

1987 Report to USGA on Research on
Spring Dead Spot of Bermudagrass

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The project was completed in September 1987. The results are given in three different areas of emphasis.

I. Isolation of fungi that cause spring dead spot of bermudagrass

Plants of Tifway bermudagrass were collected in October from golf course fairways that had SDS symptoms the previous spring. Sclerotia and dark, septate mycelium of fungi were removed from stolons and placed on 0.25X potato dextrose agar to which 100 ppm streptomycin (0.25X PDAS) had been added. Plates were then incubated on a laboratory bench in both light and darkness. Fungi isolated grew slowly, requiring 10 to 14 days to cover 9 cm diameter plates. Three isolates were obtained from this process and are hereafter referred to as SDSA, SDSB, and SDSC.

Hybrid Tifway bermudagrass (Tifton 419) was inoculated with these fungi. Bermudagrass stolons were planted and grown on a greenhouse bench for 2 mo at which time complete grass cover was achieved in the pots.

Mycelium from the 0.25X PDAS was transferred to autoclaved oat (Avena sativa L.) seeds and allowed to grow for 1 mo. A 75 cc sample from each inoculated oat seed sample was then transferred to the root and shoot areas of bermudagrass plants in pots. These oat-inoculated Tifway bermudagrass pots were placed in a greenhouse for 1 mo and then placed outside in a sand box with the top of the pots at sand level from January to May 1987. Six

replications of the pots were placed outside to induce winter dormancy of the bermudagrass which is normally associated with SDS expression in North Carolina. In May, after ample green-up time had elapsed, pots were removed from the sand box and soil was dispersed by washing. Green shoots and roots ^{low} before the thatch layer were clipped, dried, and weights recorded. Statistical analysis of treatments were performed on shoot and root weights by analysis of variance. Two sets of the replications were saved and examined for fungi on the plants.

Tifway bermudagrass inoculated with SDSB produced symptoms similar to those seen on SDS affected field-grown bermudagrass. Root and shoot regrowth of inoculated SDSB plugs exposed during the winter were greatly reduced. Average shoot weight was 92% less for the SDSB inoculated pots as compared to the check. Root growth had a similar trend with SDSB inoculated pots having 71% less weight versus the check.

Similar dark septate mycelium and black disc-shaped sclerotia initially associated with SDS affected bermudagrass were observed and isolated from the SDSB inoculated pots. Symptoms of brown to black lesions associated with SDS affected Tifway stolons and roots were also observed. When placed on 0.25X PDAS, these sclerotia produced mycelium identical to that used to inoculate oat seed initially.

Tifway bermudagrass roots, stolons, rhizomes, and shoots from the field had the typical brown lesions that eventually coalesced to produce blackened, dead tissue. In the fall, the bermudagrass which had regrown over previous SDS spots had the black, septate hyphae growing throughout the roots, stolons, and rhizomes. Black disc-shaped sclerotia were located

periodically in these regions, especially underneath basal leaf sheaths. Regrowth on artificial media produced white mycelium which turned grey and eventually dark brown to black in color.

Upon further examination of inoculated plants, perithecia were seen in May following fall inoculation and were erumpent within basal leaf sheaths. Asci containing 8 ascospores emerged from the perithecia. Thickened lateral necks were associated with perithecia and cylindrical to clavate unitunicate asci (175 x 14 um) narrowed to form a foot-like base. Asci were widest in the middle, tapering towards the ends which were rounded. Eight ascospores that were hyaline and septate were produced in the asci. Ascospores measured (78) 94-125 (140) x 3.9 um which are shorter than those described for L. korrae [(120) 140-170 (180) x 4-5 (4.4 um)]. The type of ascus and the length of ascospores produced by the fungus isolated from bermudagrass and used in this study indicates that the fungus was not a Leptosphaeria sp. as reported from bermudagrass in California and Australia. Mycelium produced from single spore isolation was identical in appearance to that grown from sclerotia. J. Walker, mycologist with the New South Wales Department of Agriculture, Australia, L. F. Grand, and C. S. Hodges, both from North Carolina State University, identified the fungus as Gaeumannomyces graminis (Sacc.) v. Arx. & Oliver var. graminis. Gaeumannomyces spp. are associated with take-all of cereals which is similar in symptom expression and environmental requirements to SDS.

The causal fungi appear to grow most actively when temperatures are cool and the soil moist. Soil temperatures of 10-20 C are most favorable for the growth of Gaeumannomyces species. Bermudagrass root growth is extremely slow at 15 C, and most rapid at 35 C. Therefore, the fungus has

a distinct advantage in North Carolina from autumn to spring. Cold weather has been associated with the development of SDS of bermudagrass and may be the factor that actually kills the grass.

This is the first report of Gaeumannomyces graminis being associated with SDS of bermudagrass. The same fungus was identified in May from perithecia and ascospores on naturally-infected samples collected from North Carolina and Alabama. More observations of the perfect stage are needed to determine if this fungus is the cause of SDS throughout the southeastern U.S.

Table 1. Shoot and root bermudagrass weights 4 months following spring dead spot inoculation.

Isolates	Weights*	
	Shoots	Roots
Check	1.305 a	2.457 a
SDSC	1.114 a	2.160 ab
SDSA	0.586 b	1.438 bc
SDSB	0.105 c	0.719 c

*Means separation was by Waller-Duncan K-ratio t-test at the 1% level.

II. Control of spring dead spot with fungicides

Ten treatments were applied to bermudagrass fairways in the fall of 1986 where spring dead spot occurred in the spring of 1986. Four test sites were used and areas were located in Alabama, North Carolina, and Virginia. Preliminary results were obtained in the spring of 1987 and the tests have been established again in the fall of 1987 to obtain final results in the spring of 1988. Rubigan at 1 ounce per 1000 square feet in

September, and Tersan 1991 at 6 ounce per 1000 square feet gave best control of SDS.

III. Effect of fungicide treatments on winter hardiness of bermudagrass

Previous research on spring dead spot control has indicated the involvement of cold weather in disease development and protection from cold damage by the fungicide Tersan 1991. Fungicide and fertilizer treatments were applied to Tifway bermudagrass plots in the fall of 1986. Plugs of turf and soil were taken from the treatments in the winter and placed in a controlled temperature cold chamber. The plugs were exposed to different temperatures near or below freezing and then were placed in a greenhouse for regrowth. Preliminary data confirmed that the fungicide Tersan 1991 increased the cold hardiness of bermudagrass by a few degrees. This experiment is being repeated in the fall of 1987 and we will have final results in the spring of 1988.

Fighting Spring Dead Spot

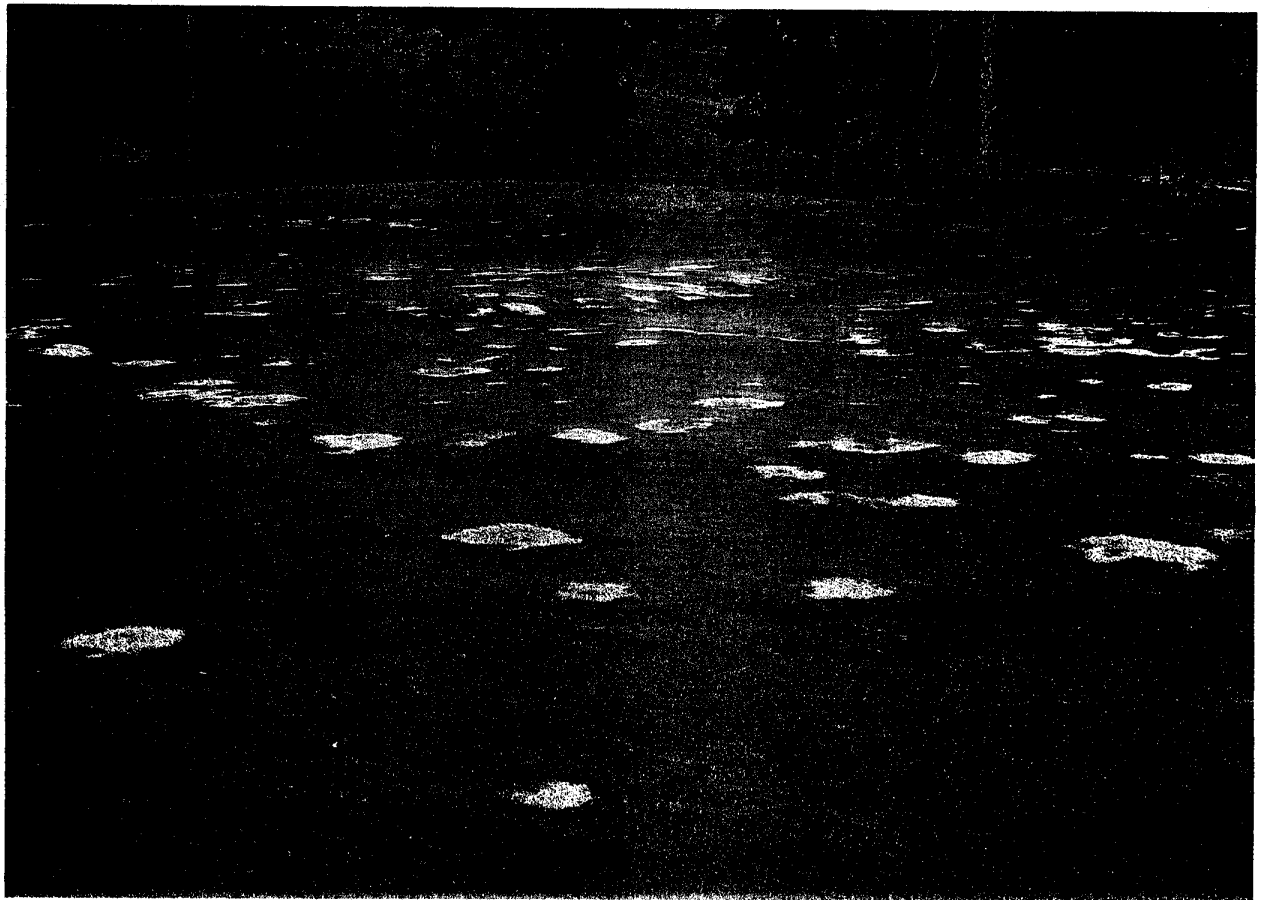
Researchers suggest reduced late summer nitrogen application, minimizing thatch and maintaining adequate soil potassium levels, along with strategic fungicide treatments.

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A fairway planted in Tifway bermudagrass shows the symptoms of spring dead spot. The disease causes circular dead spots to appear as bermudagrass begins to green up from winter dormancy.

Spring dead spot (SDS) of bermudagrass was first described in the 1950s in Oklahoma and has since been observed throughout the South and in

Kansas and California. The disease, which also has been seen in Australia and Japan, occurs in almost any area where bermudagrass grows and winter

temperatures are conducive for bermudagrass winter dormancy.

In general, severity of the disease in

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the spring has been related to severity of the previous winter. Symptoms include circular dead spots from 6 inches to more than 3 feet in diameter

The grass in the dead spots has the typical straw color of dormant bermudagrass.

as bermudagrass begins to green up from winter dormancy. The grass in the dead spots has the typical straw color of dormant bermudagrass. But on closer examination, SDS-affected bermudagrass has black and rotted stolons, roots and rhizomes.

Early SDS symptoms include small elliptically shaped tan to black lesions on rhizomes and stolons that eventually coalesce. Under higher magnification, ectotrophic, dark-colored mycelium and flattened disc-shaped sclerotia can be observed on basal leaf sheaths and stolons. After the death of the bermudagrass in the spots, bermudagrass grows back slowly in the summer, and weeds such as crabgrass often invade the areas, further reducing the turf's recovery and aesthetic quality.

SDS usually is seen more often on higher maintained hybrid bermudagrass after heavy nitrogen fertilization and in the presence of excessive thatch layering. The spots usually appear three to five years after turf establishment, a delay that may be related to the time necessary to allow ample thatch to

develop.

SDS commonly occurs in many of the same spots yearly, enlarging for several years and then developing into rings during the third and fourth years. Afterwards the spots may disappear or develop in other areas. Environmental and cultural parameters that trigger disease expression and development remain unknown.

Cause Of SDS

The cause of SDS has been almost as much a mystery as to why and where it occurs. Investigators in Australia reported in 1972 that the ascomycetes *Leptosphaeria namari* and *L. korrae* were the fungi that cause SDS. For years scientists in the United States could not isolate either of these organisms, thus compounding the frustration of working with an unknown causal agent.

In 1984, workers in California reported *L. korrae* associated with SDS of Tifgreen bermudagrass. Similar fungi have been isolated recently by the authors in North Carolina. Pseudothecia (spore housing sacs) of these fungi containing asci and ascospores were found within basal leaf sheaths of Tifway bermudagrass. Typical SDS symptoms were produced on bermudagrass inoculated with these fungi and grown in clay pots outdoors during the winter in North Carolina. Knowing the causal agent of SDS in the southeastern United States will allow more efficient and effective screening for control and investigation into disease development.

Chemical Control Options

Chemical control options for SDS are

few and expensive but somewhat effective. Consistent control has been achieved in North Carolina using 6 to 8 oz./1,000 sq. ft. of benomyl (Tersan 1991) applied in October or November. Treated areas may remain slightly darker green in the fall, and spots that do develop usually recover quicker the following summer as compared with non-treated areas.

These observations have indicated that benomyl may increase the winter hardiness of bermudagrasses in addition to functioning as a fungicide. Bermudagrass treated with 1 oz./1,000 sq. ft. of benomyl in mid-October resulted in increased regrowth of plants sampled during the winter even after plants were artificially exposed to -3 C for four hours and -5 C for five hours.

Recently, fenarimol (Rubigan) has received a label for SDS control. The manufacturer suggests application as 1 oz./1,000 sq. ft. in mid-September, or 1½ oz./1,000 sq. ft. in mid-October or 2 oz./1,000 sq. ft. in mid-November. The best control with this fungicide in North Carolina, Tennessee and

Some cultural practices have been found to reduce the severity of the disease.

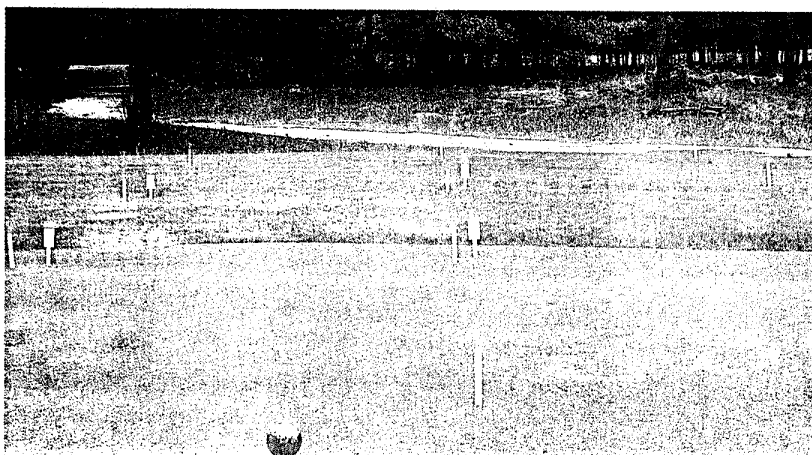
Alabama was obtained by using the lower rates in mid-September through early October. The timing and rates for optimal control may vary with geography and seasonal environmental conditions; therefore, the authors suggest experimenting with all three application dates and rates for your area.

Effects Of Cultural Practices

Cultural practices that have reduced disease severity include applying minimum nitrogen rates, especially late in the season. Nitrogen stimulates top-growth at the expense of roots and also decreases winter hardiness as plants remain green and succulent longer into the fall and winter seasons. Research in North Carolina indicates SDS severity increased up to 50 percent when 1 pound of nitrogen was applied per 1,000 square feet in September and October over those areas not receiving any additional nitrogen.

Minimizing thatch also reduces disease severity. Other cultural practices

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Spring dead spot control with benomyl at 8 oz. per 1,000 sq. ft. applied in October (left) contrasts with an untreated plot.

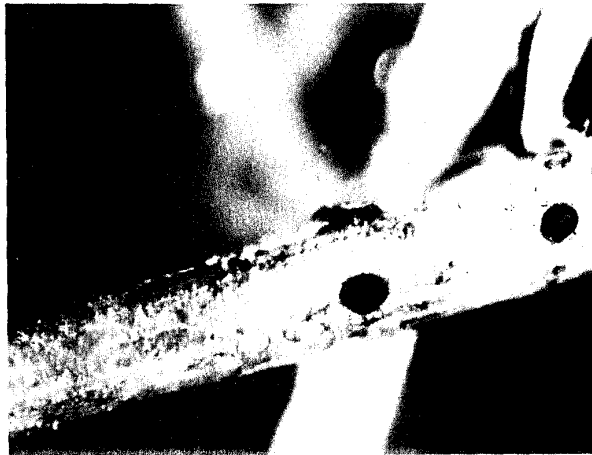
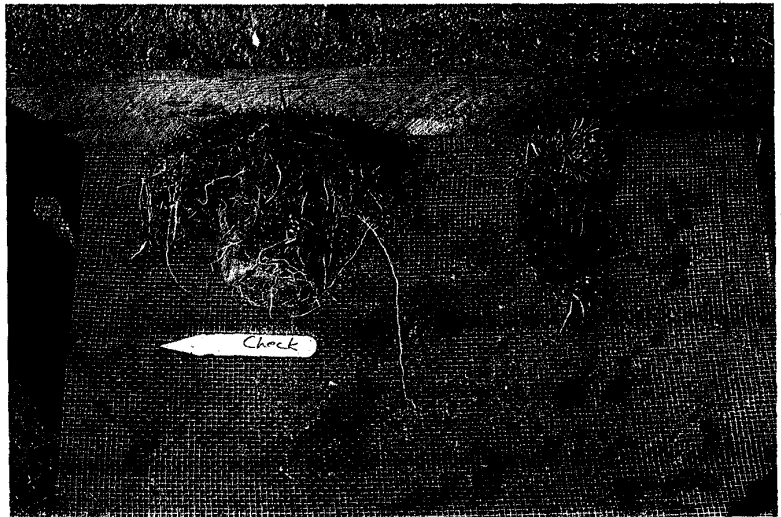
SPRING DEAD SPOT from p. 62

suggested for better SDS control involve those normally recommended to reduce damage from cold temperatures, including maintaining adequate water availability in early spring when the bermudagrass is initiating green-up and maintaining adequate soil potassium levels. Ample potassium is necessary for plants to endure such stresses as low temperatures.

In conclusion, a fungus similar to *L. korrae* has been isolated from bermudagrass with SDS in North Carolina, and reinoculation resulted with disease on bermudagrass during the winter. Bermudagrass grown under high nitrogen fertilization with excessive thatch accumulation is usually more susceptible to the disease.

Suggested cultural practices for control include reduced late summer nitrogen application, preventing excessive thatch accumulation and maintaining adequate soil potassium levels. Supplement these management practices on areas that had the disease the previous spring with application of benomyl at 6 to 8 oz./1,000 sq. ft. or fenarimol at 1 to 1½ oz./1,000 sq. ft. in mid-September to mid-October.

(The financial support for this research was generously provided by Hall Thompson, president of the Shoal Creek Country Club in Birmingham, Ala., and by the USGA Green Section. Rubigan is a registered trademark of Elanco Products Co., a division of Eli Lilly and Company.)



The top photo shows the spring shoot and root re-growth of SDS-inoculated Tifway bermudagrass compared with a healthy plug. The bottom photo shows mycelium and sclerotia growing on SDS-affected bermudagrass stolons.

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