Pathogenicity of *Rhizoctonia zeae* on Tall Fescue and Other Turfgrasses

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ABSTRACT

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Isolates of *Rhizoctonia zeae* were collected from various turfgrass sources and compared with one isolate of *R. solani* (anastomosis group 1) from tall fescue (*Festuca arundinacea*) and one isolate of *R. zeae* from corn roots (*Zea mays*) morphologically and for ability to induce foliar blight on turfgrasses in greenhouse studies. Tall fescue, perennial ryegrass (*Lolium perenne*), Kentucky bluegrass (*Poa pratensis*), creeping bentgrass (*Agrostis palustris*), common bermudagrass (*Cynodon dactylon*), and centipedegrass (*Eremochloa ophiuroides*) were inoculated with each of five isolates of *R. zeae* and one isolate of *R. solani*. *R. zeae* isolates blighted cool-season turfgrasses more severely than warm-season turfgrasses. Isolates of *R. zeae* originally obtained from lesions on grasses were more virulent on cool-season grasses than an isolate from corn roots and one from bentgrass affected with a summer dry wilt condition. Isolates of *R. zeae* were only mildly pathogenic on warm-season grasses. The *R. solani* isolate was more virulent than any of the *R. zeae* isolates tested.

Additional key words: brown patch, *Rhizoctonia cerealis*

Several species of *Rhizoctonia* are capable of inducing disease on several turfgrass species (1–3). *R. solani* Kühn induces brown patch on cool- and warm-season turfgrasses and is the most studied of these species (3). Brown patch is recognized as one of the major foliar diseases on turfgrasses in geographic areas with warm temperatures and high relative humidities (3).

It has been observed, however, that diseases of turfgrasses caused by fungi resembling *R. solani* also occurred under conditions cooler than "normal" for brown patch. These diseases have been referred to as "cool-weather brown patch" and have been demonstrated to be caused by binucleate fungi resembling *R. solani* (9). The binucleate *Rhizoctonia*-like fungus (RLF) incitant has been identified as *R. cerealis* van der Hoeven and the term "yellow patch" proposed for the disease it induces (1). Some binucleate fungi with *Rhizoctonia* mycelial states are species of *Ceratobasidium* (2), although a perfect state for *R. cerealis* has not been induced or identified in nature. Some binucleate RLF have been assigned to anastomosis groups similar to those of *R. solani* (2,7). In routine isolations of *Rhizoctonia* spp. and RLF from diseased turfgrasses in North Carolina, *R. zeae* Voorhees was isolated in hot (greater than 32 C) summer weather from foliar lesions on tall fescue (*Festuca arundinacea* Schreb.) typical of those induced by *R. solani*.

Turfgrasses were inoculated in greenhouse experiments with pure culture isolates of *R. zeae* obtained from these lesions and reisolated in pure culture from resulting foliar lesions (*unpublished data*). Isolates of *R. zeae* were also found more frequently in organic debris of tall fescue turf soil than were isolates of binucleate RLF and *R. solani* (*unpublished data*).

Young agar cultures of *R. oryzae* Ryker are similar to *R. zeae* agar cultures (8,10). Both *R. oryzae* and *R. zeae* have multinucleate hyphal cells. *R. oryzae* forms salmon to orange-colored sclerotial masses that are indefinite in size and shape (8), whereas isolates of *R. zeae* form smaller (0.5–1.0 mm), more regularly spheric sclerotia in culture that are white to cream-colored when young and become orange, red, and eventually brown to dark brown at maturity (13). Temperature-growth characteristics of *R. oryzae* and *R. zeae* are virtually identical (8). Sprague (10) recognized the pathogenicity of *R. oryzae* on some grasses and postulated that *R. oryzae* and *R. zeae* were possibly variants of *R. solani* derived from basidial segregates. This has not been proven to be the case. Perfect states for both *R. oryzae* and *R. zeae* have not been induced or identified.

R. zeae was first described from corn as an ear, root, and seedling pathogen (13). Sumner and Bell (12) recently discussed ecology of root diseases in corn induced by *R. zeae* and *R. solani*. *R. zeae* was also isolated from pearl millet by Luttrell (5) and noted to cause "mild infection on several grasses...." Voorhees (13) demonstrated that the fungus grew optimally in agar culture at 32–33 C and continued growth at 38–40 C. He postulated this high temperature tolerance to be a reason for infection of corn ears exposed to direct sunlight and high air temperatures during the summer.

The purpose of this study was to evaluate the pathogenicity of several isolates of *R. zeae* from different grass sources on several turfgrass species. Greenhouse conditions were used to resemble hot summer conditions in the southeastern United States in an attempt to clarify the role of *R. zeae* as a pathogen of turfgrasses.

MATERIALS AND METHODS

Isolates of *R. zeae* included RZ 197 and RZ 42 from diseased tall fescue turf from Raleigh, NC; RZ 227 from a diseased bentgrass (*Agrostis palustris* Huds.) golf course green in Rocky Mount, NC; RZ 215 from a bentgrass green in Raleigh affected with dry wilt symptoms in summer; and isolate C 2 (provided by S. I. Cohen) originally isolated from lesions

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on roots of corn. One pathogenic isolate of *R. solani*, RS 44 (anastomosis group 1) isolated from tall fescue in Raleigh, was included for comparison. These isolates had been previously obtained and identified (*unpublished data*).

Isolates were maintained on potato-dextrose agar (PDA) plates at 20–25 C. Inoculum was prepared for pathogenicity tests by growing isolates on a sterile tall fescue seed and water medium (1:1, w/v). Wetted fescue seed in 250-ml flasks was sterilized in an autoclave (121 C, 20 psi) for 30 min. After cooling, a 1-cm-diameter infested disk from a PDA culture of the appropriate test isolate was introduced into the sterile medium. This inoculum was grown for 2 wk at 28 C in the dark, and flasks were shaken periodically to promote uniform mycelial growth. Sclerotia were not formed in this medium during the incubation period.

Six grass hosts representing cool- and warm-season turfgrasses were utilized. Cool-season species included tall fescue Kentucky 31, perennial ryegrass (*Lolium perenne* L. 'Yorktown II'), Kentucky bluegrass (*Poa pratensis* L. 'Sydsport'), and creeping bentgrass (*Agrostis palustris* 'Penncross'). Warm-season turfgrasses included common bermudagrass (*Cynodon dactylon* (L.) Pers.) and centipedegrass (*Eremochloa ophiuroides* (Munro) Hack.).

Grasses were grown for 4 wk prior to inoculation in 10.2-cm clay pots in a pasteurized loam-sand mix (3:1, v/v) amended with 1.0 g of 8-8-8 granular fertilizer per kilogram of soil. Grasses were seeded at rates sufficient to produce dense swards approximating turf growing conditions. Foliage of all grasses was cut periodically to 6.5 cm and trimmed to that height before inoculation.

Grasses were inoculated by placing 0.5 g (fresh weight) of fescue seed inoculum around plant crowns on the soil surface in the center of each pot. Control plants of each species were treated similarly, with sterile, noninfested seed added around plant crowns. Isolate-grass treatment combinations were replicated six times and arranged in a completely random design on a greenhouse bench. Foliage in pots was covered with plastic bags supported from inside with a wooden stake, and pots were kept moist by subirrigation. Disease severity ratings were made 3, 7, and 10 days after inoculation by estimates based on the Horsfall-Barratt scale (4). The experiment was repeated, and greenhouse temperatures during experiment 1 were 28–33 C (day) and 26–28 C (night). Temperatures were cooler in experiment 2, with temperatures of 24–28 C (day) and 20–24 C (night).

Samples of foliage with lesions were collected from each grass after the final rating. Leaf pieces 2–5 mm long were cut from infected leaves, washed in three changes of distilled water, blotted dry on a sterile paper towel, and plated on 1.5% water agar. Hyphae resembling *Rhizoctonia* species were transferred as hyphal tips onto PDA plates and resulting isolates compared with original isolates used to prepare inoculum.

RESULTS

Lesions induced by *R. zeae* or *R. solani* were similar to previous descriptions (3) and became apparent 3 days after inoculation. Control plants of each host species remained free of infection. Disease severity continued to increase over the duration of both experiments; however, only the 10-day ratings were analyzed and reported because this latest rating best separated disease severity reactions induced by particular isolates. In all cases, reisolation yielded a culture identical in appearance to the inoculated strain.

Analysis of variance of disease severity ratings in both experiments 1 and 2 indicated significant isolate × grass interactions. The nature of these interactions was revealed by partitioning of mean squares into single degree-of-freedom linear contrasts (11) among disease severities of different turfgrass groupings (with emphasis on tall fescue, which is more frequently grown under conditions approximated in these experiments). The most striking difference indicated by contrasts was between cool- and warm-season turfgrasses in both experiments (Tables 1 and 2). This contrast and graphs of disease severities for specific isolates (Figs. 1 and 2) indicated the much greater disease severity on the cool-season grasses (Fig. 1) compared with bermudagrass and centipedegrass (Fig. 2). This effect was apparent for all isolates (Tables 1 and 2), including both *R. zeae* and the *R. solani* isolate.

Overall disease severities induced by *R. zeae* isolates were lower in experiment 2 than in experiment 1 (Figs. 1 and 2). *R. solani* isolate RS 44 induced approximately the same amount of disease in both experiments except on centipedegrass, which had some lesions in experiment 2 and very few in experiment 1 (Figs. 1 and 2). The *R. solani* isolate was more virulent than any *R. zeae* isolate tested. Disease severity differed between tall fescue and other cool-season grasses, depending upon isolate (Tables 1 and 2). Even though the disease severities induced by *R. zeae* isolates on the different cool-season grasses were lower in experiment 2, relative trends of disease response by specific isolates remained very similar, with the relative disease response induced by each isolate on each grass being similar within and between experiments (Fig. 1). These trends were associated with particular isolates of *R. zeae*, with the isolates originally recovered from grass foliar lesions (RZ 197, RZ 227, and RZ 42) inducing greater

Table 1. Single degree-of-freedom linear contrasts among disease severities[a] on turfgrass species in response to inoculations with *Rhizoctonia zeae* (RZ 197, RZ 227, C 2, RZ 42, RZ 215) and *R. solani* (RS 44) (experiment 1)

	Rhizoctonia isolate					
Grass contrasts	**RZ 197**	**RZ 227**	**RS 44**	**C 2**	**RZ 42**	**RZ 215**
Cool- vs. warm-season grasses	39.01**[b]	34.72**	173.2 **	10.89**	70.01**	18.00**
Between warm-season grasses	2.08	0.00	24.08**	3.00**	0.33	1.33
Tall fescue vs. perennial ryegrass	6.75**	12.00**	3.30	0.08	0.00	0.08
Tall fescue vs. Kentucky bluegrass	1.33	0.00	2.21	8.33**	14.08**	36.75**
Tall fescue vs. creeping bentgrass	21.33**	27.00**	3.30	0.75	3.00**	0.33
Error[c]	0.54	0.39	0.85	0.36	0.71	0.71

[a] Disease severity rated according to Horsfall-Barratt system.
[b] Mean squares from analyses of disease severity by specific *Rhizoctonia* isolates, where * and ** refer to significance at $P = 0.05$ and $P = 0.01$ levels, respectively.
[c] Error mean squares and contrast mean squares can be used to calculate F tests of significance with 1 and 179 degrees of freedom.

Table 2. Single degree-of-freedom linear contrasts among disease severities[a] on turfgrass species in response to inoculations with *Rhizoctonia zeae* (RZ 197, RZ 227, C 2, RZ 42, RZ 215) and *R. solani* (RS 44) (experiment 2)

	Rhizoctonia isolate					
Grass contrasts	**RZ 197**	**RZ 227**	**RS 44**	**C 2**	**RZ 42**	**RZ 215**
Cool- vs. warm-season grasses	16.05**[b]	10.13**	93.38**	1.38*	19.01**	4.01**
Between warm-season grasses	0.33	0.75	1.33	3.00**	0.33	0.33
Tall fescue vs. perennial ryegrass	1.33	0.08	8.33**	2.08**	0.00	0.33
Tall fescue vs. Kentucky bluegrass	0.33	3.00**	5.33**	0.75	3.00**	0.75
Tall fescue vs. creeping bentgrass	5.33**	1.33*	12.00**	1.33*	0.08	0.33
Error[c]	0.41	0.24	0.73	0.23	0.31	0.38

[a] Disease severity rated according to Horsfall-Barratt system.
[b] Mean squares from analyses of disease severity by specific *Rhizoctonia* isolates, where * and ** refer to significance at $P = 0.05$ and $P = 0.01$ levels, respectively.
[c] Error mean squares and contrast mean squares can be used to calculate F tests of significance with 1 and 179 degrees of freedom.

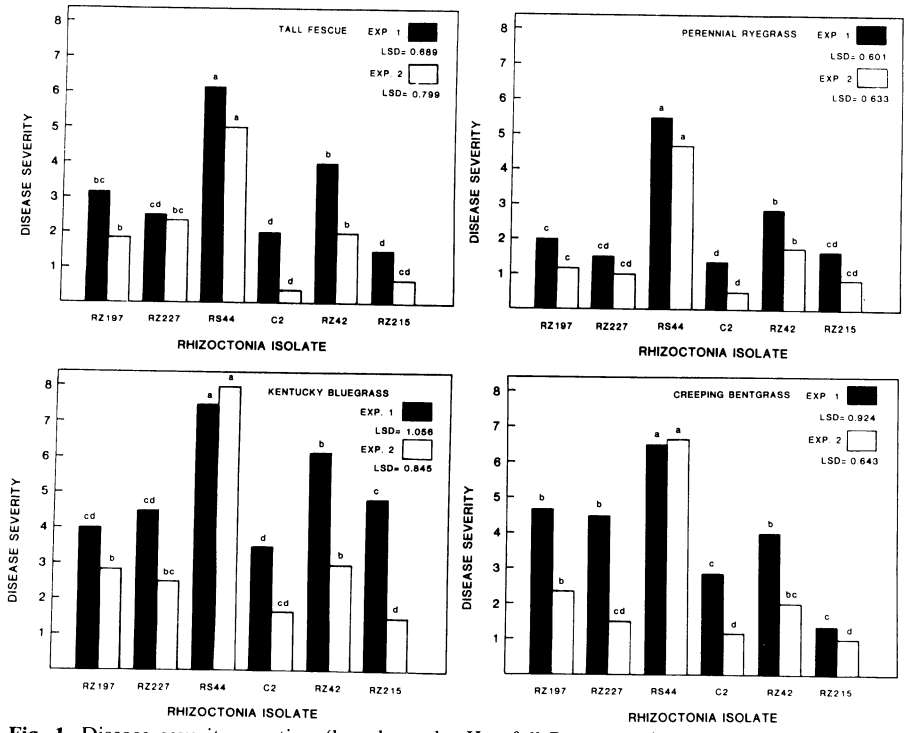

Fig. 1. Disease severity reaction (based on the Horsfall-Barratt rating scale) of cool-season turfgrass species inoculated with *Rhizoctonia zeae* (RZ 197, RZ 227, C 2, RZ 42, and RZ 215) and *R. solani* (RS 44) 10 days after inoculation. Bars with the same letter only within experiment are not significantly different (k ratio = 100) by Waller-Duncan's k ratio t test.

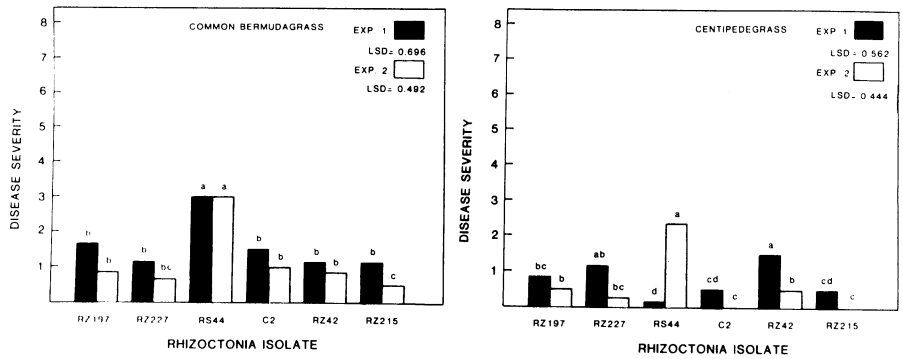

Fig. 2. Disease severity reaction (based on the Horsfall-Barratt rating scale) of warm-season turfgrass species inoculated with *Rhizoctonia zeae* (RZ 197, RZ 227, C 2, RZ 42, and RZ 215) and *R. solani* (RS 44) 10 days after inoculation. Bars with the same letter only within experiment are not significantly different (k ratio = 100) by Waller-Duncan's k ratio t test.

disease severities than the isolates from corn roots (C 2) and the isolate from bentgrass affected with a dry wilt condition (RZ 215).

DISCUSSION

Differences in virulence existed among *R. zeae* isolates used in this study; however, none of the *R. zeae* isolates was as virulent as the *R. solani* isolate on any grass studied. This particular isolate of *R. solani* (RS 44) is typical of isolates, most of which are in anastomosis group 1, from brown patch diseases of cool-season grasses in North Carolina (*unpublished data*).

Severity of disease induced by the *R. zeae* isolates was greater when greenhouse temperatures were warmer (experiment 1) than when temperatures were cooler (experiment 2). Because experiments were not designed to test the effect of temperature on disease response, the results suggest only that temperature may have been responsible for the differences. *R. zeae* was more frequently isolated, however, from lesions on tall fescue during periods of very hot weather even though *R. solani* was also isolated during these periods (*unpublished data*). Reports of *R. zeae* on corn (13), rice (8), and pearl millet (5) and the temperature-growth characteristics of the fungus in vitro (13) would support the contention that *R. zeae* induces more disease under hot conditions.

In the case of disease on cool-season grasses, there were differences in virulence based on isolate source. All isolates were pathogenic, but the disease severity was greater on grasses inoculated with isolates obtained from lesions on turfgrasses. The differences in disease severity induced between cool- and warm-season grasses were not surprising because cool-season grasses are under physiologic stress during periods of hot weather (6). The *R. zeae* isolates used in these experiments appeared to be less virulent than the *R. solani* isolate, although these differences in virulence could have been affected by temperature. Relative effects of mixed inoculum of *R. zeae* and *R. solani* under controlled environmental conditions may clarify the relative influence of each species under specific environmental conditions and lead to a better understanding of the disease complex induced by *Rhizoctonia* spp. on turfgrasses.

LITERATURE CITED

1. Burpee, L. 1980. *Rhizoctonia cerealis* causes yellow patch of turfgrasses. Plant Dis. 64:1114-1116.
2. 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.
3. Couch, H. B. 1973. Diseases of Turfgrasses. 2nd ed. Robert E. Krieger Publishing Co., Huntington, NY. 348 pp.
4. Horsfall, J. G., and Barratt, R. W. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
5. Luttrell, E. S. 1954. Diseases of pearl millet in Georgia. Plant Dis. Rep. 38:507-514.
6. Murray, J. J., and Powell, J. B. 1979. Tall Fescue. Agron. Ser. 20. Am. Soc. Agron., Crop Sci. Soc. Am., Soil Sci. Soc. Am., Madison, WI. 351 pp.
7. Parmeter, J. R., Jr., Sherwood, R. T., and Platt, W. D. 1969. Anastomosis grouping among isolates of *Thanatephorus cucumeris*. Phytopathology 59:1270-1278.
8. Ryker, T. C., and Gooch, F. S. 1938. Rhizoctonia sheath spot of rice. Phytopathology 28:233-246.
9. Sanders, P. L., Burpee, L. L., and Cole, H., Jr. 1978. Preliminary studies on binucleate turfgrass pathogens that resemble *Rhizoctonia solani*. Phytopathology 68:145-149.
10. Sprague, R. 1950. Diseases of Cereals and Grasses in North America. Ronald Press, New York. 538 pp.
11. Steele, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics: A Biometrical Approach. 2nd ed. McGraw-Hill, New York. 663 pp.
12. Sumner, D. R., and Bell, D. K. 1982. Root diseases induced in corn by *Rhizoctonia solani* and *Rhizoctonia zeae*. Phytopathology 72:86-91.
13. Voorhees, R. K. 1934. Sclerotial rot of corn caused by *Rhizoctonia zeae*. Phytopathology 24:1290-1303.