UPDATING 20 YEARS OF RESEARCH:

Spring Dead Spot

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Spring dead spot is the most important disease of bermudagrass, so say the turf researchers of the southern land grant institutions. There is some evidence that this disease has been affecting turf since 1936. These reports came from the transition zone but evidently SDS was not too great a problem before 1954. It is surmised that the reasons for its lack of importance at that time may be two-fold. First, the management of fine turf had not yet reached a high level of sophistication, and, second, the hybrid and other selections of bermudagrasses had not yet made their appearance.

The first research with this disease was by D.F. Wadsworth and H.C. Young, Jr. of the Oklahoma State University in the spring of 1954. In 1960 these workers were the first to describe the symptoms, host, range, probable causal organisms, and attempted controls of the disease. It is essential to point out that what these workers said about SDS at that time is still basically true today. For example, it was observed only on bermudagrasses, not associated with any one type of soil or topography, observed only under conditions of management producing high quality turf, not reproducible or controlled consistently in any tests, and the true cause unknown.

At about the same time, W.A. Small, of Mallinckrodt Chemical Works, conducted trials with a number of fungicides and other chemicals in an attempt to find a means of control. From these efforts came the program of the application of 85 percent nabam (disodium ethylenebisdithiocarbamate) four times at monthly intervals beginning at least six weeks before the average killing frost date in the fall.

SDS was present on bermudagrass golf greens in Georgia as early as 1960, but oddly enough, it was not golf turf that gave the Georgia program its impetus. In 1962, a building boom had set in in the city of Atlanta as a result of which, in 1965, complaints came from homeowners who had purchased homes during that boom. The hybrid bermudagrass lawns of these homes were now severely affected by SDS. From the complaints came the establishment of a research project to study SDS in all of its aspects. The project was initiated in the Department of Plant Pathology & Plant Genetics at the University of Georgia with the author as project leader and with workers in other departments and stations cooperating.

The Tifway (419) bermudagrass lawn of the City of Athens, Georgia, Sewage Disposal Plant was severely affected by SDS and provided an excellent test site for studies of fungicides and of cultural methods for control of SDS. For four years, 1965-1969, we tested a large number of fungicides. Our criterion for control was the reduction or elimination of the spots and on this basis no one chemical was found that consistently controlled SDS when sprayed or drenched into bermudagrass in late summer or fall. However, in a test on a Tifgreen bermudagrass golf green, five fungicides reduced the number of spots over a two-year period with no re-appearance of spots in treated plots the third year. This points out the irregularity of results associated with SDS research. Concurrently with the fungicide tests, we conducted trials to determine the feasibility of core aerification and/or vertigrooving, complete turf renovation, liming, soil removal and replenishment, on SDS. One of these test sites is shown in Figure 1. Only complete renovation by rototilling to a depth of 12 inches reduced the amount of spots permanently. All other treatments were inconsistent from one year to the next.

Simultaneously, we surveyed the fungi and nematodes associated with the roots of grass affected by SDS. Many parasitic and saprophytic species of fungi were found, the most important being the *Helminthosporiums, Pythiums, Fusariums,* and *Curvularias.* Isolates from these were inoculated onto Tifway and Tifgreen bermudagrass either singly or in combinations in the greenhouse and field, but we were unable to induce any symptoms of the disease. At least five genera of nematodes were found, but they were present only in extremely low numbers and only in a few instances. Thus, we had to assume that these played no role in SDS development. We also determined that myco-

on Bermudagrass

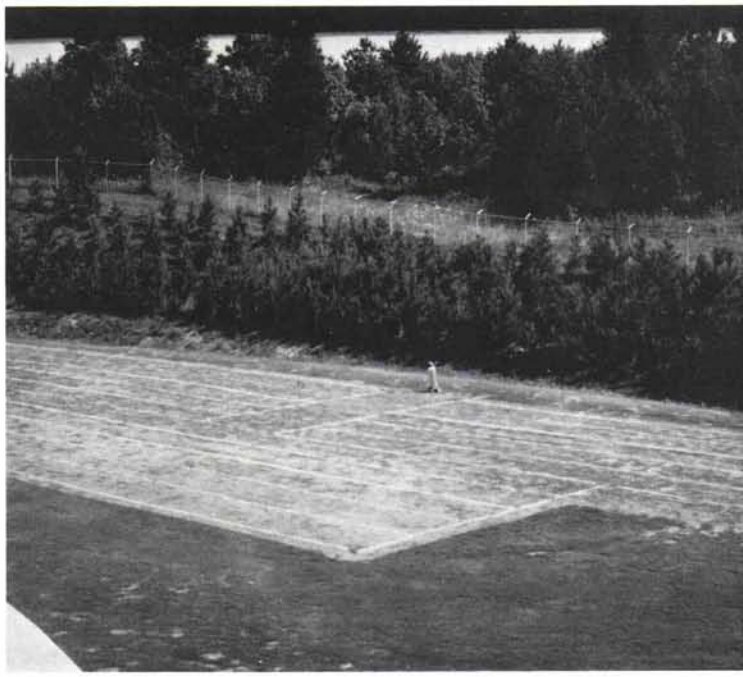

Figure 1. Test site for cultural control of SDS, one year after initiation. The high incidence of spots all over the area is clearly evident.

plasma (virus-like organisms) were not causal agents.

Since the counting of spots was an unreliable indicator for control, it became necessary to re-evaluate our hypothesis. We adopted a rating system based on necrosis (rotting) of roots, rhizomes and stolons. This is proving to be a more reliable and accurate indicator of the activity of SDS. Counting of spots, at times, supplements the rating system.

By now we were led to adopt the hypothesis that we were dealing with a weak parasite whose effect on the host was subtle, occuring in the summer-time too late to be affected by late summer application of fungicides, but such that a severe winter would bring about death of grass. If this hypothesis were sound, then prevention of this weak parasitism could be accomplished by applying the fungicides as the grass emerged from dormancy. Since our work with fall applications of fungicides revealed no favorable data, we decided to apply fungicides in the spring. We established a test in 1970 on an infected Tifway bermudagrass golf course fairway and applied fungicides on a monthly basis beginning in March. Data for 1970 and 1971 show that necrosis due to SDS was reduced substantially by two fungicides, captan and terrazole, but that most others could bring about some reduction as well. Interestingly, the number of spots did not appear to be reduced in the two years, but the test area had no SDS the third year.

The new rating system allowed the initiation of studies to determine the actual time of death of bermudagrass affected by SDS. Four years data (1969-1972) show that the grass dies in the winter (January-February) but the amount of grass that dies is dependent on the severity of the winter. Complete weather information for this period is collected, and once it is fully analyzed and correlated with the necrosis data it should provide, an accurate picture of how this disease complex works.

INTERESTING FILTRATES

Previous research in Arkansas by Diaz and Dale in 1964 and in Oklahoma by McCoy and Young in 1968 showed that filtrates from *helminthosporium spiciferum* could induce rotted root systems on bermudagrass in the laboratory. Our work in this vein was designed to obtain information on root rotting under as natural conditions as possible. At first we subjected SDS-infected bermudagrass sod (never completely or wholly dead) and passed water through it, doing the same with unaffected sod. The leachates thus collected were passed through 3-year old "healthy" sod which had been growing in 6-inch pots in the greenhouse. The results showed a subtle reduction of topgrowth, as evidenced by clipping weights, attributed to the leachate. We have now taken SDS-affected and not-affected soils and leached them under controlled temperatures. Using annual ryegrass germinating seedlings in rag dolls, we found that the leachate apparently contained a toxin, or toxins, which seemed to substantially reduce the respiration rate (as

Figure 2. Top Crop Bush Beans. Stems have become necrotic and are broken in addition to being retarded in growth.

Figure 3. Bragg Soybeans. Some necrosis has occurred and yellowing and retardation are evident.

evidenced by growth). A second series of the same tests apparently corroborates the findings of the first, and a third series is currently underway. The source of the toxin(s) is not yet known. The toxin can be a metabolite of a fungus, of the fungus and host, or from dead plant material (thatch). The need to identify the toxin is paramount because we feel that such identification will answer the most important questions about SDS.

It would be highly desirable to have a grass that could be seeded into the spots, flourish, and blend in to give a pleasant appearance. Our greenhouse work shows that certain plants, when seeded into SDS soils are stimulated in growth during the two weeks after seeding. These are common bermudagrass, Pennfine perennial ryegrass, Pencross bentgrass, Golden Cross Bantam sweet corn, Rogers barley, Bragg soybeans, Top Crop beans, and Yellow Straightneck squash. These plants do not exhibit such stimulation when grown in soil from the same site but which is unaffected by SDS. Thereafter, however, the trend is reversed and retarded growth occurs in SDS soils. Growth in unaffected soil is normal. Figures 2 and 3 are illustrations of the abnormal growth in two legumes. Aerial stems (stolons) of grasses sprigged into SDS and non-SDS soils also show retardation in the SDS soils. Among those tested were: Tifway, Tifgreen, Tifdwarf, Tufcote and common bermudagrasses, Meyer and Emerald Zoysiagrasses and Pencross bentgrass. The most flagrant exception to the behavior described is volunteer *Poa annua* which exhibits extraordinary growth in SDS soils over that in non-SDS soils. This is shown vividly in Figure 4.

INTERESTING GYPSUM

At the present time, we are looking at an interesting aspect. We have a little evidence that gypsum ($CaSO_4$) may have been instrumental in reducing SDS in the field. Work in the greenhouse with gypsum incorporated into a clay soil and into a sandy loam soil at four different percentages by volume, using healthy U-3 and Tifway bermudagrasses, showed that the pH of soil and availability of calcium and magnesium increase with the higher concentrations of gypsum but that phosphorus and potassium decrease as the gypsum concentrations increase. Weights of the first clipping were greatest in both the clay and sandy loam 1 percent (least gypsum) amended soils. Two other clippings have altered this picture. Now, weights for three clippings in the clay soils are greatest in the un-amended soil, whereas the weights in sandy loam soil remain greatest at the 1 percent level. This apparently is another irregularity which keeps arising in SDS research and we have no explanation for it at this time. We intend to progress into the incorporation of gypsum into actual SDS soils.

SDS usually appears the third year after establishment of bermudagrass where the grass is managed at a high level of maintenance, but this may not be necessarily binding in each case. It has also been said that soils high in organic matter may be free of SDS. We have worked with a sward that has been SDS-free for five years, its soil amended with high amounts of sewage sludge (direct from the drying beds), with fungicides applied, and yet severely affected with SDS the sixth year, and thereafter for two years. It has also been assumed that SDS is confined to the heavier soils. Georgia's first

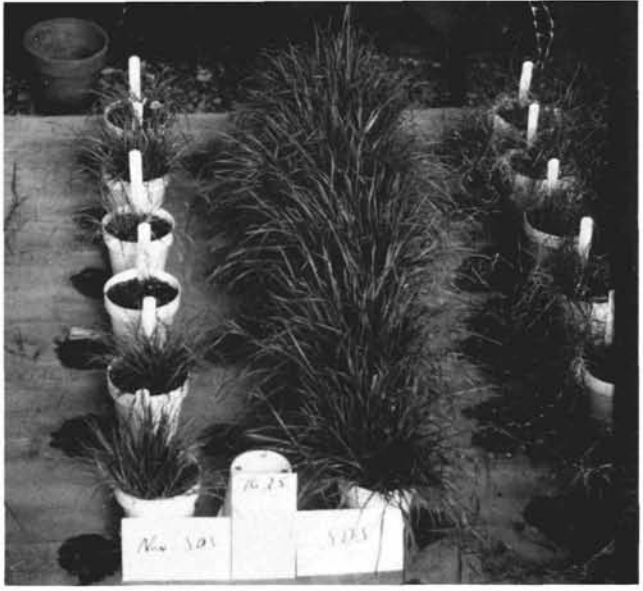

Figure 4. Volunteer Poa annua. *Extra-ordinary growth in SDS soil on the right is evident. Number of plants in each soil at the beginning of the experiment was the same.*

Figure 5. SDS from tee to green on a golf hole composed of sandy soil.

SDS report on golf turf came from middle Georgia from sandy greens. Figure 5 shows a heavily infested fairway from a golf course located in the sandy coastal plain of North Carolina, the picture taken in May 1973.

SDS is a disease which remains unsolved today. However, a great deal has been learned about the disease which will ultimately lead to a fuller understanding of it. On the basis of our work and observations we believe SDS to be a disease (root rot) of bermudagrasses which appears because the grass is predisposed to one or a group of fungal organisms (saprophytes, weak parasites, or parasites) by factors of management. We feel that the best means of control is preventive maintenance; i.e., the use of sound principles of turf management. We propose the following:

1. Apply only enough nitrogen to maintain the grass for play or other intended use. Any nitrogen over that amount may result in SDS.

2. Keep thatch at a minimum by not overfertilizing. Use the verticut and other equipment and methods to control the thatch. Sensible topdressing goes a long way toward keeping thatch under control.

3. Prevent compaction by routing traffic and aerify when compaction does occur.

4. Sensible use of water is absolutely necessary.

5. Use a preventive schedule of fungicides for the control of all turf diseases. If you have SDS, we feel that most fungicides will reduce it in time, but only when applied at the right time. Our work shows that time to be early spring into summer.

ABOUT THE AUTHOR:

George M. Kozelnicky has been with the University of Georgia since 1951. He received the B.S. and M.S. in agriculture from that institution, majoring in plant breeding and plant pathology. Since 1961 he has been researching turfgrass diseases.