Pathogenicity of *Rhizoctonia solani* AG-2-2 and *Ophiosphaerella herpotricha* on Zoysiagrass

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ABSTRACT

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Rhizoctonia solani AG-2-2 was consistently associated with a large patch disease of zoysiagrass in Kansas, Missouri, and Tennessee. In field inoculation tests, patch symptoms developed during both spring and fall as the turfgrass was entering or breaking winter dormancy. The fungus infected and colonized leaf sheaths, but not stolons or roots, at 10–30 C, with optimal infection at 20–25 C. Rhizoctonia large patch development was suppressed in summer by thatch temperatures exceeding 30 C. *Ophiosphaerella herpotricha* was isolated infrequently from diseased zoysiagrass but did cause extensive root discoloration and weight loss in the greenhouse and circular dead patches 1 yr after field inoculations.

Additional keywords: binucleate *Rhizoctonia* spp., *Gaeumannomyces incrustans*, spring dead spot, *Zoysia japonica, Zoysia tenuifolia*

Zoysiagrass (*Zoysia japonica* Steud.) is a warm-season, perennial turfgrass that is used for amenity purposes in warm or transitional climates because it forms a uniform, low-growing, high-quality sod. Major problems of zoysiagrass include freeze damage, lack of wear tolerance during dormancy, insects, nematodes, and excessive thatch. Although zoysiagrass is subject to injury by numerous plant pathogens, it is reported to have fewer debilitating diseases than other turfgrass species (1). Nevertheless, damage caused by a large, patch-type disease has become a serious problem for turfgrass managers, particularly along the extreme northern range of zoysiagrass adaptation and use in North America.

In Kansas, patch symptoms are most common in spring and mid- to late fall as the turfgrass enters or breaks winter dormancy. Patches may also develop in shaded, moist areas during unusually cool midsummer weather. The etiology of this patch disease is not well defined. Although *Rhizoctonia solani* Kühn (1,5,14,21,23,26,30), binucleate *Rhizoctonia* spp. (5,7,8,18,21), and root ectotrophic fungi (26,31,32) are reported to be associated with patch symptoms, their pathogenicity to zoysiagrass in the field has not been fully documented. It is also unclear whether various names given to describe zoysia patch diseases, including brown patch, Rhizoctonia patch, large patch, winter brown patch, spring dead spot, and zoysia patch (3,18,23,31), refer to the same etiological agent or to a group of diseases with similar symptoms but caused by different fungi. Furthermore, the environmental conditions that favor patch development are poorly understood.

The purpose of our research was: 1) to determine the pathogen(s) of a spring and fall patch disease of zoysiagrass in Kansas, 2) to study the seasonal development of the disease, and 3) to determine the effects of temperature on infection and colonization of zoysiagrass by the pathogen.

MATERIALS AND METHODS

Fungal isolation and identification. Fungal isolations were attempted from the roots, stolons, and leaf sheaths of diseased zoysiagrass collected from locations in Kansas, Missouri, and Tennessee in the spring and fall when patch symptoms were present. Samples were washed with water to remove soil and thatch. Then, 1- to 2-cm sections of leaf sheaths, roots, or stolons were surface-sterilized with 0.5% NaOCl for 2 min, blotted dry, and placed on one-fourth strength potato-dextrose agar amended with chloramphenicol (10 mg/L) and streptomycin (10 mg/L) (one-fourth PDA^{++}). Ectotrophic fungi were maintained on PDA at 5 C, while *Rhizoctonia* spp. were stored on a sterile wheat bran and soil mix (4) at 25 C.

The nuclear condition of *Rhizoctonia* isolates was determined by 4′,6′-diamidino-2-phenylindole (DAPI) staining techniques (17). *R. solani* isolates were assigned to anastomosis groups (AG) by pairing with known AG strains and observing hyphal fusion (22). Intraspecific groups (ISG) within AG-2-2 were identified by growth of isolates at 35 C on PDA (27) and by cellular fatty acid analysis (28). *Gaeumannomyces incrustans* Landschoot & Jackson isolates were identified by ascospore morphology after pairing cultures with opposite mating types (15), and *Ophiosphaerella herpotricha* (Fr.:Fr.) J.C. Walker isolates were identified by genomic DNA hybridization techniques (25). Several other root ectotrophic fungi were isolated from roots and stolons, but these isolates were sterile in culture and could not be identified.

Pathogenicity of *R. solani*. Stolons of healthy zoysiagrass cv. Meyer were collected from field plots at Rocky Ford Turfgrass Research Station, Manhattan, Kansas. Stolon sections, including some shoot and root tissues, were surface-sterilized for 1–2 min in 0.5% NaOCl, rinsed in tap water for 1–2 min, and propagated in a calcined clay (Turface) at 22–28 C under an intermittent mist system in the greenhouse. After 1–4 mo, stolon sections containing three to 10 shoots were transferred to 2.5-cm-diameter × 10-cm-deep plastic pots (Cone-Tainers) containing a steamed soil:peat:perlite mixture (2:2:1, v/v) and allowed to grow for an additional 1–2 mo before inoculation.

Zoysiagrass was inoculated with five to eight oat kernels infested with *R. solani* AG-2-2 (isolate KS118) or uninfested, steam-sterilized oat kernels by placing the inoculum on the soil surface. Oat inoculum was prepared as described by Tisserat et al (29). Twenty-eight pots inoculated with *R. solani* AG-2-2 plus five pots amended with uninfested oats were placed in racks standing in 7.5-cm-deep plastic trays filled with water. Racks were loosely covered with translucent plastic bags to maintain a high relative humidity (>95% at 25 C) and placed in growth chambers maintained at constant temperatures of 10, 15, 20, 25, or 30 C with a 13-hr photoperiod. At 3-day intervals, four pots infested with *R. solani* AG-2-2 were removed from each chamber and individually rated for disease incidence by determining the percentage of shoots with distinct, water-soaked lesions on the leaf sheaths. Leaf sheaths exhibiting symptoms were surface-sterilized and placed on one-

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fourth PDA^{++} to verify the presence of *R. solani*. After 21 days, the five pots amended with uninfested oats were removed and rated in a similar manner. The experiment was repeated three times (blocks), with temperatures randomly assigned to chambers after each experiment. Areas under the disease progress curve (AUDPC) were calculated (2) at each temperature for the 21-day interval and compared by Duncan's new multiple range test following analysis of variance.

Pathogenicity of other fungi. Several ectotrophic fungi were isolated from roots and stolons of zoysiagrass exhibiting early spring patch symptoms. The pathogenicity of some of these isolates (Tables 1 and 2) to roots and stolons of zoysiagrass was tested in greenhouse experiments. Although *R. solani* AG-2-2 was consistently isolated from leaf sheaths but not from roots of diseased plants, it was also included to determine if it could cause a root rot.

Zoysiagrass was grown in 10-cm-diameter × 40-cm-deep plastic pots as previously described, then inoculated with 2 g of oats infested with one of the fungal isolates or with uninfested oats. Plants and soil were removed intact from the pots, and oat inoculum was uniformly inserted into the soil surrounding roots and stolons at approximately 2 cm below the soil surface. The inoculated turfgrass was then repotted, and pots were placed in holes punched in a 0.5-m-deep Styrofoam block in the greenhouse. Soil was maintained at ambient greenhouse temperatures (20–30 C) or at 14–16 C by means of a chilled, circulating water system (29). Plants were watered and fertilized as needed with Osmocote.

After 3 mo, plants were washed with water to remove soil from roots. Roots and stolons were rated for discoloration on a scale of 0–5, in which 0 = no discoloration, 1 = 0–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–75%, and 5 = >75% discoloration, then oven-dried for 96 hr at 50 C and weighed. Before being dried, subsamples of the roots and stolons were surface-sterilized and placed on one-fourth PDA^{++} as previously described.

Greenhouse inoculations were repeated four times in completely randomized, randomized block, or factorial (temperature × isolate) designs with five to 10 replicates. Not all fungi were represented in each experiment, but all species except the binucleate *Rhizoctonia* KS119 and the ectotrophic isolate KS120 were included in at least two of the experiments.

Field pathogenicity tests. Zoysiagrass cv. Meyer was inoculated 15 March 1991 at the Rocky Ford Turfgrass Research Station with uninfested oats or oats infested with *R. solani* AG-2-2 isolate KS118, binucleate *Rhizoctonia* isolates KS117 and KS119, *O. herpotricha* isolate KS115, *G. incrustans* isolate KS13, or unidentified root ectotrophic isolates KS4, KS84, KS85, KS120, and KS127. Inoculations with each isolate were replicated 20 times in a randomized complete block design. The turfgrass was divided into 3 × 3 m blocks with a 0.3-m barrier strip between blocks, then inoculated at one of 16 grid intersections spaced 0.7 m apart within each block by removing a 10-cm-diameter × 5-cm-deep cylindrical turf and soil core, inserting approximately 5 g of infested or uninfested oats in the hole, and replacing the core. Five of the grid intersections were left uninoculated within each block.

A second field inoculation plot was established on a blend of 12 cultivars of *Z. japonica* and *Z. japonica* × *Z. tenuifolia* Willd. ex Trin. at the Horticultural Research Center, Wichita, Kansas, in August 1991. Ninety sites were inoculated with *R. solani* AG-2-2 KS118 on a 0.5 × 1 m spacing. Other fungal isolates were not included.

Patch symptoms and diameters were recorded weekly in 1991 and monthly in 1992. Patches were considered active if leaf sheath lesions were observed at the patch margins. Minimum and maximum air and thatch temperatures were recorded daily by an electronic data logger in Manhattan and Wichita except in spring and summer of 1991, when only air temperatures were recorded. Rainfall and irrigation in plots were recorded manually or with a tipping-bucket rain gauge.

RESULTS

Symptoms. Patch symptoms on zoysiagrass were observed during both fall and spring. Fall patch symptoms developed in September or October as

Table 1. Root discoloration and root dry weight of zoysiagrass cv. Meyer 90 days after inoculation in the greenhouse with *Ophiosphaerella herpotricha* or the unidentified ectotrophic isolate KS4

		Experiment 1		Experiment 2	
Isolate[x]	Temp. (C)	Root rating[y]	Root dry weight (g)	Root rating	Root dry weight (g)
O. herpotricha-like KS4	15	2.8 ab[z]	0.68 a	4.1 a	0.89 a
	25	...	...	2.7 b	1.55 bc
O. herpotricha KS6	15	3.2 a	0.80 a	2.7 b	1.21 ab
	25	...	...	1.8 c	1.85 cd
O. herpotricha KS34	15	2.1 b	0.70 a	...	...
	25	...	...	...	...
Control	15	0.4 c	1.11 a	0.2 d	1.99 cd
	25	...	...	0.2 d	2.26 d

[x]Plants were inoculated with 2 g of sterile oats (control) or oats infested with Kansas isolate KS6 or KS34 of *O. herpotricha* originally isolated from bermudagrass or with an unidentified *O. herpotricha*-like isolate KS4 isolated from zoysiagrass.
[y]Mean root discoloration on 0–5 scale, in which 0 = no discoloration, 1 = 1–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–75%, and 5 = >75% discoloration.
[z]Means in the same column not followed by the same letter are significantly different ($P \leq 0.05$) according to Duncan's new multiple range test.

Table 2. Root discoloration and root dry weight of zoysiagrass cv. Meyer 90 days after inoculation in the greenhouse with fungi associated with patch-type symptoms of zoysiagrass in the field

		Experiment 1		Experiment 2	
Isolate[x]	Temp. (C)	Root rating[y]	Root dry weight (g)	Root rating	Root dry weight (g)
Ophiosphaerella herpotricha KS115	15	2.6 a[z]	0.36 ab	...	...
	25	2.8 a	0.37 ab	...	...
Gaeumannomyces incrustans KS69	15	0.2 b	0.54 a–e	0.2 c	0.64 a
	25	0.2 b	0.37 ab	...	...
Gaeumannomyces-like KS120	15	0.4 b	0.34 ab	...	...
	25	0.2 b	0.49 abc	...	...
Rhizoctonia solani AG-2-2 KS118	15	0.2 b	0.17 a	1.2 b	0.40 a
	25	0.0 b	0.43 abc	...	...
Binucleate *Rhizoctonia* KS117	15	0.2 b	0.79 cde	2.2 a	0.36 a
	25	0.0 b	0.50 b–e	...	...
Binucleate *Rhizoctonia* KS119	15	0.0 b	0.43 abc	...	...
	25	0.0 b	0.82 de	...	...
Control	15	0.6 b	0.89 e	0.0 c	0.53 a
	25	0.0 b	0.53 a–e	...	...

[x]Plants were inoculated with 2 g of sterile oats (control) or oats infested with Kansas isolate KS115 of *O. herpotricha*, KS69 of *G. incrustans*, KS118 of *R. solani* AG-2-2, or KS117 or KS119 of binucleate *Rhizoctonia* spp.
[y]Mean root discoloration on a 0–5 scale, in which 0 = no discoloration, 1 = 0–12%, 2 = 13–25%, 3 = 26–50%, 4 = 51–75%, and 5 = >75% discoloration.
[z]Means in the same column not followed by the same letter are significantly different ($P \leq 0.05$) according to Duncan's new multiple range test.

roughly circular, slightly matted areas of bright orange turfgrass that eventually faded to tan or dull brown. The margins of expanding patches remained bright orange. Small, water-soaked, reddish brown to black lesions were present on leaf sheaths but not on leaf blades, stolons, or roots of infected plants. Individual shoots within the diseased area were killed, resulting in a progressive thinning of the turfgrass. Patches ranged in diameter from less than 1 m to more than 8 m. During wet weather, patches expanded rapidly (>10 cm per week) and coalesced to blight large areas of turfgrass. Patch symptoms were observed until turf dormancy in mid- to late October.

Patches were again observed in the same locations in early to mid-April as zoysiagrass broke winter dormancy. Damaged turfgrass was straw colored and matted and had few healthy shoots. These early spring patches lacked bright orange margins typical of fall patches and more closely resembled spring dead spot symptoms on bermudagrass caused by *O. herpotricha* (29). Ectotrophic hyphae were noted on roots of zoysiagrass in the damaged areas. Nevertheless, most of these early spring patches developed bright orange margins and a sheath blight and began to enlarge in late April and early May as soil and air temperatures increased (Fig. 1). Disease development was suppressed by mid-June by unfavorable environmental conditions, and the zoysiagrass slowly refilled damaged areas during the summer. Many of the patches were perennial, i.e., they developed in the same locations in both spring and fall for several years.

Pathogenicity of *R. solani* AG-2-2. *R. solani* was consistently isolated from blighted leaf sheaths of zoysiagrass samples collected from patches with orange margins in both spring and fall at all locations sampled in Kansas, Missouri, and Tennessee. All *R. solani* isolates were AG-2-2; other *R. solani* anastomosis groups (AG-1, AG-4, and AG-5) commonly associated with turfgrass diseases (3) were not isolated. Isolates had optimal growth temperatures at 25–30 C, with minimal growth at 10 C and no growth at 35 C (Fig. 2). The isolates also had the same cellular fatty acid composition (analysis performed by R. K. Jones, University of Minnesota, St. Paul) as *R. solani* AG-2-2 isolates collected from St. Augustine grass (*Stenotaphrum secundatum* (Walter) Kuntze) in Texas (28).

R. solani AG-2-2 caused a sheath blight of inoculated zoysiagrass at all temperatures tested in growth chamber studies (Fig. 3). Blighted sheaths were detected 3 days after inoculation at 20–30 C. AUDPC values at 20 and 25 C were larger ($P < 0.05$) than those at 10, 15, or 30 C. Symptom development was delayed at 10, 15, and 30 C. At 10 C, no symptoms were detected 12 days after inoculation and only 11% of the shoots were blighted after 21 days. No symptoms were observed after 21 days at any temperature on plants inoculated with uninfested oats.

Field inoculations in Manhattan were made on 15 March 1991, and by 5 May, 85% (17/20) of the zoysiagrass sites inoculated with *R. solani* AG-2-2 had circular patches 5–30 cm in diameter. By 30 May, 95% of the sites had patches 18–71 cm in diameter. Zoysiagrass within the patches developed a sheath blight identical to that observed on naturally infected turfgrass. Sheath blighting continued until mid-June at air temperatures ranging from 2 to 30 C. The turfgrass completely recovered by mid-July as air and thatch temperatures increased.

Patch symptoms did not develop in Manhattan in fall 1991, but scattered diseased sheaths were found near inoculation sites. In Wichita, patches developed in early September and were active through fall dormancy (1 November). Although average daily thatch temperatures (7–24 C) were similar at both locations in fall 1991, the Wichita plot received abundant rainfall and supplemental irrigation in September and October, whereas the Manhattan plot received less than 2.0 cm rain and no supplemental irrigation. At both locations, patches 1.5–4.5 m in diameter developed in May 1992. The patches remained active through early to late June in Wichita and Manhattan, respectively, then slowly recovered in July and August. Patches redeveloped at both locations in September 1992.

Pathogenicity of other fungi. Although *R. solani* AG-2-2 was consistently isolated from zoysiagrass exhibiting fall and late spring patch symptoms, it was isolated infrequently (<10% of samples) from patches without orange margins in early April. In most cases, these patches developed orange margins in late April and *R. solani* was subsequently isolated. Nevertheless, other fungi, including *O. herpotricha, G. incrustans*, binucleate *Rhizoctonia* spp., and several unidentified ectotrophs, were isolated from discolored root samples collected from these early spring patches. Binucleate

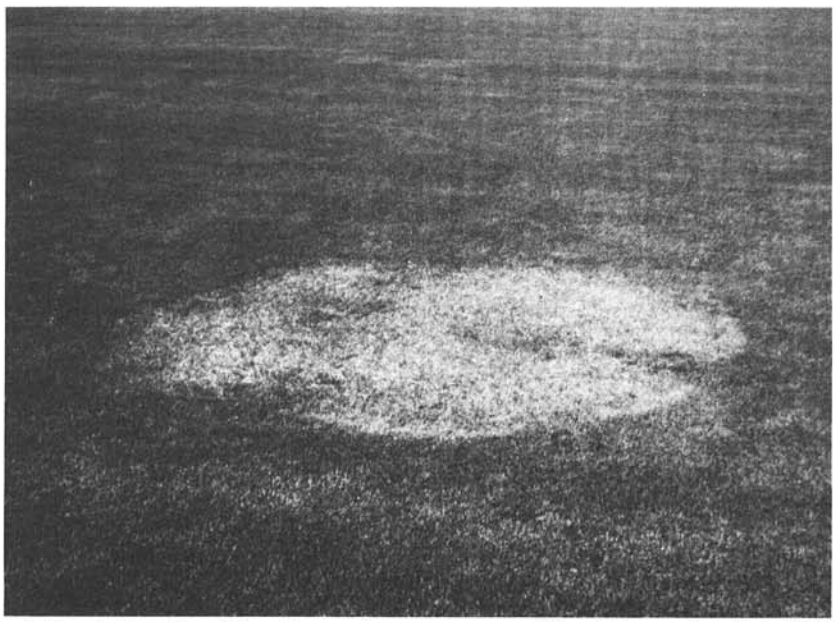

Fig. 1. *Zoysia japonica* with spring patch symptoms caused by *Rhizoctonia solani* AG-2-2.

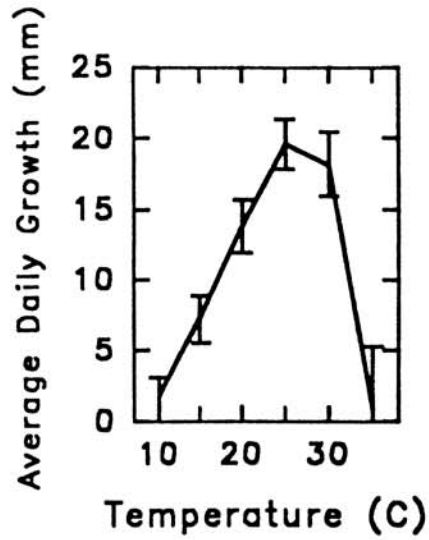

Fig. 2. Mean growth rate of *Rhizoctonia solani* AG-2-2 on potato-dextrose agar at different temperatures. Mean diameter growth rates are averages of six replicate plates of nine isolates. Bars indicate standard deviation. All isolates had no growth at 35 C after the first 24 hr.

Rhizoctonia isolates failed to pair with either CAG 1 or CAG 3 and remained unidentified. The isolation frequency of each fungus was not recorded, but none was consistently associated with patch symptoms. Since several of these fungi were previously reported to be associated with patch symptoms on zoysiagrass or other turfgrasses (29,31,32), we tested their pathogenicity to zoysiagrass in greenhouse and/or field experiments.

O. herpotricha KS115 and the *Ophiosphaerella*-like isolate KS4 caused extensive discoloration and, in some experiments, weight reduction of zoysiagrass roots 3 mo after inoculation in greenhouse experiments (Tables 1 and 2). Root discoloration and weight reduction were generally more severe at 15 C. Two isolates of *O. herpotricha* from bermudagrass also caused root discoloration on zoysiagrass (Table 1). *G. incrustans* KS69, the binucleate *Rhizoctonia* isolates KS117 and KS119, and the unidentified *Gaeumannomyces*-like ectotroph KS120 did not cause root discoloration, but the binucleate *Rhizoctonia* isolate KS119 and the ectotroph KS120 did cause a reduction in root dry weight at 15 C. Although *R. solani* AG-2-2 was previously shown to cause a sheath rot in growth chamber experiments, it did not cause root or stolon rotting when inoculum was placed in soil. In one experiment (Table 2), extensive sheath rot by *R. solani*, but not root rot, reduced root dry weight at 15 C.

In March 1992, 1 yr after inoculation, circular patches 10–30 cm in diameter developed on 85% (17/20) of field sites inoculated with *O. herpotricha*. The straw-colored patches persisted through April but did not enlarge. *O. herpotricha* was consistently isolated from rotted roots and stolons. No discoloration or death of turf was observed during 1991–1992 in areas amended with uninfested oats or oats infested with the unidentified root ectotrophic fungi, the binucleate *Rhizoctonia* isolates, or *G. incrustans*.

DISCUSSION

R. solani AG-2-2 has previously been shown in North America to cause patch symptoms on several warm-season turfgrasses (3,11,12) but not on zoysiagrass. The fungus has been associated with a large patch disease of zoysiagrass in Japan (21). Our results demonstrate for the first time that *R. solani* AG-2-2 is a common cause of a perennial, spring and fall large patch disease of zoysiagrass along the northern range of its adaptation in the United States.

All zoysiagrass isolates of *R. solani* AG-2-2 failed to grow at 35 C and were tentatively placed in ISG IV (20). However, cellular fatty acid composition of the zoysiagrass isolates was the same as that of the AG-2-2 isolates collected from St. Augustine grass in Texas but different from that of AG-2-2 IIIB or IV isolates from other host plants (R. K. Jones, *personal communication*). Therefore, the zoysiagrass isolates should be designated as AG-2-2 Turf (28) or ISG 2D (16).

Patch symptoms on zoysiagrass that developed in early April as zoysiagrass resumed spring growth typically lacked sheath blighting and bright orange patch margins associated with *R. solani*. Furthermore, *R. solani* was difficult to isolate from leaf sheaths or stolons in early spring. Several other soilborne fungi were isolated from the roots during this early spring period. Although some of the ectotrophic fungi caused root discoloration and/or root weight reduction in greenhouse inoculations, only *O. herpotricha*, a cause of spring dead spot of bermudagrass (29), caused patch symptoms in field inoculations. However, the low isolation frequency (two samples) of *O. herpotricha* suggests this fungus is not a common cause of spring dead spot symptoms on zoysiagrass. We believe that the majority of early spring patch symptoms are the result of sheath blighting caused by *R. solani* AG-2-2 in October before winter dormancy. Since zoysiagrass cannot produce new shoots while dormant, the damaged areas reappear as the turfgrass resumes spring growth. We noted that early spring patch symptoms were common in inoculated field plots that had been severely damaged by *R. solani* AG-2-2 the previous fall.

Patch symptoms in other warm-season turfgrasses caused by *R. solani* AG-2-2 normally develop during relatively cool, moist weather (3,11,12). Similarly, patch symptoms observed on zoysiagrass caused by *R. solani* AG-2-2 are most common in spring and fall as the turfgrass enters or breaks winter dormancy. In our studies, thatch temperatures during these intervals were often between 15 and 25 C and were near optimum for infection, based on results of growth chamber experiments. We also found that a reduced rate of infection could occur at 10 C. This may help explain the discrepancy in perennial patch diameters in spring and fall. It is likely that some undetected sheath blight continues after turfgrass dormancy in the fall or perhaps early spring. Thatch temperatures were often above 10 C for extended periods during turfgrass dormancy in our field plots.

Rhizoctonia large patch development on zoysiagrass is suppressed in summer by thatch and soil temperatures, which often exceed 30 C. These high temperatures are conducive for zoysiagrass root and shoot growth but inhibit fungal growth. Since *R. solani* AG-2-2 does not kill stolons or roots, the zoysiagrass can recover during the summer by forming new shoots on existing healthy stolons within diseased patches. In contrast, patch diseases on warm-season turfgrasses caused by root-colonizing ectotrophic fungi damage both roots and stolons (6,9,10,19,29), and turfgrass recolonization occurs by healthy stolon invasion from outside the diseased patch.

Leaf wetness and/or high relative humidity near the leaf surface are known to be important for infection of cool-season turfgrasses by *R. solani* (13,24, 26). We noted that patch symptoms of zoysiagrass were more severe on compacted, poorly drained soils and during periods of excessive rain. In fall 1991, patch symptoms developed in Wichita field plots that received excessive irrigation and rain; plots in Manhattan with similar thatch temperatures but less

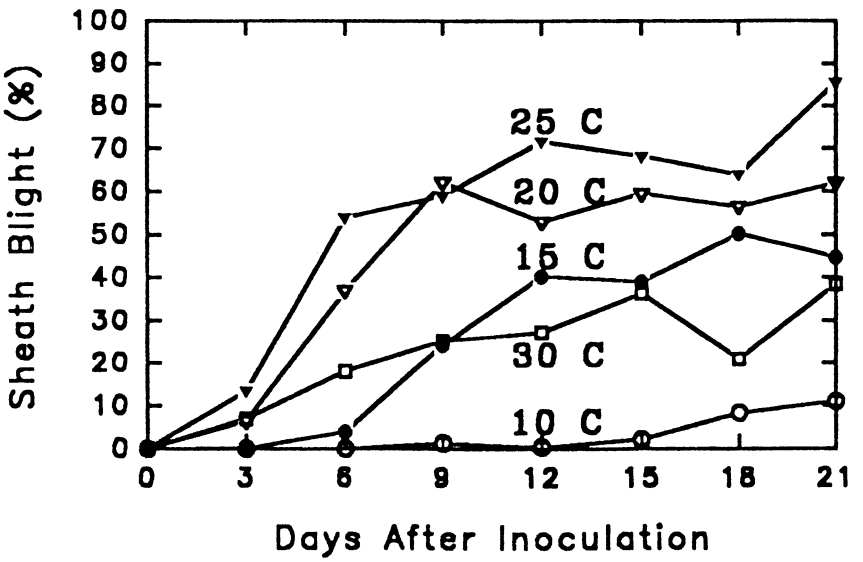

Fig. 3. Development of sheath blight on zoysiagrass over a 21-day interval following inoculation with *Rhizoctonia solani* AG-2-2 in growth chambers at different temperatures. AUDPC values for 20 and 25 C were greater ($P < 0.05$) than those at 10, 15, and 30 C. AUDPC values for 10 C were also less ($P < 0.05$) than those at 15, 20, 25, and 30 C.

moisture had only scattered sheath blight and no patch development. In growth chamber studies, sheath blight developed after inoculated plants were incubated in water-saturated soil and high relative humidity. Interestingly, infection also occurred when inoculated turfgrass was incubated in pots containing saturated soil but placed in the greenhouse with low relative humidity (*data not shown*). This suggests that relative humidities or surface moisture in thatch, and not the upper plant canopy, influences the infection process. Further studies on the effect of thatch and soil moisture on disease development are needed.

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