has been identified as a cause of SDS in California (3) and Maryland (1) and is also responsible for necrotic ring spot, a patch disease of Kentucky bluegrass (*Poa pratensis* L.) in the United States (10,20).

Previous research in Kansas showed that symptoms of SDS could be produced in the field 2 yr after plugging diseased sod into symptomless bermudagrass (7), but the cause of the disease was not determined. Since this report and others on the etiology of SDS, we have made numerous attempts to isolate *L. korrae* or *L. narmari* in Kansas. To date, we have not recovered these fungi from any turfgrass affected with a patch disease. However, another ectotrophic fungus, *Ophiosphaerella herpotricha* (Fr.) Walker (= *Ophiobolus herpotrichus*), was consistently isolated from SDS-affected bermudagrass. The purpose of this paper is to report on the pathogenicity of *O. herpotricha* to bermudagrass in greenhouse and field experiments. Preliminary reports on this disease have been published (15,16).

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MATERIALS AND METHODS

Isolations of ectotrophic fungi were attempted from roots and stolons of SDS-affected bermudagrass from 14 locations throughout Kansas. The indirect baiting technique described by Smiley and Craven Fowler (10) was used in early isolations. Diseased bermudagrass roots and stolons were collected from SDS-affected patches, washed in water for 30 min, and finely chopped. A 2-cm layer of the debris was placed in a 2 × 15 cm cylindrical plastic pot (Cone-Tainer Nursery, Canby, OR) containing vermiculite. Wheat (*Triticum aestivum* L.) seed was germinated in the pots, and roots were allowed to grow through the debris. After 10–21 days, plants were removed from pots and the roots were gently washed with water to remove the vermiculite. Root pieces were examined microscopically for the presence of ectotrophic hyphae, then were surface-sterilized in 0.5% NaOCl for 1–3 min, blotted dry, and placed on potato-dextrose agar acidified (2% w/v) with lactic acid (APDA). Ectotrophic hyphae were not observed on wheat roots grown in vermiculite only. In subsequent studies, ectotrophic fungi were isolated directly from diseased roots by the methods described by Worf et al (20). All fungal isolates were maintained on PDA at 25 C under cool-white fluorescent lights (12 hr light/12 hr dark).

Growth rates of four isolates of *O. herpotricha* (mass-mycelium isolates KS3, KS10, and KS11 and ascospore isolate KS27) were compared in culture. A small piece of PDA containing mycelium of each isolate was placed in the center of each of three replicate PDA plates and incubated in darkness at 5-C intervals from 5 to 35 C. Colony diameters were measured daily in two directions on each plate and averaged over a 14-day period to determine mean growth per day.

Inoculum for greenhouse and field experiments was prepared as follows: 150 g of oats (*Avena sativa* L.) and 150 ml of water were placed in 950-ml canning jars and autoclaved on successive days at 121 C for 30 min. After the jars cooled, small pieces of PDA containing *O. herpotricha* were inserted into the jar onto the sterile oats; the jars were recapped and kept at 25 C for 1 mo. Infested oats were then removed from the jar and air-dried. Inoculum of other fungal species was prepared in the same manner. All inoculum was stored at 25 C and used within 2 mo of preparation.

Pathogenicity of *O. herpotricha* to bermudagrass was compared with that of other ectotrophic fungi associated with or known to cause SDS. Arizona com-

mon bermudagrass was seeded (1 g/pot) into 6 × 25 cm plastic pots (McConkey Co., Sumner, WA) containing a steamed soil:perlite:peat mixture (1:1:1, v/v). Plants were inoculated 3 mo after seeding with 2 g of sterile oats or oats infested with isolate KS3 or KS27 of *O. herpotricha*, a Kentucky bluegrass isolate of *L. korrae* (obtained from G. Worf, University of Wisconsin-Madison), or an ascospore isolate of *Gaeumannomyces graminis* (Sacc.) von Arx & Oliver var. *graminis* Walker isolated from SDS-affected bermudagrass in North Carolina (obtained from L. Lucas, North Carolina State University, Raleigh, and identified by J. Walker, Australia). Plants and soil were removed from pots, and inoculum was uniformly inserted into soil surrounding roots and stolons; the turfgrass was then repotted. Pots were placed in the greenhouse in a randomized complete block design with five replicates and were watered and fertilized with Osmocote 14-14-14 (Sierra Chemical Co., Milpitas, CA) as needed. Soil temperatures during the experiment ranged from 21 to 28 C. Plants were incubated for 90 days, then washed with water to remove soil from roots. Roots were rated for discoloration on a 0–5 scale (0 = no discoloration, 1 = 1–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–76%, and 5 = >76% of roots discolored) as an indication of the amount of root rot present, oven-dried for 96 hr at 50 C, and weighed. Before drying, samples of root tissue showing ectotrophic hyphae were removed, surface-sterilized, and placed on APDA as previously described. All fungi used in inoculations were recovered from diseased roots and stolons, and no cross-contamination in the pots was detected.

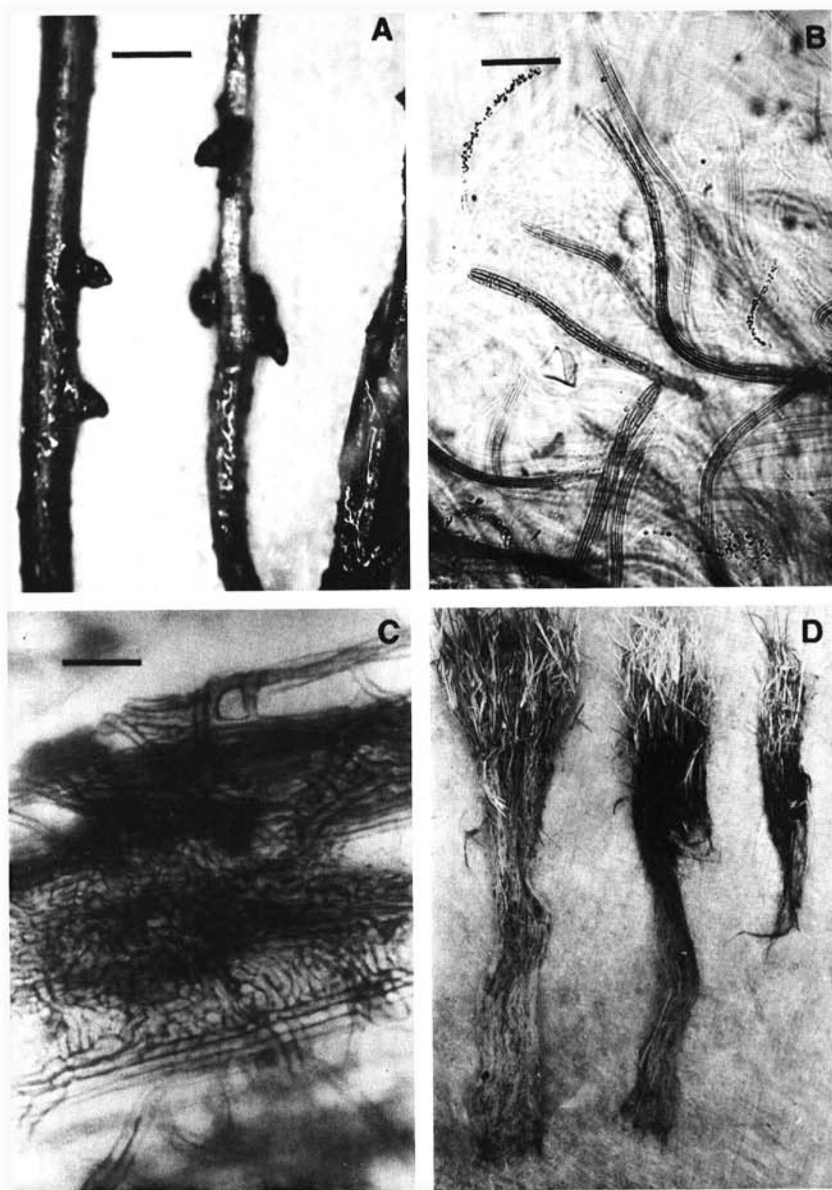

Fig. 1. Symptoms and signs of *Ophiosphaerella herpotricha* on bermudagrass: **(A)** Pseudothecia on stolons. Scale bar = 600 μm. **(B)** Asci and ascospores. Scale bar = 30 μm. **(C)** Ectotrophic hyphae on roots. Scale bar = 20 μm. **(D)** Root discoloration after 90 days on (left) noninoculated Arizona common bermudagrass, (middle) plant inoculated with *Leptosphaeria korrae*, and (right) plant inoculated with *Ophiosphaerella herpotricha*.

The effects of soil temperature and fungal isolates on root discoloration and weight were studied. Stolon pieces, 2–5 cm long, of bermudagrass clone A-22 were surface-sterilized for 1–3 min in 0.5% NaOCl, then rinsed in distilled water. Sample stolon pieces were placed on APDA, but no ectotrophic fungi were recovered. Two stolon pieces were then transplanted into pots containing a U-shaped segment of epoxy-coated copper tubing (0.3 cm i.d.) embedded in the soil. The tube openings remained 3 cm above the soil surface. Plants were placed in the greenhouse for 3 mo, then inoculated with one of three isolates of *O. herpotricha* (KS3, KS11, or KS27) as described previously. Pots were then inserted into holes in a 45-cm-deep polystyrene block. Soil temperatures in half of the pots were maintained at 15 ± 2 C by connecting in series the ends of the copper tubing in each pot with plastic tubing and then circulating refrigerated water through the system according to the method described by Russell (8). The rest of the pots were not connected to the system and maintained soil temperatures of 25 ± 4 C throughout the experiment. Plants were incubated in the greenhouse for 67 days, then processed as previously described. The experimental design was a randomized complete block factorial (temperature × isolate) with five replicates. The same experiment was repeated for clone E-29, except that pots were inoculated with *L. korrae*, *G. g.* var. *graminis*, or isolate KS27 of *O. herpotricha* and the incubation period was 126 days.

A total of three field inoculations of bermudagrass were made in September 1986 and September 1987 at two locations in Kansas. In 1986, a 1-yr-old plot of clone A-29 was inoculated at the Horticultural Research Center in Wichita. The plot received regular irrigation and >3 lb of A nitrogen per growing season throughout the experiment. Forty 3 × 5 cm soil cores were removed with a soil probe at 1.2-m intervals across the turf. Approximately 1 g of oat seed infested with a mixture of *O. herpotricha* isolates KS3, KS11, and KS27 (1:1:1, v/v) was added to each hole and the soil plug was reinserted. For control, 1 g of sterile oat seed was inserted in the bottom of each of 40 additional holes that were offset from inoculation sites by 0.5 m. Plots were rated in the spring of 1987 and 1988 for development of SDS symptoms. The experiment also was conducted in 1986 on an established MidMo (clone S16) cultivar plot near Manhattan. In September 1987, inoculations were made in Wichita on 28 bermudagrass clones or cultivars. Two equally spaced 10-cm-diameter plugs were removed from each of three replicated 9-m^2 clone plots arranged in a randomized complete block design. Then, 10 g of sterile oats or oats

infested with a mixture of *O. herpotricha* isolates KS3, KS11, KS23, and KS27 (1:1:1:1, v/v) was randomly added to the bottom of one hole on each plot; the plugs were then replaced. SDS ratings were made in May 1987.

RESULTS

Two ectotrophic fungi, *O. herpotricha* (identified by J. Walker, Australia, and R. Shoemaker, Canada) and *G. incrustans* Landschoot & Jackson (5), were consistently isolated from roots and stolons of bermudagrass affected with SDS in Kansas. In preliminary experiments, only *O. herpotricha* caused extensive root discoloration (15), and no further tests were conducted with *G. incrustans*.

O. herpotricha was sterile in culture, and all attempts to initiate ascocarp formation on artificial media were unsuccessful. Nevertheless, the fungus occasionally produced fertile pseudothecia on stolons and crowns of inoculated plants in the greenhouse. Pseudothecia were 250–400 μm in diameter, with filiform, eight to 20 septate, ascospores 130–215 (152) × 2–3 (2.5) μm (Fig. 1). Ascospores were slightly twisted within the ascus. A complete taxonomic description of *O. herpotricha* is given by Walker (17). The fungus also is reported to have a pycnidial anamorph, *Scolecosporiella* sp. (6,19), but we did not observe pycnidial formation in culture or on diseased plant tissue. The identity of nonsporulating field isolates of *O. herpotricha* was determined by comparison with single ascospore isolates using restriction fragment length polymorphism analysis (14).

O. herpotricha produced a white, septate, cottony mycelium on PDA and malt agar (MA) that turned light tan to brown after 3–7 days. A few isolates produced a greenish tint in the center of the colony. Mycelium submerged in agar eventually turned tan to dark brown. Optimal growth rates for *O. herpotricha* isolates on PDA were 3.5–4.1 mm per day at temperatures between 20 and 25 C (Fig. 2). There was little growth at 30 C except for isolate KS11, and no growth was observed at 35 C.

In the greenhouse, *O. herpotricha, G. g.* var. *graminis*, and *L. korrae* significantly ($P < 0.05$) increased root discoloration ratings and decreased root dry weights on Arizona common bermudagrass; the two *O. herpotricha* isolates caused the most extensive root damage (Fig. 3). Roots and crowns of infected plants were dark brown to black and covered with dark ectotrophic hyphae (Fig. 1). These mycelial strands or cords, consisting of two to five hyphae joined together, commonly grew parallel to the root surface. Bulbillike structures of densely packed ectotrophic hyphae were frequently observed on roots and stolons.

A second group of experiments was conducted to determine the effect of soil temperature and inoculation with *O. herpotricha, L. korrae*, or *G. g.* var. *graminis* on root deterioration. There were no temperature × fungal isolate interactions on clones A-22 and E-29, so only treatment effects are reported in Table 1. The mean root weight of all inoculum treatments (including controls) was significantly ($P < 0.05$) higher at 25 C than at 15 C for both clones, but root discoloration ratings were not. All *O. herpotricha* isolates increased root discoloration ratings on A-22; the ascospore isolate (KS27) caused significantly ($P < 0.05$) more root discoloration than either mass-mycelium isolate. Nevertheless, only the ascospore isolate significantly ($P < 0.05$) reduced root weights. On clone E-29, both *L. korrae* and *O. herpotricha* increased root discoloration, but only *L. korrae* caused a significant ($P < 0.05$) reduction in root weight. Plants inoculated with *G. g.* var. *graminis* had little root discoloration and no significant reduction in root weight.

In May 1988, circular dead areas 15–30 cm in diameter developed on 55% (22/40) of the inoculation points made in September 1986 on clone A-29 in field plots. Similarly, 40% (16/40) of the inoculation points made on MidMo in September 1986 developed circular dead areas in 1988. No symptoms were observed on either plot in 1987. *O. herpotricha* was consistently reisolated from samples of diseased roots at the margin of dead

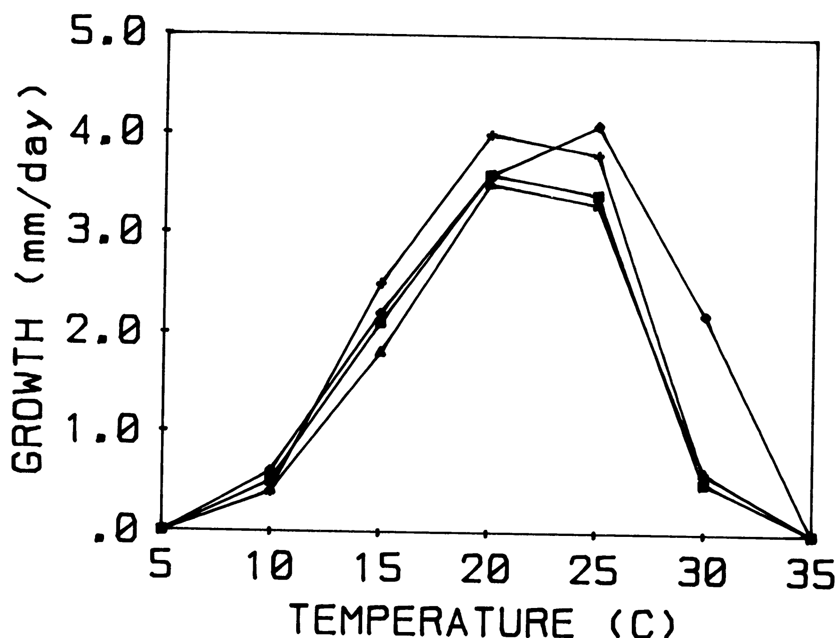

Fig. 2. Mean growth rates of four isolates (KS3, KS10, KS11, and KS27) of *Ophiosphaerella herpotricha* on potato-dextrose agar at different temperatures. Mean growth rates are averages of three replicate plates at each temperature. All isolates had similar growth rates except KS11, which had more growth at 30 C.

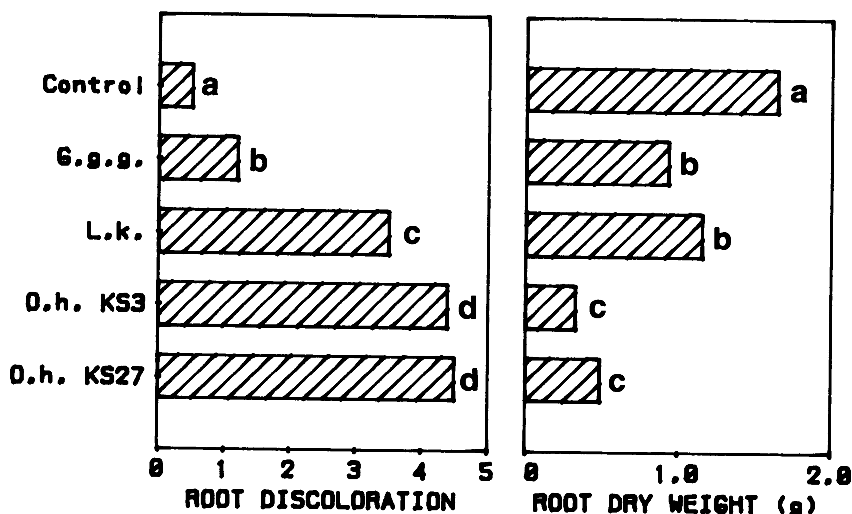

Fig. 3. Mean root discoloration (0 = no discoloration, 1 = 1–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–76%, and 5 = >76% of roots discolored) and root dry weights of Arizona common bermudagrass 90 days after inoculation with *Gaeumannomyces graminis* var. *graminis* (G.g.g.), *Leptosphaeria korrae* (L.k.), or two isolates of *Ophiosphaerella herpotricha* (O.h. KS3 and O.h. KS27). Bars without the same letter are significantly different ($P < 0.05$) by FLSD test.

patches. No discoloration or death of turf was observed in areas amended with sterile oats, and *O. herpotricha* was not isolated from root samples taken from these plots.

In May 1988, 63% of all plugs inoculated September 1987 with *O. herpotricha* in the replicated clone and cultivar field trial were dead. Turfgrass death was restricted to the area of the plug (10 cm diameter) that had been removed for inoculation. In contrast, none of the control plugs amended with sterile oats showed any discoloration or death of the turfgrass. Several clones suffered extensive winter injury and were not included in the ratings (Table 2). All other selections except Midiron and CT 23 had at least one plot in which the inoculated plug died. *O. herpotricha* was reisolated from roots in several of the diseased plugs.

DISCUSSION

O. herpotricha is found on a number of grass species in Europe, North America, and Australia (2,6,13,17,19) and is considered to be a saprophyte or weak pathogen of wheat (13). In early phytopathological literature, *O. herpotricha* was confused with *G. g.* var. *tritici* as the cause of take-all of wheat (2,13). Our results show that *O. herpotricha* is pathogenic to and capable of causing symptoms on bermudagrass identical to those reported for SDS. We have reproduced symptoms of SDS on 18 bermudagrass clones or cultivars in field trials. Ironically, Smith (11) first reported *Ophiobolus herpotrichus* (= *O. herpotricha*) as a cause of SDS in Australia in 1965; however, his isolates were later reidentified as *L. korrae* (18).

Several ectotrophic fungi, including *L. korrae, L. narmari*, and *O. herpotricha* are capable of causing symptoms of SDS on bermudagrass. *L. narmari* has been reported only in Australia (18), whereas *L. korrae* has been found in Australia (18) and throughout much of the northern portion (1,9,10,20), but not the south-central or southeastern part, of the United States. Whether *L. korrae* has a restricted geographic distribution or simply has escaped detection in southern regions is not known. We have consistently isolated *O. herpotricha* but not *L. korrae* in Kansas. *G. incrustans* and *G. g.* var. *graminis* also have been isolated from bermudagrass affected with SDS in Kansas and North Carolina, respectively, but the relationship of these fungi to SDS is unclear. *G. incrustans* did not cause extensive root rot or root weight loss of bermudagrass in greenhouse trials (15); its pathogenicity under field conditions has not been tested, however. In our greenhouse trials, *G. g.* var. *graminis* caused root rot on Arizona common bermudagrass but not on clone E-29. This fungus does not appear to be as virulent as either *L. korrae* or *O. herpotricha*. The association of several ectotrophic fungi with SDS complicates etiologic studies. In some areas, SDS may be a disease complex in which more than one ectotrophic fungus contributes to symptom development. Further research is needed on the identity and distribution of fungi associated with SDS.

Differentiation of *O. herpotricha, L. korrae, L. narmari*, and other ectotrophic fungi associated with SDS can be difficult because they do not readily produce fruit bodies in culture. *L. korrae* is mostly gray when grown on PDA, whereas *O. herpotricha* and *L. narmari* are light tan to brown; isolates of each fungus are variable in culture, however. Optimal growth temperatures and growth rates for *O. herpotricha* are similar to those reported for *L. korrae* and *L. narmari* and cannot be used for identification of these fungi. Other techniques, such as development of monoclonal antibodies to *L. korrae* (9), isoelectric focusing of fungal protein extracts to distinguish *L. narmari* from *L. korrae* (4), and differentiation of *L. korrae* from *O. herpotricha* by restriction fragment length polymorphism analysis (14), have recently been successfully used for identification of ectotrophic fungi associated with patch diseases of turf.

Factors that favor infection and colonization of bermudagrass roots by SDS-causing pathogens are poorly understood. SDS on bermudagrass in Australia is considered by Smith (12) to be a cool-season disease favored by soil temperatures below 20 C. Nevertheless, Crahay et al (1) found that *L. korrae* colonized bermudagrass roots at temperatures as high as 30 C, but plant mortality was observed only at temperatures of 15 and 20 C. In our short-term studies, both *O. herpotricha* and *L. korrae* caused root rot and a reduction in root weight, but not plant mortality, at both 15 and 25 C. This suggests that infection and colonization

Table 1. Effect of soil temperature on root discoloration and root weights of bermudagrass clones A-22 and E-29 inoculated with *Ophiosphaerella herpotricha*, *Leptosphaeria korrae*, or *Gaeumannomyces graminis* var. *graminis*

	Clone A-22		Clone E-29	
Treatment	**Root rating**[w,x]	**Root dry weight**[w,y]	**Root rating**	**Root dry weight**
Isolate[w]				
Control	0.4 a	1.35 a	0.5 a	3.52 a
O. herpotricha KS27	4.1 c	0.88 b	2.6 b	3.20 a
O. herpotricha KS11	2.7 b	1.45 a	···	···
O. herpotricha KS3	2.8 b	1.22 a	···	···
G. g. var. *graminis*	···	···	0.6 a	3.12 a
L. korrae	···	···	4.3 c	2.13 b
Temperature[z]				
15 C	2.5	0.95 a	2.0	2.44 a
25 C	2.4	1.51 b	2.0	3.54 b

[w] Plants inoculated with 2 g of sterile oats (control) or oats infested with Kansas isolates KS3, KS11, or KS27 of *O. herpotricha*; a Kentucky bluegrass isolate of *L. korrae*; or a bermudagrass isolate of *G. g.* var. *graminis*. Root discoloration and weights are combined means of inoculations at 15 and 25 C. Data in the same column not followed by the same letter are significantly different ($P < 0.05$) by FLSD test.

[x] Mean root discoloration on a 0–5 scale (0 = no discoloration, 1 = 1–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–76%, and 5 = >76%) 67 and 126 days after inoculation for A-22 and E-29, respectively.

[y] Mean root dry weights for isolate and temperature treatments 67 days and 126 days after inoculation for A-22 and E-29, respectively.

[z] Temperature effects on root discoloration and root weight for all treatments. There were no temperature × isolate interactions for either clone.

Table 2. Proportion of bermudagrass clone or cultivar plots developing symptoms of spring dead spot 1 yr after inoculation with *Ophiosphaerella herpotricha*

Clone or cultivar	Origin[a]	Proportion of dead plugs[b]
CT 23	California	0/3
NM 43	New Mexico	3/3
NM 375	New Mexico	3/3
NMS 14	New Mexico	1/3
MSB 10	Mississippi	2/3
MSB 20	Mississippi	2/3
Vamont	Virginia	2/3
E-29	Kansas	2/3
A-29	Kansas	1/3
Midiron	Kansas	0/3
A-22	Kansas	2/3
RS 1	Kentucky	2/3
Texturf 10	Texas	3/3
Tufcote	Maryland	2/3
Tifgreen	Georgia	3/3
Tifway	Georgia	2/3
Tifway II	Georgia	2/3
Guymon	Oklahoma	2/3
Arizona Common	···	2/3

[a] Location where clones or cultivars were originally selected.

[b] Proportion of turf plugs (10 cm diameter) in three replicated plots that were dead 8 mo after inoculation with *O. herpotricha*. NM 72, NM 471, NM 507, NMS 1, NMS 2, NMS 3, and NMS 4 from New Mexico, MSB 30 from Mississippi, and FB 119 from Florida suffered extensive winter injury and could not be rated. Soil plugs amended with sterile oats in all clone or cultivar plots, except those suffering winter injury, remained alive.

of roots by *O. herpotricha* or *L. korrae* could occur any time soil temperatures are between 10 and 25 C, but that low temperatures and/or dormancy factors play a major role in foliar symptoms and plant mortality. In greenhouse tests, we did not induce dormancy of the turfgrass; disease ratings were based on root weight reduction and not plant death. In the field, roots of susceptible cultivars may be damaged sufficiently by the fungus to decrease tolerance to cold temperatures during dormancy. In contrast, winter-tolerant cultivars such as Midiron may be able to tolerate root colonization by the pathogen. Further experiments are needed to determine the effects of colonization by *O. herpotricha* on winter hardiness of bermudagrass.

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LITERATURE CITED

1. Crahay, J. N., Dernoeden, P. H., and O'Neill, N. R. 1988. Growth and pathogenicity of *Leptosphaeria korrae* in bermudagrass. Plant Dis. 72:945-949.
2. Drechsler, C. 1934. Phytopathological and taxonomic aspects of *Ophiobolus, Pyrenophora, Helminthosporium*, and a new genus, *Cochliobolus*. Phytopathology 24:953-981.
3. Endo, R. M., Ohr, H. D., and Krausman, E. M. 1985. *Leptosphaeria korrae*, a cause of spring dead spot of bermudagrass in California. Plant Dis. 69:235-237.
4. Hawkes, N. J., and Harding, H. W. J. 1985. Isoelectric focusing as an aid to the identification of *Leptosphaeria narmari*, a cause of spring dead spot in turf. Aust. Plant Pathol. 14:72-76.
5. Landschoot, P. J., and Jackson, N. 1989. *Gaeumannomyces incrustans* sp. nov., a root-infecting hyphopodiate fungus from grass roots in the United States. Mycol. Res. 93:55-58.
6. Leuchtmann, A. 1984. Ueber *Phaeosphaeria* Miyake und andere bitunicate Ascomyceten mit mehrfach querseptierten Ascosporen. Sydowia 37:75-194.
7. Pair, J. C., Crowe, F. J., and Willis, W. G. 1986. Transmission of spring dead spot disease of bermudagrass by turf/soil cores. Plant Dis. 70:877-878.
8. Russell, C. C. 1984. A randomizable, gravity drained, soil temperature control system. Page 79 in: Proc. Int. Congr. Nematol. 1st.
9. Shane, W. W., and Nameth, S. T. 1988. Monoclonal antibodies for diagnosis of necrotic ring spot of turfgrass. (Abstr.) Phytopathology 78:1521.
10. Smiley, R. W., and Craven Fowler, M. 1984. *Leptosphaeria korrae* and *Phialophora graminicola* associated with Fusarium blight syndrome of *Poa pratensis* in New York. Plant Dis. 68:440-442.
11. Smith, A. M. 1965. *Ophiobolus herpotrichus*, a cause of spring dead spot of couch turf. Agric. Gaz. N.S.W. 76:753-758.
12. Smith, A. M. 1971. Spring dead spot of couch grass in New South Wales. J. Sports Turf Res. Inst. 47:54-59.
13. Sprague, R. 1950. Diseases of Cereals and Grasses in North America. Ronald Press, New York. 538 pp.
14. Tisserat, N. A. 1988. Differentiation of *Ophiosphaerella herpotricha* and *Leptosphaeria korrae* by restriction fragment length polymorphism analysis. (Abstr.) Phytophathology 78:1613.
15. Tisserat, N. A., Nus, A., and Pair, J. C. 1986. Characteristics and pathogenicity of two fungi isolated from bermudagrass affected with spring dead spot. (Abstr.) Phytopathology 76:1130.
16. Tisserat, N. A., Pair, J. C., and Nus, A. 1988. *Ophiosphaerella herpotricha* associated with spring dead spot of bermudagrass in Kansas. (Abstr.) Phytopathology 78:1613.
17. Walker, J. 1980. *Gaeumannomyces, Linocarpon, Ophiobolus*, and several other genera of scolecospored ascomycetes and *Phialophora* conidial states, with a note on hyphopodia. Mycotaxon 11:1-129.
18. Walker, J., and Smith, A. M. 1972. *Leptosphaeria narmari* and *L. korrae* sp. nov., two long-spored pathogens of grasses in Australia. Trans. Br. Mycol. Soc. 58:459-466.
19. Webster, J., and Hudson, H. J. 1957. Graminicolous Pyrenomycetes VI. Conidia of *Ophiobolus herpotrichus, Leptosphaeria luctuosa, L. fuckelii, L. pontiformis*, and *L. eustomoides*. Trans. Br. Mycol. Soc. 40:509-522.
20. Worf, G. L., Stewart, J. S., and Avenius, R. C. 1986. Necrotic ring spot disease of turfgrass in Wisconsin. Plant Dis. 70:453-458.