# Transmission of Spring Dead Spot Disease of Bermudagrass by Turf/Soil Cores

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**ABSTRACT**

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Transmission of spring dead spot disease of bermudagrass (*Cynodon dactylon*) was accomplished with turf/soil cores taken in 1973. Cores from edges of dead turf patches in a bermudagrass lawn were transplanted into 24 established bermudagrass clones in an area where the disease had never been observed. Two to 4 yr were required for symptom expression. Once symptoms appeared at a given transplant location, spring dead spot tended to recur in the same location in successive seasons, increasing in size in subsequent years. After 1977, no new sites developed the disease, but by 1982, the number of sites diminished because spring dead spot failed to recur at some sites and other sites converged as they enlarged. Symptoms appeared at 6.7, 19.9, 36.0, 35.6, and 25.5% of the 192 inoculation sites in 1975, 1976, 1977, 1978, and 1982, respectively. No disease occurred either at sites into which symptomless cores were transplanted or at otherwise random locations in or near the test plots. In addition to demonstrating transmissibility, this technique appears promising to screen bermudagrass clones for resistance to spring dead spot.

Additional key words: resistance screening

Spring dead spot of bermudagrass (*Cynodon dactylon* (L.) Pers.) was first described by Wadsworth and Young in 1960 in Oklahoma (9), but the disease may have been observed as early as 1936 (6). Symptoms are circular dead spots of bermudagrass ranging in diameter from a few centimeters to more than a meter in early spring as bermudagrass begins to break dormancy and turn green. During the growing season, the bermudagrass eventually may spread back into the dead areas. The disease usually does not occur on turf established for less than 3 or 4 yr; however, once it appears, spots may recur in the same location and become larger each year or decrease in size and eventually fail to recur. The disease has plagued most strains of bermudagrass, particularly hybrids, but also occurs in common bermudagrass. In general, spring dead spot is associated with cold winters and springs and with management factors that reduce hardiness, delay fall dormancy, or promote early spring greenup (5).

Etiology of spring dead spot is probably complex. Moderate control has been achieved with fungicides (2,6), indicating a fungal pathogen. A patch disease of *C. dactylon* in Australia with symptoms nearly identical to those of spring dead spot was reported by Smith in 1965 (7). He identified the causal fungus as *Ophiobolus herpotrichus* and proved pathogenicity. In a followup report in 1972, Walker and Smith (10) renamed the pathogen *Leptosphaeria korrae* and described another closely related species, *L. narmari*; both cause patch symptoms on several turfgrasses including bermudagrass. When inoculated onto cereals, these fungi produced symptoms similar to take-all caused by *Gaeumannomyces* spp. Recently, Endo et al (3) reported *L. korrae* causing spring dead spot of bermudagrass in California. Symptoms they described are very similar to those we observed in Kansas, but we do not know what pathogen was involved in our plots. We report for the first time that spring dead spot is transmissible under field conditions.

## MATERIALS AND METHODS

Field inoculation was used to determine susceptibility of bermudagrass to spring dead spot disease. The experimental area included two plots 3 × 3 m, each of 24 bermudagrass clones, which initially had been sprigged for observational purposes in June 1971 at the Horticulture Research Center in Wichita, KS. These clones included the cultivars Midway, Sunturf, Reno, and Midiron, and the numbered clones A-6, A-7, A-8, A-12, B-1, D-17, E-7, J-8, J-9, M-10, P-1, P-8, P-11, P-17, Q-12, R-4, S-7, S-15, T-3, and T-16. The soil is classified as a sandy loam, Canadian Waldeck series. However, half of the plots (one of two replicates) were located on a very sandy portion; the other half were planted on an area of higher clay content. Each season, plots received 1.8 kg N/are (1 are = 100 $m^2$) distributed monthly during the growing season. Mowing was at 2.5 cm and clippings were removed.

Half of each plot was inoculated on 8 October 1973 at 61-cm intervals with turf/soil cores 5.7 cm in diameter × 7.5 cm deep (soil = 5 cm, turf + thatch ≃ 2.5 cm) taken from the peripheries of spots in a Midway bermudagrass lawn that had shown symptoms of spring dead spot the previous spring. Each plot received four cores, for a total of 4 × 2 × 24 = 192 inoculation sites. A single control plug was removed from the center of the other half of each plot and replaced immediately to check for winter injury from the plugging operation. The temperature at the time of plugging was 21 C at 15-cm soil depth, and soil moisture was excellent for transplant survival. Observations were made for symptoms each succeeding spring.

## RESULTS AND DISCUSSION

No symptoms occurred the spring after inoculation, but by the spring of 1975, numerous diseased spots typical of spring dead spot were observed and all diseased spots were precisely at the plug transplant locations.

Some bermudagrass clones showed symptoms for the first time as late as 1977, almost 4 yr after the initial inoculation. No symptoms developed within uninoculated halves of plots or away from the sites of core transplants in inoculated halves of plots during 1975–1978. Percentage of disease did not change appreciably from 1977 to 1981, although during this period, some spots continued to increase in diameter while others became smaller. By 1982, some large spring dead spots began to overgrow each other and the control sites, although the disease also failed to recur at a few locations where it previously had occurred. These dead spots were not due to dieback of the grass colony from the original plug. In fact, there was no indication of any plug of Midway

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surviving the first winter when plugged in October. Midway is a dark green, fine-textured cultivar that could have readily been identified growing among clones of different color and texture.

For the hypothesis that inoculations were not responsible for observed differences in disease compared with uninoculated sites, two comparisons were made. For the first, spring dead spot symptoms at inoculated core sites were compared with noncored, uninoculated locations in the opposite plot halves; a total of 192 inoculated sites and 192 uninoculated sites were compared. For a more conservative test of the hypothesis, the 192 inoculated core sites were compared with the 48 uninoculated core sites originally included for use in winter injury determinations. This hypothesis was rejected by chi-square analyses for each year. For the first test of hypothesis, the chi-squared $\chi^2 = 26.91$, 39.98, 81.67, and 83.12 were found for 1975, 1976, 1977, and 1978, respectively. The chi-squared test in the second method of comparison was $\chi^2 = 4.27$, 11.98, 24.68, and 25.18 for 1975, 1976, 1977, and 1978, respectively. $\chi^2$ (1 df) $\geq$ 10.827 and $\geq$ 3.84 would be expected with a probability of less than 0.001 and 0.05, respectively. Thus we concluded spring dead spot disease did result from our inoculations. Excluded from these comparisons were the 1982 data because of the apparent spread of the disease over various neighboring inoculated and uninoculated sites.

Symptoms in the field occasionally subside or disappear several years after appearance or even after the disease worsens for a period of years (11). Such a phenomenon may have occurred in our test because inoculation sites in certain clones varied in symptom expression from year to year. In particular, spring dead spots at some sites in 1977 and 1978 became smaller in diameter by 1982, and some spring dead spots later failed to appear altogether after appearing for several years. In contrast, numerous other spring dead spots continued to reappear and increase in size each year. Ultimately, spring dead spot sizes ranged from 15 to 75 cm in the various plots and among clones.

A degree of clonal resistance to the disease may exist, but statistical inferences cannot be drawn from only two replicates per clone. A-6, Midiron, and P-1 were slow to develop spring dead spot symptoms. The cultivar Midiron has been observed to be quite resistant to the disease on golf courses in Kansas. Midiron also showed only small spots in 1977 and 1978 and did not have readily identifiable spring dead spot symptoms in 1982 although winterkill was more severe than in any previous year. Midiron has been shown to be among the hardiest bermudagrass clones available (1) but is often slow to turn green in the spring. All other clones except Sunturf eventually showed disease symptoms. Sunturf (*C.* × *magennisii* Hurcombe) is a dense, dark green, low-growing African cultivar (2). Midway, a hardy, open-growing, dark green, medium-textured cultivar (4), Midiron, a more aggressive, athletic field type, and most other numbered clones are hybrids (*C. dactylon* × *transvaalensis*) produced in a polycross nursery.

Although we could not distinguish relative resistance among cultivars, we believe such differences do exist both in the appearance of spots and in the size of spots. A severity index was calculated by rating the sizes of dead spots on a scale of 0–9 and multiplying by the number of spots in each plot. Although one plot of clone S-15 in 1978 showed spring dead spot in all four areas plugged, the small size of diseased spots resulted in a rating of only 16, whereas the combined four dead spots in both plots of clone T-3 produced a rating of 30 because the spots were larger. Such a severity index, perhaps using spot diameters instead of an arbitrary rating, might be used in future experiments to determine cultivar susceptibility to spring dead spot.

More dead spots were observed in plots in the heavier soil than in the sandier soil of the experimental area. This seems to support an earlier report that soil type may influence spring dead spot (12).

The spring dead spot disease of bermudagrass can be transmitted successfully with diseased turf/soil cores. Our data support other evidence (3,7,8,10) that the disease is infectious. Differences in susceptibility to the disease among bermudagrass clones appear real, but demonstration of statistical differences would require a larger number of replicates.

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