

## Necrotic Ring Spot Disease of Turfgrass in Wisconsin

G. L. WOLF, Professor, and J. S. STEWART, Project Specialist, Department of Plant Pathology, University of Wisconsin-Madison, Madison 53706, and R. C. AVENIUS, Project Specialist, Department of Plant Pathology, University of Illinois at Urbana-Champaign, Urbana 61801

### ABSTRACT

Wolf, G. L., Stewart, J. S., and Avenius, R. C. 1986. Necrotic ring spot disease of turfgrass in Wisconsin. *Plant Disease* 70:453-458.

A dark, ectotrophic fungus was consistently found in association with rotted crowns and roots of turf affected by a disease of uncertain cause. Symptoms were variable, but necrotic rings and patches predominated, and the disease was named "necrotic ring spot." Kentucky bluegrass was the primary host affected, but the organism was also isolated from other grasses. The fungus was isolated on several media, but the most consistent results were achieved after the tissue was washed and surface-disinfected, dried for 18 hr, then plated on potato-dextrose agar plus novobiocin. Pathogenicity was demonstrated on *Poa pratensis* and *P. annua* sod plugs and on seedlings of several members of Gramineae. A teleomorphic stage developed on inoculated roots of wheat, oats, and perennial ryegrass and has been tentatively identified as *Leptosphaeria korrae*.

Many turfgrass areas in Wisconsin are affected with a serious disease of undetermined cause that has increased in prevalence and severity since the mid 1970s. The disease is more prevalent on Kentucky bluegrass (*Poa pratensis* L.) than on other species. Its symptoms are variable. Distinct rings or patches of tan-colored turf sometimes occur, varying in size from 15 to 45 cm, similar to those of *Fusarium* blight (4). However, dark, ectotrophic fungal mycelium is associated

with the rotted crown and root tissues, and *Fusarium* is not always present in tissue, especially during incipient stages. Furthermore, the ergosterol biosynthesis inhibitors, which were reportedly very effective in controlling *Fusarium* blight (3,12), do not control this disease (16). These differences, together with unknown etiology, prompted our coining the name "necrotic ring spot" (NRS) to distinguish between the two diseases.

More subtle symptoms are sometimes observed. Irregular patches resembling those caused by drought or chinch bug injury contain living plants scattered among dead or very weak ones. Turf is stunted and often discolored but in these instances does not develop distinct rings or spots. Where rings occur, symptoms often expand slowly throughout the season or the turf may recover only to show symptoms again during the same or the following years. Necrotic patches often occur without living plants in their centers. The underlying thatch in such

patches often decomposes rapidly, leaving depressions or "crater spots" in the lawn.

Symptoms are most commonly observed in midsummer but can occur throughout the growing season. Turf 2-8 yr old is most likely to show symptoms (18), and the disease is more likely to develop on sloping or slightly elevated sites than in low areas. Heavy rains often interrupt symptom development until drier weather returns.

The variable pattern of patch development suggested that a complex of pathogens may be involved, but the uniformity of root and crown symptoms and common association with a dark, ectotrophic fungus led us to suspect a primary pathogen. Fine roots of diseased plants are brown, and rhizomes are often tan, brown, or black. Nodes of rhizomes, crowns, and bases of tillers often show distinct red or brown lesions (Fig. 1C). Some crown buds may remain alive, permitting plants to survive in a weakened condition. Under more severe conditions, crowns collapse and plants die. Darkly pigmented ectotrophic hyphae are consistently associated with affected roots and crowns and are abundant on severely diseased tissue. The fungus produces right-angle branches, often with a mantle of runner hyphae, and mycelial aggregations that resemble *Gaeumannomyces* spp. associated with cereals and grasses (Fig. 2A,B). However, we were unable to isolate any organism resembling the fungus observed from diseased tissue in preliminary trials.

Research supported by the College of Agricultural and Life Sciences, University of Wisconsin-Madison, Hatch Project 2614, the Wisconsin Turfgrass Association, and the Sod Growers Association of Mid-America.

Accepted for publication 28 October 1985.

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.

© 1986 The American Phytopathological Society

This study was undertaken to isolate the suspect fungus, determine its pathogenic role, and identify it. A preliminary report of the study was made earlier (17).

## MATERIALS AND METHODS

**Fungal isolation.** During the 4-yr period from 1979 through 1982, recently diseased plants from more than 400 NRS-affected turf sites were examined microscopically, and isolations were attempted from more than half of them. Kentucky bluegrass was the principal host, but annual bluegrass (*P. annua* L.), red fescue (*Festuca rubra* subsp. *rubra* L.), and bentgrass (*Agrostis palustris* Huds.) were included. The following media were used to isolate or study the suspect NRS fungus (NRSF): 2% water agar (WA); WA + 50 ppm of streptomycin (WAS); potato-dextrose agar (PDA); acid PDA (APDA); cornmeal agar (CMA); V-8 juice agar (V-8); PDA + 50 ppm of novobiocin (PDAN); PDA + 50 ppm of chloramphenicol (PDAC); soil-extract agar (SEA); and natural media agars consisting of bluegrass tissue either with dextrose (BDA) or without dextrose (BA) prepared similarly to Tuite's orchardgrass agar medium (13). Diseased crown, rhizome, root, and occasionally, leaf tissues were washed thoroughly in tap water, sectioned into pieces about 5–10 mm long, and treated variously before plating. Treatments included rinsing in sterile water only for 1–15 min as well as surface-sterilizing with one of the following: 1% NaOCl for 10–240 sec, 95% ethanol for 5–15 sec, and a 1:1 mix of 1% NaOCl and 95% ethanol for 15–60 sec. Tissue was then rinsed in sterile water. Some samples were blotted dry and plated immediately; others were allowed to dry as long as 18 hr on filter paper in sterile petri plates before plating. Not all combinations were used with all media.

Polk winter wheat (*Triticum aestivum* L.), Wright oats (*Avena sativa* L.), and Penncross bentgrass were used as fungal trap plants (8) by planting them over plugs of diseased turf that had first been inverted and placed over sterilized soil in 10-cm-diameter clay pots. Seeds were covered with sterilized soil. Isolations were then made from root and crown tissue on which the suspect fungus could be observed microscopically.

**Pathogenicity tests.** We conducted eight inoculation trials involving 11 NRSF isolates. Inoculations were conducted on seeded Baron Kentucky bluegrass; on 2-yr-old field-grown Kentucky bluegrass sod containing a blend of Baron, Glade, and Adelphi; and on annual bluegrass sod. Sod plugs were cut to fit into 10-cm-diameter clay pots, transplanted over sterilized soil, and usually allowed to become established for 7–14 days before inoculation. Inoculum was prepared in 250-ml flasks containing

45 g of millet, oat, or wheat grains hydrated for 18 hr in 60 ml of water, autoclaved twice for 30 min 24 hr apart, then inoculated with mycelial plugs that had grown on PDAN for 14–21 days. Inoculum was ready for use 4 wk later. About 5 cm<sup>3</sup> of inoculum was placed in a slit made in the center and around the edges of the sod plugs after transplanting. In two studies, inoculum (as described earlier) was placed between the soil and sod plugs before transplanting. In another study, sod plugs were thoroughly washed to remove the muck soil before transplanting and inoculation. After each inoculation, pots were watered thoroughly, then covered with plastic for about 48 hr. Where higher humidity was desired, pots were covered periodically for 2–4 days after watering. Effects on growth were determined by cutting the turf to a uniform height, then measuring plant height several days later. Four or more replicates were included in each trial. Temperatures of 24 C were used unless otherwise indicated. Hoagland's nutrient solution was used as needed to maintain the turf. The foliage condition was judged on a disease scale of 0–5, where 0 = healthy; 1 = slight chlorosis, <50% of turf affected; 2 = moderate chlorosis/necrosis, >50% of turf affected; 3 = 50–75% of turf chlorotic/necrotic; 4 = 75–100% of turf chlorotic/necrotic; and 5 = 100% of turf killed.

Most trials with sown grass were conducted in 5-cm-diameter plastic pots containing steam-sterilized white silica sand. Loam, muck, and mixtures of the three soil types were satisfactory, but seedling establishment was more uniform and root examinations more readily made when sand was used. Sand particles averaged between 0.5 and 0.7 mm in diameter. Seedlings were inoculated after 30 days by inserting 5 cm<sup>3</sup>/pot of the inoculum, prepared as described, into four shallow slits (three around the edge and one in the center).

Colonization and discoloration of host crowns and roots were examined further by sowing seeds of Baron Kentucky bluegrass, Penncross bentgrass, annual bluegrass, Pennlawn creeping red fescue, Wright oats, and Polk wheat. Aluminum foil pans (23 × 28 cm) with drain holes were filled 5 cm deep with steam-sterilized silica sand. Thirty cubic centimeters of infested millet was diluted with sand to facilitate uniform distribution, then spread over the surface. Half of each seed lot was drilled into the sand; the remainder was sown on the surface and lightly covered with sand. Pans were watered, then covered with plastic. Plants were dug 50 days later, washed free of sand, and examined visually for growth, discoloration, and decay and microscopically for fungal appearance by a severity scale of 0–4, where 0 = crown and roots healthy and white; 1 = roots gray; 2 = roots black but neither root nor foliage

growth visibly reduced; 3 = root growth reduced with substantial cortical decay and some foliage discoloration; and 4 = roots stunted, black, and severely rotted, foliage chlorotic. Isolations were also made.

**Fungal characteristics.** Hyphal and cultural morphology were examined principally on PDAN and to a lesser extent on V-8 and PDA. Efforts to detect or induce an anamorphic or teleomorphic stage included periodic examination of naturally infested turf over a period of 2 yr, inoculation and observation of the fungus on various hosts, and systematic examination of cultures growing on the previously described media for periods as long as 6 mo at temperatures ranging from 4 to 32 C and maintained either in the dark or in alternating light and dark environments. Growth rates were determined for five isolates by plating 7-mm PDA agar plugs of inoculum onto fresh PDA and incubating at four-degree increments from 4 to 32 C. The fungus was also compared with cultures of *Gaeumannomyces graminis* var. *tritici*, *G. graminis* var. *avenae*, *Phialophora graminicola*, a *Phialophora* sp. with lobed hyphopodia (provided by P. T. W. Wong), and an isolate with simple hyphopodia believed to be *G. graminis* var. *avenae* that was obtained from a bentgrass golf fairway in Wisconsin.

## RESULTS

**Isolation studies.** Success in isolating and recognizing the NRSF was first obtained from trap-plant tissues. The organism had been overlooked in isolation plates made earlier because of its slow growth, frequent contamination with other microflora, and our unfamiliarity with it. No single technique was best for isolating the fungus from all turf sites. The most consistent results, however, were obtained when washed sections were rinsed in sterile water for 15–30 min, then surface-disinfected in a 1% NaOCl-95% ethanol solution (1:1) for 30 sec and rinsed again in sterile water for 1 hr. Sections were then dried on filter paper for 18–24 hr at room temperature. Most media supported growth of the suspected causal agent, but fewer contaminations and the most consistent isolations were obtained when PDAN was used and the tissue was dried for several hours before plating. Pure cultures were often obtained with this method (Fig. 2C). A small amount of growth was usually evident about 7 days after plating and became identifiable after another 10 days.

**Pathogenicity trials.** Centers and edges of inoculated sod plugs, near the inoculum, began to turn tan or yellow after 21–30 days. After another month, symptoms extended outward from these centers to affect virtually all of the turf in pots (Table 1, Fig. 1B). The first-affected turf was brown and dead, whereas most of the

remaining turf was tan, yellow, or red. The NRSF was present over crown and root tissue and was readily reisolated from inoculated seedlings and sod.

Inoculations were generally successful, although no symptoms developed during two trials. No significant or consistent differences were observed among treatments inoculated with the same rates of millet, wheat, or oat grains, in contrast to preliminary trials, so the results were pooled. A trace of dark mycelium resembling NRSF was sometimes observed microscopically in the sod plugs used for the tests. Washing the soil from sod plugs before use did not influence symptom development in the inoculated turf but did increase turf height at 30 days and improve turf appearance of the noninoculated plugs after 40 days (Table 2). Symptoms developed at both 20 and 28 C, but periods of exposure to high humidity were necessary for severe disease development at the lower temperature (Table 3). At 20 C and low humidity, symptoms sometimes failed to develop. When they did develop, the turf appeared weakened and the stand was reduced, but complete kill was not observed.

Crown and root tissues of grasses planted in artificially infested silica sand became necrotic and were dark brown to black (Table 4). Cereal grasses were easier to examine because of their larger size. Wheat was more susceptible than oats. However, roots of all crops examined were severely damaged under the conditions of this test. Not only were the root tissues discolored and decayed but the fungal mass over the surface was sometimes visible even to the unaided eye. In a subsequent test, ryegrass roots (*Lolium perenne*) were discolored, but plants continued to grow and new roots developed. Runner hyphae, plates of mycelium, and mycelial aggregations typical of those observed on naturally infected plants from the field were evident under the microscope (Fig. 2A). The fungus also grew more than 2 mm beyond the tissue, apparently using the host as a base from which it grew through the sand.

Although lesions similar to those of field infections were sometimes observed, inoculated tissue was usually darker than that typically seen in the field. That may have resulted from the age or nutrition of host tissue, level of inoculum used, or absence of competitive organisms. Most but not all plants that were not sacrificed for root and crown examination died a short time later. Roots and crowns from control treatments generally remained white and firm, although *Alternaria* and other contaminants caused minor discoloration in some instances.

We tested susceptibility of annual bluegrass sod plugs at 16 and 24 C to isolates obtained from Kentucky bluegrass and annual bluegrass crowns. Symptoms began to appear within 2 wk. Plants

turned yellow and grew slowly. Leaves then turned tan and plants died. Symptoms were more severe with both isolates at 24 than at 16 C after 30 days.

**Fungal characteristics.** Growth on PDA was initially off-white to gray and somewhat floccose. As growth continued, both the media and older mycelium became almost black. Aerial growth of the fungus maintained some gray color,

but the reverse side was always black. The advancing hyphal margin sometimes showed a trace of curling back toward the center, typical of *Gaeumannomyces*, but this was not common. Hyphae of seven isolates averaged 3.6–4 μm in diameter, similar to hyphae of the *Gaeumannomyces* and *Phialophora* cultures provided by Wong. However, margins of Wong's isolates curled back more distinctly than

**Table 1.** Pathogenicity of six isolates of the necrotic ring spot fungus on Kentucky bluegrass seedlings and sod plugs 30 and 57 days after inoculation

Isolate	Disease severity <sup>a</sup>			
	Seedlings		Sod plugs	
	30 Days	57 Days	30 Days	57 Days
6-17-7	1.3	3.5	2.1	2.7
8-5-8	0.5	3.8	1.7	3.2
8-28-17	2.8	3.5	2.1	3.3
9-15-27	1.5	2.3	1.9	2.6
9-21-8	1.0	2.7	2.0	3.1
11-4-10	2.0	2.8	2.0	3.1
Millet control	0.0	0.7	0.3	1.3
No treatment	0.0	1.3	0.9	1.2

<sup>a</sup>Disease severity index based on a rating of 0 = no chlorosis or necrosis to 5 = 100% of turf killed (average of seven replicates per entry).

**Table 2.** Comparison of inoculated Kentucky bluegrass sod plugs washed free of soil vs. nonwashed plugs<sup>y</sup>

Isolate	Treatment	Disease severity <sup>w</sup>		Turf height (cm) <sup>x</sup>	
		30 Days <sup>y</sup>	40 Days	30 Days	40 Days
7-16-2-1B	Washed	2.4 b <sup>z</sup>	3.9 c	5.1 ab	3.8 a
	Not washed	2.0 b	3.8 c	4.8 a	5.1 b
Millet control	Washed	0.0 a	0.5 a	6.4 c	6.4 c
	Not washed	0.1 a	1.0 b	5.1 ab	6.4 c

<sup>y</sup> Millet infested with necrotic ring spot fungus was placed below the turf at transplanting.

<sup>w</sup>Disease severity index based on a rating of 0 = no chlorosis or necrosis to 5 = 100% of turf killed (average of eight replicates per entry).

<sup>x</sup>Turf was cut to a height of 3.5 cm 4 and 7 days before the two recorded dates, respectively.

<sup>y</sup>Number of days after transplanting.

<sup>z</sup>Small letters indicate modified Tukey's *t*-test groupings of treatments that do not differ significantly at *P* = 0.05.

**Table 3.** Pathogenicity of two isolates of the necrotic ring spot fungus on Baron Kentucky bluegrass at 20 C (low and high humidity) and 28 C (low humidity only)

Isolate	Inoculum placement <sup>a</sup>	Temperature (C)	Humidity	Disease severity <sup>b</sup>		Turf height (cm) <sup>c</sup>	
				39 Days <sup>d</sup>	48 Days	36 Days	43 Days
7-16-2-1B	Within	28	Low	1.1	3.5	4.8	5.6
7-16-2-1B	Below	28	Low	2.1	3.6	5.6	5.6
6-29-2-3A	Below	28	Low	1.6	3.8	6.1	4.3
Grain control	Within	28	Low	0.3	1.1	6.1	5.8
	Below	28	Low	0.7	1.9	6.4	5.8
7-16-2-1B	Within	20	High	2.2	4.3	4.3	4.6
7-16-2-1B	Below	20	High	0.5	3.1	6.1	6.6
7-16-2-1B	Below	20	Low	0.5	1.4	5.1	4.8
6-29-2-3A	Below	20	Low	0.3	2.1	4.8	5.1
Grain control	Within	20	High	0.1	0.6	6.6	7.6
	Within	20	Low	0.1	0.3	6.4	6.6
	Below	20	High	0.2	1.5	6.4	7.1
LSD ( <i>P</i> = 0.05)				0.6	0.4	0.6	0.6

<sup>a</sup>Infested grain was placed below sod plugs or within crowns after transplanting.

<sup>b</sup>Disease severity index based on a rating of 0 = no chlorosis or necrosis to 5 = 100% of turf killed (average of 12 replicates, four each of infested millet, oat, and wheat entries).

<sup>c</sup>Turf was cut to a height of 3.5 cm 4 and 7 days before the two recorded dates, respectively.

<sup>d</sup>Number of days after inoculation.

NRSF cultures and also grew more rapidly (Table 5). One culture we obtained from bentgrass was very similar to *G. graminis* var. *avenae* in these characteristics and was tentatively identified as that species. Optimal

temperatures for NRSF growth were 20–28 C; growth was scant at 4 C and was inhibited at 32 C.

Erumpent pseudothecia (Fig. 2D) containing bitunicate asci were observed on roots and lower stems of wheat, oats,

red fescue, and ryegrass about 45 days after they were planted in the inoculated sand media. Ascospores measured  $4\text{--}5 \times 105\text{--}163 \mu\text{m}$ . Smiley and Craven Fowler (11) recently reported a disease in New York whose symptoms and pattern of development closely resemble those of NRS. On the basis of a comparison of the Wisconsin pathogen with their description, as well as with the fruiting structures of *Leptosphaeria korrae* Walker & Smith that Smiley provided, we believe the two are the same.

## DISCUSSION

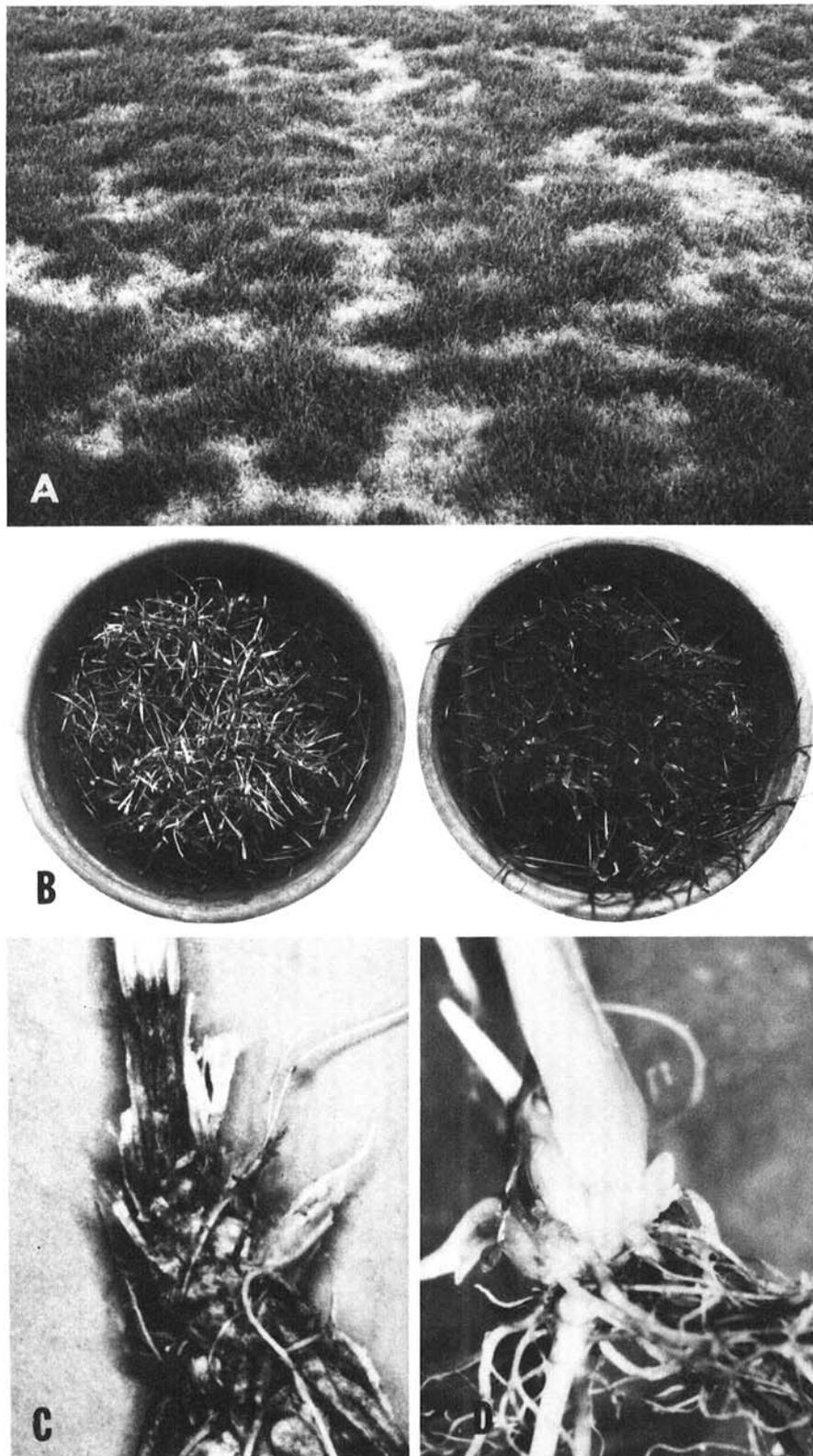
Successful isolation of ectotrophic fungi similar to NRSF is often difficult to accomplish (5); however, NRSF could be isolated on several media and with various tissue preparation techniques. The primary reasons for the initial difficulty encountered were the uncertainty over what the fungus should look like when it started to grow out of plated tissue and our early expectation that it was likely to be a *Rhizoctonia* or *Gaeumannomyces* sp. Its slow growth, particularly during the first 2 wk, and the overgrowth by faster-growing microorganisms also contributed to the problem. Novobiocin adequately inhibited most bacterial contaminants and did not interfere with fungal growth. NRSF apparently resists desiccation. Drying the sections for as long as 24 hr after surface-sterilizing also reduced contamination and improved the success of some isolation efforts. Consequently, once we developed this method, we were successful in isolating the fungus from most suspect samples. In 1982, it was isolated from 16 of 19 sites and routinely in subsequent years. Successful isolations from suspect turf were made every month from May through November.

NRS is a disease of mature turf. Consequently, emphasis was given to evaluating pathogenicity of the suspect fungus on mature sod plugs. Inoculating

**Table 4.** Crown and root responses of several grasses and cereals when planted in silica sand infested with the necrotic ring spot fungus

Host	Disease severity <sup>a</sup>	
	Infested sand	Noninfested sand
Kentucky bluegrass	2.9	0.3
Annual bluegrass	2.2	0.3
Bentgrass	2.8	0.3
Red fescue	2.9	0.3
Oats	1.6	0.5
Wheat	2.7	0.7

<sup>a</sup>Disease severity: 0 = crown and roots healthy and white; 1 = roots gray; 2 = roots black, but neither root nor foliage growth visibly reduced; 3 = root growth reduced with substantial cortical decay, and some foliage discoloration; and 4 = roots stunted, black and severely rotted, foliage chlorotic. Readings are an average of 50 plants, except oats and wheat were 60 plants each.



**Fig. 1.** Symptoms of necrotic ring spot disease: (A) Field symptom showing rings and serpentine pattern of distribution. (B) (Left) Collapse of inoculated sod plugs compared with (right) noninoculated plug 6 wk after inoculation. (C) Necrotic crown, buds, and base of tiller. (D) Healthy crown, buds, and base of tiller.

sod plugs produced both foliage and crown-root symptoms more typical of natural infections than inoculating seedlings. Sometimes, rapid symptom development resulted in seedling collapse, and typical field foliage discoloration was not observed. Infected crowns and roots became covered with a heavy mantle of fungal growth, and the mycelial aggregations observed in the field were present on crowns and roots of both older and younger plants. This was an important observation, because crown and root deterioration, combined with abundance of the ectotrophic fungus, is a distinguishing characteristic among the range of symptoms and conditions observed in the field. The autoclaved-grain inoculation techniques useful for *Gaeumannomyces* proved to be a satisfactory method for inducing symptoms on both seedlings and mature sod of several members of Gramineae.

The study involving temperature, moisture, and relative humidity may partially explain how variations in the range of field symptoms can occur. For instance, symptoms on inoculated sod plugs maintained with low humidity at 20 C were subtle and slow in their development. With high humidity at that temperature, control plants were extremely healthy, but inoculated plants collapsed nearly as rapidly as those maintained at 28 C, where the plants were under obvious heat stress. Therefore, higher temperature per se was not necessary for disease development; factors that contribute to fungal growth may be more important. We have observed that the fungus is present at trace levels in healthy turf of many types. The disease is much more likely to be severe in Kentucky bluegrass ranging from 2 to 8 yr old, after which the acute symptoms frequently disappear (18). This phenomenon is reminiscent of take-all patch decline (6), and similar factors may be at work with NRS.

Interaction with other organisms also may be important in contributing to symptom variations. Several new pathogens have recently been associated with diseased bluegrass (9). It is possible that one or more of them may contribute to the range of symptoms observed. *Fusarium* spp. were frequently isolated. We could not demonstrate their pathogenicity, and combination inoculations of NRSF with isolates of *Fusarium* we obtained or several other isolated fungi did not enhance disease development (*unpublished*). Moreover, the disease appeared on cultivars reportedly resistant to Fusarium blight (14), and ergosterol biosynthesis inhibitor fungicides, which were reported to control Fusarium blight elsewhere (3,12), were not effective in our trials (16). Yellow patch, a cool-weather disease incited by *Rhizoctonia cerealis*, has been reported to cause symptoms and produce fungal growth patterns on

bluegrass similar to these we observed (1,2,7). The similarity of fungal descriptions in relation to the host tissue suggests that both could sometimes be present and

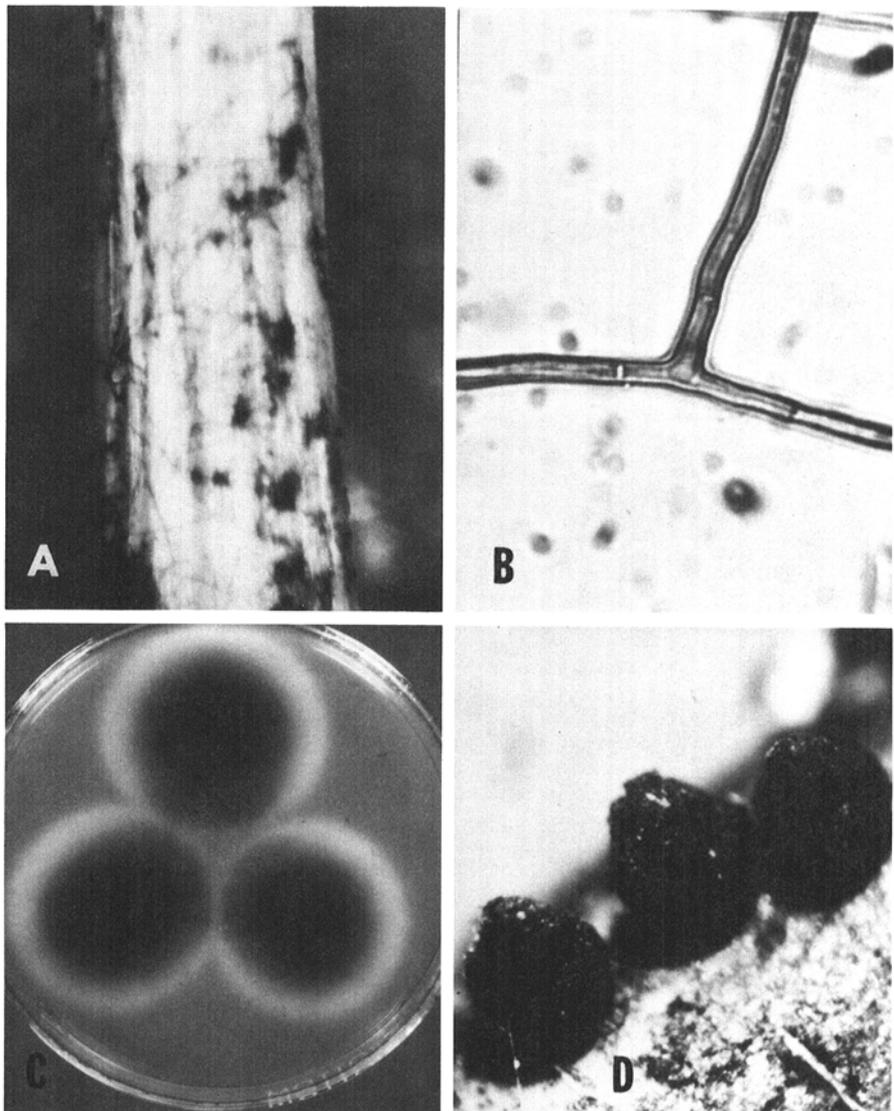
could contribute to variation in symptom expression as well as to fungicidal response (16). However, we isolated *R. cerealis* infrequently from the foliage and

**Table 5.** Effects of temperature on daily growth rates (mm) of five necrotic ring spot fungus (NRSF), three *Gaeumannomyces*, and two *Phialophora* isolates

Isolate	Source	Temperature (C) <sup>a</sup>							
		4	8	12	16	20	24	28	32
NRSF 10-20-2-32A	Annual bluegrass	0.4	1.2	1.9	2.6	3.9	4.3	4.7	0.0
NRSF 6-29-2-12A	Kentucky bluegrass	0.2	1.2	1.7	1.9	2.2	2.6	4.1	0.0
NRSF 10-21-2-5A	Kentucky bluegrass	0.3	1.1	1.8	2.0	2.8	3.9	2.7	0.0
NRSF 10-21-2-32A	Annual bluegrass	0.1	0.9	1.7	2.4	3.3	3.9	6.3	0.0
NRSF 10-22-2-31A	Kentucky bluegrass	0.3	1.2	1.7	2.1	2.4	3.9	2.7	0.0
<i>Gaeumannomyces</i> sp.	Bentgrass	0.8	1.9	3.5	7.5	10.7	13.2	14.3	0.0
<i>G. graminis</i> var. <i>graminis</i>	Wong <sup>b</sup>	0.0	3.8	5.2	7.9	11.0	11.4	7.8	6.9
<i>G. graminis</i> var. <i>avenae</i>	Wong	2.4	2.6	6.1	9.0	10.6	11.6	6.4	0.0
<i>Phialophora</i> sp. (lobed hyphopodia)	Wong	0.8	3.0	6.0	7.5	8.8	12.6	9.3	0.0
<i>P. graminicola</i>	Wong	0.2	1.5	3.3	3.6	5.9	6.2	4.1	0.0

<sup>a</sup>Mean of five replicates.

<sup>b</sup>Isolates provided courtesy of P. T. W. Wong.



**Fig. 2.** Vegetative and fruiting structures of *Leptosphaeria korrae*: (A) Rhizomorphs and plate mycelium on a leaf sheath of Kentucky bluegrass. (B) Dark-colored hyphae that average 3.6–4 μm in diameter and frequently produce right-angle branching. (C) A typical 15-day-old isolation from infected crown tissue. (D) Black, erumpent pseudothecia on oat seed planted and inoculated in sterilized silica sand.

rarely from the crowns of plants showing these symptoms.

It is interesting to note the taxonomic proximity of *Leptosphaeria* and *Gaeumannomyces* (15) as well as the similarity of disease patterns they create. Smiley and Craven Fowler (11) also identified *Phialophora graminicola* (Deacon) Walker as the incitant of another new turf disease, now called summer patch (10), which we have not observed. This study demonstrates *L. korrae* to be the causal agent of NRS. Dark runner hyphae are frequently observed on roots of many Gramineae including turf. More fungi are being reported as pathogens. Although we cannot assume that all such fungi observed are pathogens, their identity, role, and ecological relationship with one another and with their hosts have become important in turf pathology.

#### ACKNOWLEDGMENTS

We wish to thank P. T. W. Wong, Agricultural Research Centre, Tamworth, N.S.W. 2340, Australia, for fungal cultures; R. W. Smiley, Cornell University, for herbarium material; Harold Burdsall and Michael Larson, USDA Forest Products

Laboratory, for mycological consultation; Russell Spear for laboratory assistance; and Steve Vicen for photographic assistance.

#### LITERATURE CITED

1. Brown, C. L., Vargas, J. M., and Roberts, D. L. 1983. The occurrence of yellow patch on turf in Michigan. (Abstr.) *Phytopathology* 73:839.
2. Burpee, L. 1980. *Rhizoctonia cerealis* causes yellow patch of turfgrasses. *Plant Dis.* 64:1114-1116.
3. Burpee, L. L., Sanders, P. L., and Cole, H., Jr. 1977. Control of Fusarium blight under golf course conditions, 1976. *Fungic. Nematic. Tests* 32:153.
4. Couch, H. B., and Bedford, E. R. 1966. Fusarium blight of turfgrass. *Phytopathology* 56:781-786.
5. Davies, F. R. 1935. Superiority of silver nitrate over mercuric chloride for surface sterilization in the isolation of *Ophiobolus graminis* Sacc. *Can. J. Res.* 13:168-173.
6. Glynn, M. D. 1965. Crop sequence in relation to soil-borne pathogens. Pages 423-435 in: *Ecology of Soil-Borne Pathogens*. K. F. Baker and W. C. Snyder, eds. University of California Press, Berkeley.
7. Joyner, B. G., Partyka, R. G., and Larsen, P. O. 1977. *Rhizoctonia* brown patch of Kentucky bluegrass. *Plant Dis. Rep.* 62:749-752.
8. Scott, P. R. 1970. *Phialophora radicola*, an avirulent parasite of wheat and grass roots. *Trans. Br. Mycol. Soc.* 55:163-167.
9. Shurtleff, M. C. 1983. *Nigrospora* or *Rhizoctonia*? *Am. Lawn Appl.* 4(1):10-13.
10. Smiley, R. W. 1984. Characteristics of pathogens causing patch diseases of *Poa pratensis* in New York. (Abstr.) *Phytopathology* 74:811.
11. 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.
12. Smiley, R. W., Craven, M. M., and O'Knefski, R. C. 1977. Fungicides for controlling Fusarium blight of turfgrasses. *Fungic. Nematic. Tests* 32:151.
13. Tuite, J. 1969. *Plant Pathological Methods*. Burgess Publishing, Minneapolis. 239 pp.
14. Turgeon, A. J. 1976. Effects of cultural practices on Fusarium blight incidence in Kentucky bluegrass. *Weeds, Trees, Turf* 15:38-40.
15. Walker, J. 1981. Taxonomy of take-all fungi and related genera and species. Pages 15-74 in: *Biology and Control of Take-all*. M. J. C. Asher and P. J. Shipton, eds. Academic Press, London. 538 pp.
16. Worf, G. L. 1979. Control of a turf disease resembling Fusarium blight. *Fungic. Nematic. Tests* 36:163.
17. Worf, G. L., Avenius, R. C., and Stewart, J. S. 1982. A *Gaeumannomyces*-like organism associated with diseased bluegrass in Wisconsin. (Abstr.) *Phytopathology* 72:975-976.
18. Worf, G. L., Brown, K. J., and Kachadoorian, R. B. 1983. Survey of "necrotic ring spot" disease in Wisconsin lawns. (Abstr.) *Phytopathology* 73:839.