Rhizoctonia spp. Associated with Brown Patch of Saint Augustinegrass

B. Hurd and M. P. Grisham

Former graduate teaching assistant and assistant professor, respectively, Department of Plant Sciences, Texas A&M University, College Station 77843.

Portion of a thesis submitted by the senior author in partial fulfillment of the requirements for the M. S. degree, Texas A&M University, College Station.

Accepted for publication 1 July 1983.

ABSTRACT

Hurd, B., and Grisham, M. P. 1983. *Rhizoctonia* spp. associated with brown patch of Saint Augustinegrass. Phytopathology 73:1661-1665.

Isolates of *Rhizoctonia* from St. Augustinegrass (*Stenotaphrum secundatum*) displaying brown patch symptoms were either *R. solani* anastomosis group 2 type 2 (AG2-2) or unidentified *Rhizoctonia* species with binucleate hyphal cells. In the laboratory, *R. solani* AG2-2 isolates produced brown patch symptoms on stolon sections of cultivar Texas Common St. Augustinegrass at 24 C. An *R. solani* AG2-2 from St. Augustinegrass also caused postemergence damping-off in two cultivars of *Festuca arundinacea* and pre- and postemergence damping-off in five cultivars of *Lolium perenne*. No seedling disease was produced in two cultivars of *Poa pratensis*, one cultivar of *P. trivialis*, or one cultivar of *Agrostris palustris*. Isolates of the binucleate *Rhizoctonia* species were not pathogenic on cultivar Texas Common St. Augustinegrass or on any cool-season turfgrass tested. Symptom severity and rate of disease development varied among four cultivars and 11 accessions of St. Augustinegrass inoculated with *R. solani* AG2-2.

Additional key words: genetics, species concept.

Brown patch is a major fungal disease of St. Augustinegrass [*Stenotaphrum secundatum* (Walt.) Kuntze] affecting its production, maintenance, and utilization. In Texas, the disease is most severe in the fall, but may occur anytime during the growing season if environmental conditions favor disease development.

Rhizoctonia solani Kuhn was first reported as the causal agent of brown patch of turf in 1919 (19). Since then, Rhizoctonia brown patch has been reported to affect more than 100 species of grass (8). Endo (12) isolated several strains of *R. solani* from diseased turf that had brown patch symptoms, but not all were pathogenic. Luttrell (14) also noted differences in pathogenicity among isolates and classified turfgrass isolates of *R. solani* into four categories on the basis of cultural characteristics.

Recently, a fungus with predominantly binucleate hyphal cells and resembling *R. solani* has been isolated from turfgrass exhibiting symptoms atypical of brown patch disease (5,9,21,22). Isolates causing the disease known as yellow patch of turfgrasses were identified as *R. cerealis* van der Hoeven (5). Sanders et al (22) found that the binucleate fungi resembling *R. solani* exhibited pathogenicity towards creeping bentgrass from 10 to 27 C.

Essentially no reseeding occurs in established St. Augustinegrass turf, and consequently the chance for natural selection against brown patch has been limited. Sporadic flowering and poor germination have slowed the breeding of improved St. Augustinegrass cultivars. The development of methods to maximize flowering and improve seed germination and the release of pest-resistant cultivars have promoted the incorporation of disease resistance into St. Augustinegrass (11,23). Collections of St. Augustinegrass clones from several geographical areas may provide new sources of resistance genes. In the laboratory, Allen et al (1) demonstrated variable resistance of St. Augustinegrass seedlings to the brown patch pathogen, but indicated a need to correlate field studies of mature clones with laboratory results.

This investigation was undertaken to characterize the *Rhizoctonia* species associated with St. Augustinegrass exhibiting brown patch symptoms, to test their pathogenicity on St. Augustinegrass and several cool-season turfgrasses, and to develop a rapid screening technique to test clones of St. Augustinegrass for resistance to isolates of the brown patch pathogen.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

MATERIALS AND METHODS

Isolate collection and characterization. Observations of, and collections from St. Augustinegrass displaying symptoms of brown patch were made during the fall of 1979 and the spring and fall of 1980 and 1981 from areas in Bryan and College Station, TX. Stolons and sheaths of diseased plants were cleared and prepared for microscopic observations (18).

Surface-disinfested portions of diseased sheaths and nodal tissue were incubated on 1.5% water agar (WA) and examined after 48 hr for fungal growth. Hyphal tips from fungi resembling *R. solani* were transferred to potato-dextrose agar (PDA). Fungal characteristics as described by Parmeter and Whitney (17) were used to delineate isolates to species.

An attempt was made to induce basidospore production of each isolate using the modified soil-over-culture technique described by Tu and Kimbrough (25). The number of nuclei per hyphal cell was determined for each isolate by using the rapid staining technique of Tu and Kimbrough (24). Hyphal morphology and branching pattern were observed on the same slides used to determine nuclear number. Septal parenthesomes stained deep blue.

Mycelial growth rate was determined by transferring a 5-mm-diameter mycelial plug of each isolate onto PDA and incubating at 24 C. Colony diameter was measured after 72 hr. Three replications of each isolate were used. Cultures of each isolate were incubated on PDA at 24 C and examined at 4 wk for the presence of monilioid cells and colony coloration.

Anastomosis group determinations of multinucleate isolates were made by using the method of Parmeter et al (16). Representative cultures of binucleate isolates were sent to L. L. Burpee (University of Guelph, Guelph, Ontario N1G2W1) for anastomosis grouping.

Isolate pathogenicity on St. Augustinegrass. Cultivar Texas Common (obtained from Milberger Turf Farms Co., Bay City, TX 77414) plants were grown in 15-cm-diameter pots containing a mixture of peat, perlite, and sand (1:1:1, v/v). Greenhouse temperatures ranged from 26 to 28 C. Tap water was used to maintain adequate soil moisture. A granular fertilizer (13-13-13, N-P-K) was applied at 37.5 g per m^2 1 mo before the plants were used for testing. Growing tips were cut between the first and second

nodes from stolons that extended over the sides of the containers. The tips were washed for 30 min with running water to minimize surface contaminants, drained, and placed in a laminar-flow hood on paper towels for 10 min to remove excess water. The cut end of each tip was dipped in molten Paraplast (Lancer Co., St. Louis, MO 63103) to seal the wound.

Isolates of *Rhizoctonia* spp. from St. Augustinegrass and an *R. solani* AG2 type 2 (AG2-2) anastomosis tester (isolate R-9 from carrot) were grown on PDA. Ten 5-mm-diameter plugs from the margin of each actively growing colony and ten 5-mm-diameter plugs of sterile PDA were transferred individually to petri dishes containing approximately 20 ml of 1.5% WA. All dishes were incubated 10 days at 24 C.

A prepared stolon tip was placed in each WA dish and incubated at 24 C. Stolon tips were observed daily for 6 days for water-soaking and tissue discoloration. Tips of cultivar Texas Common placed on WA dishes containing plugs of sterile PDA served as uninoculated controls.

Isolate pathogenicity on cool-season turfgrasses. Two binucleate isolates of *Rhizoctonia* from St. Augustinegrass (01002 and 01009) that did not cause disease on cultivar Texas Common and one multinucleate isolate (01017) that did cause disease on that cultivar were tested for pathogenicity on 11 cultivars representing five species of cool-season turfgrasses. WA cultures of each isolate were grown as described for isolate pathogenicity on Texas Common. Five WA cultures of each isolate and five WA dishes containing sterile PDA plugs were used for each cultivar tested.

The turfgrasses selected for evaluation of isolate pathogenicity were Kentucky bluegrass (*Poa pratensis* L. 'Birka' and 'Kimono'), rough bluegrass (*P. trivialis* L. 'Sabre'), creeping bentgrass (*Agrostis palustris* Huds. 'Penncross'), tall fescue (*Festuca arundinacea* Schreb. 'Rebel' and 'Kentucky 31'), and perennial ryegrass (*Lolium perenne* L. 'Barry,' 'Celebrity,' 'Citadel,' 'Loretta,' and 'Venloma'). Seed from each were washed for 30 min with running water and air-dried in a laminar-flow hood on paper towels. Twenty-five seeds of each grass were placed in rows in each WA dish and incubated at 24 C under continuous light. They were examined for evidence of pathogenicity at 1 and 2 wk. Parameters chosen to indicate pathogenicity included seed germination and seedling survival.

Screening for resistance to *R. solani*. An isolate of *R. solani* AG2-2 pathogenic on cultivar Texas Common was used to screen four cultivars (Texas Common, Floratam, Seville, and Raleigh) and 11 accessions (FA43, FA64, FA83, FA87, MUT6, MUT7, MUT8, New Zealand 2, PI410356, PI410364, and TX109) of St. Augustinegrass. Ten-centimeter-diameter plugs of each grass were removed from established field plots, which had been treated monthly with 4.88 kg of nitrogen per 100 m^2, and placed in 15-cm-diameter pots containing a mixture of peat, perlite, and sand (1:1:1, v/v). The plants were maintained in the greenhouse for 2 mo. Screening for resistance was conducted by a method similar to that described above for the determination of isolate pathogenicity on Texas Common. Four WA cultures of the isolate and four WA petri dishes containing sterile PDA plugs were used for each grass type tested. Grasses placed in WA dishes containing sterile PDA plugs served as uninoculated controls. Rate of disease development and symptom severity were considered in determining levels of resistance and susceptibility. Disease severity was evaluated daily using a 0–5 visual rating scale with 0 = no symptoms, 1 = small area of the node water-soaked, 2 = entire node water-soaked with discoloration of tissue, 3 = sheath tissue directly above node water-soaked, 4 = node and substantial area of sheath water-soaked, and 5 = entire tip discolored and stolon easily detached from leaf sheaths.

RESULTS

Isolate collection and characterization. Dying patches of St. Augustinegrass with typical Rhizoctonia brown patch symptoms were observed in several localities of Bryan and College Station, TX. The patches varied in size, contrasting sharply with surrounding healthy turfgrass. Patches were circular to irregular in shape, and often coalesced in severely affected turf. The leaf sheaths were soft and discolored at the point of attachment to the stolon and were easily detachable from the node. Strands of hyphae were seen traversing sheaths when they were separated from the nodes. The stolon internodes remained green, and appeared to be unaffected by the pathogen. Isolated sprigs of apparently healthy grass were often found scattered within areas of affected grass.

A network of mycelium was observed on the surface of stolons and sheaths. The hyphae were brown and more concentrated around the nodes. Small sclerotialike bodies were also observed (Fig. 1A,B). Microscopic examination of cleared stolon and sheath surfaces revealed dark, lobate structures terminating short hyphal branches; these were similar in appearance to appressoria formed by *R. solani* associated with other host plants (9,10,13,15). The appressoriumlike structures were numerous and scattered on the surface of the stolons and sheaths and were not associated with stomata (Fig. 1C). Loosely packed aggregates of hyphae were also seen and were thought to be infection cushions or sclerotial initials (Fig. 1D).

Eleven representative isolates collected from sheath and nodal tissue of St. Augustinegrass displaying brown patch symptoms were selected for identification and pathogenicity studies.

The modified soil-over-culture technique failed to induce basidiospore formation by any isolate; consequently, other characteristics were used to identify the isolates (Table 1).

After 4 wk of growth at 24 C on PDA, the isolates were divisible into two color types, brown and white. The color intensities of the brown isolates ranged from tan to dark brown. Mycelium of the dark brown and white isolates grew appressed to the agar surface with little aerial growth, while that of the tan isolates had dense surface and aerial mycelium.

On PDA, the dark brown isolates produced variable amounts of brown crusty sclerotia ranging from 0.5 to 5.0 mm in diameter. Small, scattered sclerotia 0.2 to 0.5 mm in diameter were formed by the buff isolates. The white isolates did not produce sclerotia on PDA.

Hyphal cells of white isolates were consistently binucleate. Hyphal cells of brown isolates were multinucleate with five to nine nuclei per cell. These isolates were identified as *R. solani* AG2-2. The binucleate isolates were considered to be an unidentified species of *Rhizoctonia*. They did not fit the species concept of *R. cerealis* (3), nor did they anatomose with Burpee's (6) anastomosis groups CAG 1-7 of *Ceratobasidium* (L. L. Burpee, *personal communication*).

The diameters of runner hyphae (surface hyphae with relatively thick, parallel walls) of the binucleate isolates of *Rhizoctonia* ranged from 3.2 to 9.3 μm with a mean diameter of 5.0 μm. The runner hyphae diameters of the *R. solani* AG2-2 isolates ranged from 5.0 to 9.3 μm with a mean diameter of 6.2 μm (Table 1).

Linear growth rates of the binucleate isolates of *Rhizoctonia* ranged from 11.5 to 15.6 mm/day with a mean growth rate of 12.3 mm/day. Linear growth rates of isolates of *R. solani* ranged from 7.9 to 11.2 mm/day with a mean growth rate of 9.5 mm/day (Table 1).

Branching occurred near the distal septum in all isolates. The branches were constricted and septa were formed in the branches near their point of origin. Dolipore septa and monilioid cells were observed in all isolates.

Pathogenicity of isolates of *R. solani* on St. Augustinegrass. Pathogenicity and virulence to cultivar Texas Common St. Augustinegrass differed among isolates tested. The binucleate isolates and the AG2 type 2 (R-9) tester were not pathogenic to Texas Common. Leaves in these treatments and the uninoculated controls remained green and most stolon tips initiated roots that grew into the WA. The multinucleate isolates were pathogenic, producing symptoms similar to those associated with brown patch in the field (nodal water-soaking, discoloration, and softening of sheath tissue). Six days after inoculation, most of the tips were entirely discolored and the leaf sheaths were easily detached from the stolons. Stolons remained green, but roots were not initiated.

The pathogenic isolates differed in virulence. After 6 days, minimal symptoms of water-soaking were produced in three of the

TABLE 1. Characteristics of *Rhizoctonia* spp. isolated from St. Augustinegrass displaying brown patch symptoms

Fungus	Isolate	Mycelial color[a]	Nuclei per hyphal cell[b]	Anastomosis group[c]	Diameter of runner hyphae (μm)[d]	Mean radial growth rate at 24 C (mm/d)[e]
Rhizoctonia solani	01001	dark brown	MNR	2-2	(5.26)5.40(7.89)	9.50
	01007	dark brown	MNR	2-2	(5.01)5.17(5.37)	7.89
	01008	tan/dark brown	MNR	2-2	(5.17)5.42(5.56)	...[f]
	01013	tan/dark brown	MNR	2-2	(5.17)5.23(9.31)	11.10
	01014	dark brown	MNR	2-2	(5.17)7.20(7.40)	8.10
	01016	dark brown	MNR	2-2	(5.51)6.20(6.50)	11.17
	01017	dark brown	MNR	2-2	(5.88)6.20(9.31)	9.40
Rhizoctonia sp.	01002	white-buff	BNR	...[g]	(3.16)3.42(3.95)	12.44
	01009	white	BNR	...	(4.89)5.17(9.31)	11.50
	01010	white-buff	BNR	...	(5.01)5.12(5.23)	15.58[h]
	01011	white-buff	BNR	...	(4.14)5.17(5.69)	12.28

[a] Cultures were incubated on PDA for 4 wk at 24 C.
[b] MNR = predominantly multinucleate hyphal cells; BNR = predominantly binucleate hyphal cells.
[c] Anastomosis tests were for AG-1, AG-2 type 2, AG-3, and AG-4 only.
[d] Numbers represent minimum, mean, and maximum diameters, respectively.
[e] Based upon three replicates per isolate. Colonies measured after 72 hr of growth.
[f] Isolate ceased growth in culture for unknown reasons.
[g] Did not anastomose with any of the *R. solani* anastomosis group testers.
[h] Colonies measured after 48 hr of growth.

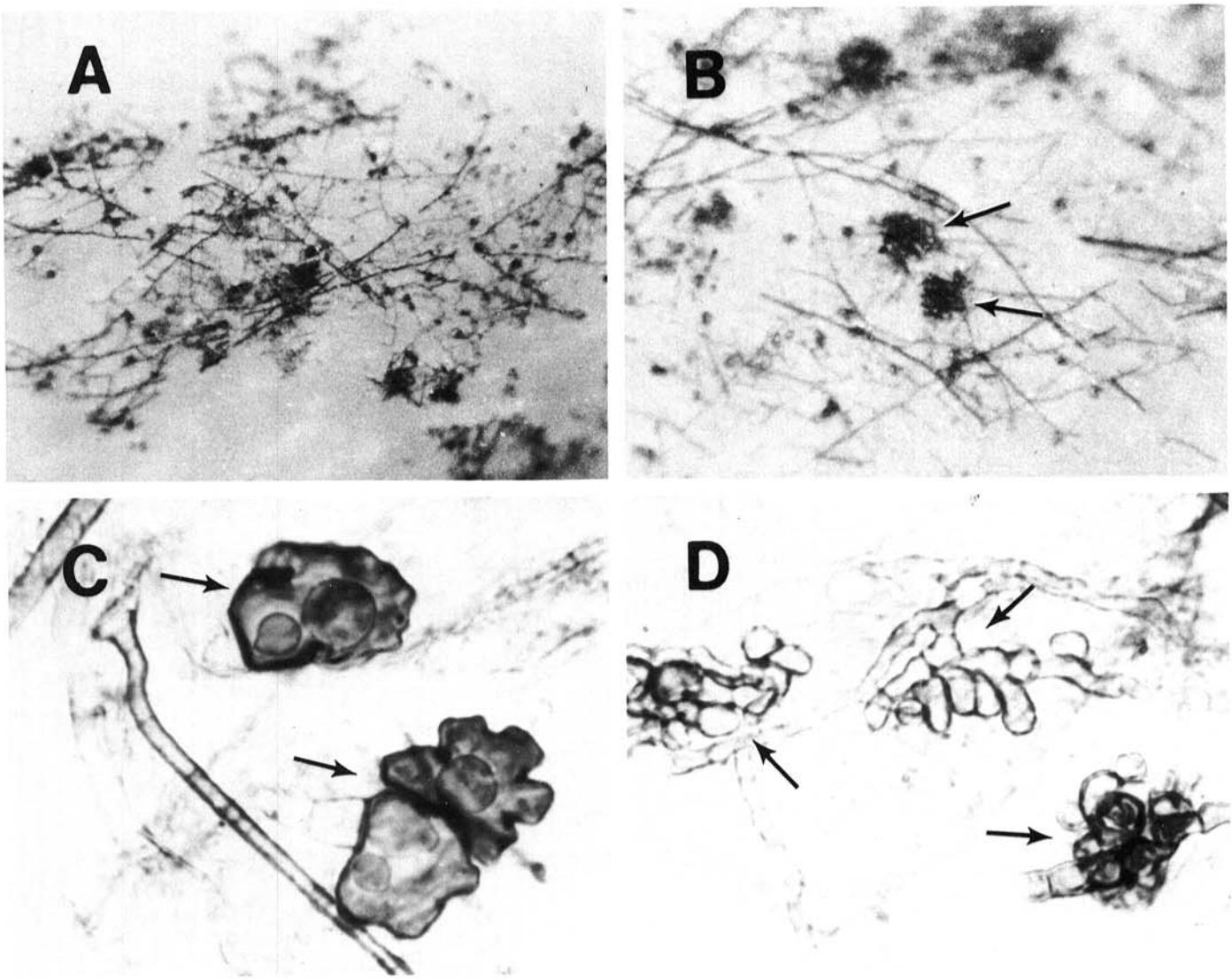

Fig. 1. *Rhizoctonia solani* AG2-2 on the surface of stolons and sheaths of St. Augustinegrass. **A and B,** Mycelium and sclerotialike bodies (arrows) (×7 and ×10, respectively). **C,** Appressoriumlike structures (×450). **D,** Possible infection cushions or sclerotial initials (×450).

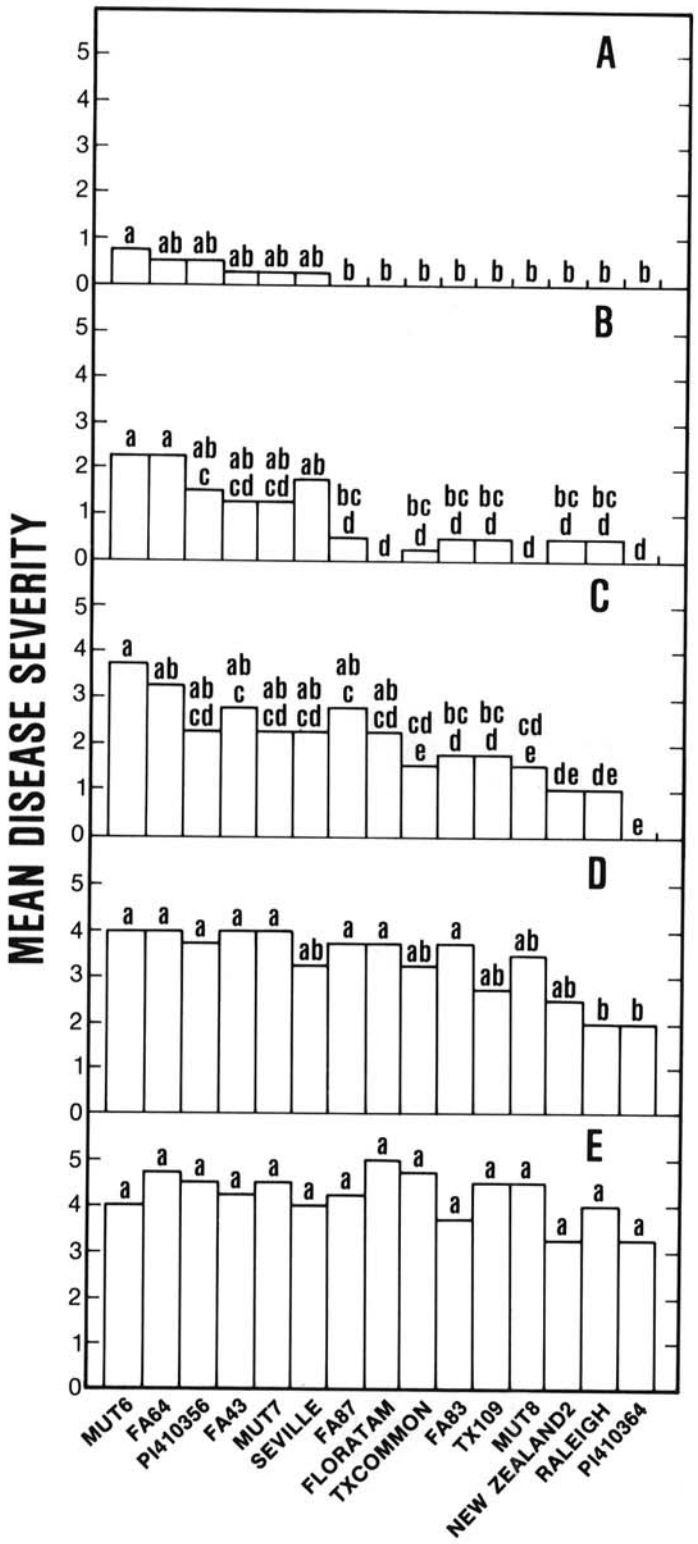

Fig. 2. Mean disease severity among four cultivars and 11 accessions of St. Augustinegrass inoculated with *Rhizoctonia solani* AG2-2 isolate 01017. **A to E,** ratings 2, 3, 4, 5, and 6 days after inoculation, respectively. **A,** 0–5 visual rating scale was used, in which 0 = no symptoms, 1 = small area of the node water-soaked, 2 = entire node water-soaked with discoloration of tissue, 3 = sheath tissue directly above node water-soaked, 4 = node and substantial area of sheath water-soaked, and 5 = entire tip discolored. Bars with the same letter at the top were not significantly different according to Duncan's multiple range test, $P = 0.05$.

ten tips inoculated with isolate 01001. Characteristic brown patch symptoms were produced in all tips inoculated with isolate 01014, but symptoms were not as advanced in tips inoculated with isolates 01007, 01008, 01013, 01016, or 01017.

Isolate pathogenicity on cool-season turfgrasses. Isolate pathogenicity and cultivar response differed among the cool-season turfgrasses (Table 2).

Binucleate isolates 01002 and 01009 did not reduce percentage seed germination or seedling survival in any turfgrass cultivar.

Multinucleate isolate 01017 was not pathogenic to *P. pratensis, P. trivialis*, or *A. palustris*. Seed germination of cultivars of *F. arundinacea* was not reduced by isolate 01017, but seedling survival was decreased. There was no decrease in seed germination of *L. perenne* 'Barry,' 'Citadel,' or 'Loretta' exposed to isolate 01017, but there was a decrease in that of cultivars 'Celebrity' and 'Venloma.' Seedling survival of all cultivars of *L. perenne* was decreased when exposed to isolate 01017 (Table 2). The coleoptile of diseased seedlings was soft and appeared discolored.

Screening for resistance to *R. solani*. All St. Augustinegrass selections tested were susceptible to isolate 01017 (*R. solani* AG2-2). In all selections, nodal water-soaking and discoloration progressed upward on the sheath tissue. The mean symptom severity ratings among the St. Augustinegrass selections differed over the test period. Only six selections displayed symptoms at 2 days (Fig. 2A). At 3 days, all but three St. Augustinegrass selections expressed symptoms (Fig. 2B), and only one turfgrass selection, PI 410364, had no symptoms at 4 days (Fig. 2C). At 5 days, symptoms were visible on all grasses (Fig. 2D). There was no difference in disease severity at 6 days; all ratings were high (Fig. 2E).

DISCUSSION

The isolates with binucleate hyphal cells were considered to be species of *Rhizoctonia* because of their hyphal dimensions, morphology, and branching pattern. Because the hyphal cells were predominantly binucleate, the isolates did not fit the species concept of *R. solani* (17). In addition, the binucleate isolates did not fit the species concept of *R. cerealis*, a previously reported binucleate *Rhizoctonia* spp. pathogenic on turfgrass (5). The binucleate isolates were not pathogenic on either cultivar Texas Common St. Augustinegrass or on any of 11 cool-season turfgrasses tested. Burpee (L. L. Burpee, *personal communication*)

TABLE 2. Seed germination and seedling survival of cool-season turfgrasses inoculated with isolate 01017 of *Rhizoctonia solani* AG2-2 collected from brown patch-diseased St. Augustinegrass

Botanical name Common name Cultivar	Germination (%)[v]		Seedling survival (%)[w]	
	Control[x]	01017	Control	01017
Festuca arundinacea				
Tall fescue				
Rebel	96.0 a[y]	96.0 a	99.2 a[z]	89.2 b
Kentucky 31	84.0 a	83.2 a	100.0 a	94.2 b
Lolium perenne				
Perennial ryegrass				
Barry	94.4 a	95.2 a	96.6 a	60.4 b
Celebrity	68.8 a	35.2 b	100.0 a	80.9 b
Citadel	96.8 a	94.4 a	95.1 a	64.4 b
Loretta	97.6 a	100.0 a	100.0 a	80.0 b
Venloma	89.6 a	60.8 b	100.0 a	85.2 b

[v] Percent germination = [(number of germinated seeds per treatment)/25] × 100.

[w] Percent seedling survival = [(number of viable seedlings after 2 wk)/(number of germinated seeds)] × 100.

[x] Control = seeds placed on 1-wk-old WA plates containing 5-mm-diameter plugs of sterile PDA.

[y] Mean of five replicates of 25 seeds per replicate. Mean percent seed germinated within a row followed by the same letter were not significantly different according to Duncan's multiple range test, $P = 0.05$.

[z] Mean of five replicates. Mean percent seedling survival within a row followed by the same letter were not significantly different according to Duncan's multiple range test, $P = 0.05$.

found that they do not anastomose with his anastomosis groups of *Ceratobasidium*, CAG 1-7. It appears that the binucleate *Rhizoctonia* spp. may play no part in the development of brown patch and that they are saprophytic species commonly found in the soil.

The isolates of *R. solani* were pathogenic on St. Augustinegrass and several of the cool-season turfgrasses tested. Burpee (4) reported that 11 of 15 isolates of *R. solani* collected from turfgrasses were AG1 and four were AG4. In this study, *R. solani* isolates from St. Augustinegrass belong to AG2 - 2 and represent a group previously unreported on turfgrasses.

Rhizoctonia brown patch of turfgrass has been generally associated with fungal penetration primarily through wounds and sometimes through stomata (8). Rowell's (20) observations of bentgrass, a cool-season turfgrass, showed that the leaf tips were affected first and the disease symptoms progressed downwards towards the plant crown. On St. Augustinegrass, the symptoms first appear at the nodal area and develop without wounds. The presence of appressoria and infection cushions on the surface of stolons and sheaths of St. Augustinegrass may indicate alternative modes of penetration by the fungus. Since the isolates of *R. solani* involved in brown patch of St. Augustinegrass belong to a different anastomosis group than those usually associated with cool-season grasses, pathogenic habit may differ. Differences in brown patch development between St. Augustinegrass and cool-season turfgrasses may result from anatomical differences among the host plants.

All selections of St. Augustinegrass tested were susceptible to *R. solani* AG2-2 isolate 01017. The rate of disease development based on symptom severity differed among selections. The mean disease severity ratings differed on days 2, 3, 4, and 5; however, after 6 days all selections were heavily diseased and there were no differences among selections. Discrepancies exist between laboratory ratings and observations of the St. Augustinegrass selections in the field. Differences between laboratory and field conditions may affect reactions of the different St. Augustinegrass cultivars; some of them display more field resistance. Host stress is an important factor in disease development and severity, and cultivars with resistance to other pests may appear less susceptible to brown patch in the field. Floratam, a cultivar of St. Augustinegrass resistant to chinch bugs and St. Augustine decline, is less susceptible to brown patch than is cultivar Texas Common in the field (2); however, when tested with our laboratory screening technique, both cultivars were rated the same. Further work is needed to correlate laboratory results with field reactions of the different St. Augustinegrass selections.

The pathogenicity studies conducted on the cool-season turfgrasses showed that an isolate of *R. solani* AG2-2 from St. Augustinegrass was not host specific and caused preemergence and postemergence damping-off in certain cool-season turfgrasses. Overseeding with cool-season turfgrasses such as fescue and perennial ryegrass during periods of brown patch development may result in a high percentage of seedling mortality. Using resistant cool-season turfgrass cultivars, such as the Kentucky bluegrass, and creeping bentgrass cultivars tested in this experiment, and not overseeding during periods favorable to brown patch development could minimize such problems.

LITERATURE CITED

1. Allen, W. W., Kilpatrick, R. A., and Bashaw, E. C. 1966. A technique for screening St. Augustine grass for tolerance to *Rhizoctonia solani*. Plant Dis. Rep. 50:622-623.
2. Beard, J. B., Toler, R. W., Crocker, R. L., and Grisham, M. P. 1982. St. Augustinegrass cultivar update. PR-4034. Pages 20-22 in: Texas Turfgrass Research—1982. Texas Agric. Exp. Stn. Consolidated PR-4032-4055. 79 pp.
3. Boerma, G. H., and van der Hoeven, A. A. 1977. A check-list for scientific names of common parasitic fungi. Series 26:Fungi on field crops: Cereals and grasses. Neth. J. Plant Pathol. 83:165-204.
4. Burpee, L. L. 1979. Identification of *Rhizoctonia* species associated with turfgrass. Pages 25-28 in: Advances in Turfgrass Pathology. B. G. Joyner and P. O. Larsen, eds. Harcourt Brace Jovanovich, Inc., Duluth, MN. 197 pp.
5. Burpee, L. L. 1980. *Rhizoctonia cerealis* causes yellow patch of turfgrasses. Plant Dis. 64:1114-1116.
6. Burpee, L. L., Sanders, P. L., Cole, H., Jr., and Sherwood, R. T. 1980. Pathogenicity of *Ceratobasidium cornigerum* and related fungi representing five anastomosis groups. Phytopathology 70:843-846.
7. Christou, T. 1962. Penetration and host-parasite relationships of *Rhizoctonia solani* in the bean plant. Phytopathology 52:291-380.
8. Couch, H. B. 1973. Diseases of turfgrass. Robert E. Krieger Publ. Co., Huntington, NY. 348 pp.
9. Dale, J. L. 1978. Atypical symptoms of *Rhizoctonia* infection on zoysia. Plant Dis. Rep. 62:645-647.
10. Dodman, R. L., and Flentje, N. T. 1970. The mechanism and physiology of plant penetration by *Rhizoctonia solani*. Pages 149-160 in: *Rhizoctonia solani*: Biology and Pathology. J. R. Parmeter, Jr., ed. University of California Press, Berkeley. 255 pp.
11. Dudeck, A. E. 1974. Flowering response of several selections of St. Augustinegrass. Pages 74-78 in: Proc. 2nd Int. Turfgrass Res. Conf. 18–21 June 1973. E. C. Roberts, ed. Virginia Polytechnic Institute, Blacksburg. 602 pp.
12. Endo, R. M. 1961. Turfgrass diseases in southern California. Plant Dis. Rep. 45:869-873.
13. Flentje, N. T. 1957. Studies on *Pellicularia filamentosa* (Pat.) Rogers III. Host penetration and resistance, and strain specialization. Trans. Br. Mycol. Soc. 40:322-336.
14. Luttrell, E. S. 1962. Rhizoctonia blight of tall fescue grass. Plant Dis. Rep. 46:661-664.
15. Marshall, D. S., and Rush, M. C. 1980. Histological aspects of infection of rice by *Rhizoctonia solani* and *Rhizoctonia oryzae*. (Abstr.). Phytopathology 70:569.
16. Parmeter, J. R., Jr., Sherwood, R. T., and Platt, W. D. 1969. Anastomosis grouping among isolates of *Thanatephorus cucumeris*. Phytopathology 59:1270-1278.
17. Parmeter, J. R., Jr., and Whitney, H. S. 1970. Taxonomy and nomenclature of the imperfect state. Pages 7-19 in: *Rhizoctonia solani*: Biology and Pathology. J. R. Parmeter, Jr., ed. University of California Press, Berkeley. 255 pp.
18. Phillips, J. M., and Hayman, D. S. 1970. Improved procedure for clearing roots and staining vesicular-arbuscular mycorrhyzal fungi for rapid assessment of infection. Trans. Br. Mycol. Soc. 55:158-161.
19. Piper, C. V., and Coe, H. S. 1919. *Rhizoctonia* in lawns and pastures. Phytopathology 9:89-98.
20. Rowell, J. B. 1951. Observation on the pathogenicity of *Rhizoctonia solani* on bentgrass. Plant Dis. Rep. 35:240-242.
21. Sanders, P. L., Burpee, L. L., and Cole, H. 1978. Preliminary studies on binucleate turfgrass pathogens that resemble *Rhizoctonia solani*. Phytopathology 68:145-148.
22. Sanders, P. L., Burpee, L. L., Sherwood, R. T., and Cole, H. 1976. *Ceratobasidium*: A pathogen of turfgrass. (Abstr.) Am. Phytopathol. Soc. 3:310.
23. Toler, R. W. 1973. A new St. Augustinegrass. Pages 67-70 in: Proc. 28th Ann. Tex. Turfgrass Conf., 10–11 December 1973, College Station, TX. 106 pp.
24. Tu, C. C., and Kimbrough, J. W. 1973. A rapid staining technique for *Rhizoctonia solani* and related fungi. Mycologia 65:941-944.
25. Tu, C. C., and Kimbrough, J. W. 1975. A modified soil-over culture method for inducing basidia in *Thanatephorus cucumeris*. Phytopathology 65:730-731.