

EXECUTIVE SUMMARY FOR 1998

Project Title: The Biology and Management of Spring Dead Spot in Bermudagrass

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Project Summary

Spring dead spot (SDS) is a serious disease of bermudagrass along the northern range of its adaptation in the USA. Three distinct root-rotting fungi called *Ophiosphaerella herpotricha*, *O. korrae*, and *O. narmari* (formerly *Leptosphaeria korrae* and *L. narmari*) cause this disease. The purpose of our research is to learn more about the distribution and biology of these SDS pathogens, and based on this understanding, to develop more effective strategies for managing this disease.

Diseased bermudagrass stolons and roots were sampled from golf courses in Kansas, Oklahoma, Mississippi, Alabama, Virginia and Kentucky. *O. herpotricha* and *O. korrae* were recovered from samples in all states, with *O. herpotricha* being more abundant in the Great Plains region and *O. korrae* more abundant in the eastern United States. *O. narmari*, previously reported only in Australia, was detected for the first time in North America from samples collected in Oklahoma and Kansas.

Little is known about the population structure of SDS pathogens on a local and regional scale. We studied the inter- and intra-specific genetic diversity of *Ophiosphaerella* isolates from North America and Australia. The population of *O. herpotricha* at a given location appears to be a mixture of many different individuals. In contrast, just a few distinct clones dominate the population of *O. korrae* in a location.

Field and greenhouse studies are being conducted to evaluate the resistance of seed- and vegetatively propagated bermudagrass selections to spring dead spot. Field trials in Oklahoma indicated that several bermudagrass entries including Guymon, Sundevil, Midlawn, Midfield, Ft. Reno, and Mirage and OKS 91-11 were more resistant to spring dead spot. We are currently developing greenhouse and laboratory methods to more rapidly screen bermudagrass selections for disease resistance. Furthermore, we are determining whether there are differences in pathogenicity to bermudagrass selections among the three SDS pathogens. Preliminary evidence suggests that *O. herpotricha* results in larger dead spots and more shoot kill within the spots than the other spring dead spot pathogens.

Various cultural and chemical control strategies have been proposed to control spring dead spot. We established a trial 1998 to evaluate the effects of some of these control recommendations, alone and in combination, for suppression of SDS. Preliminary results indicate that aggressive summer aeration accompanied by fungicide and growth regulator treatments will reduce, but not eliminate symptoms of spring dead spot.

Results for 1999 and Plans for 2000

Distribution of SDS pathogens. Our objective is to determine the distribution of the three pathogens associated with SDS. Because these fungi are extremely difficult to identify by traditional diagnostic techniques, we initially had to develop techniques for rapidly identifying these pathogenic fungi. We previously had developed species-specific DNA oligonucleotide primers to identify *O. herpotricha* and *O. korrae* but more recently Wetzal et. al. (1999) developed primers for identification of *O. narmari*. These primers have been successfully tested and are capable of differentiating the three SDS pathogens relatively quickly.

Surveys in Kansas and Oklahoma in 1996 indicated that *O. herpotricha* was the only pathogen associated with spring dead spots on two intensively sampled golf courses (>200 samples/course). On a third course, *O. herpotricha* was the most abundant pathogen, but *O. korrae* and *L. narmari* were also present, sometimes within a few meters of one another. This was the first time *O. korrae* was isolated from bermudagrass in the southern Great Plains and it was also the first report of *O. narmari* in North America (Wetzal et al 1999).

In 1999, approximately 20 SDS samples were collected from bermudagrass fairways on golf courses in Virginia, Alabama and Mississippi. *O. korrae* was the most frequently isolated fungus at all locations. Nevertheless *O. herpotricha* was recovered from a golf course in Virginia. This suggests that *O. korrae* is the primary SDS pathogen in the southeastern and eastern United States whereas *O. herpotricha* is most abundant in the Great Plains. In 2000 and 2001, we will continue to sample for SDS pathogens. We will intensify sampling in the southeastern (North Carolina, South Carolina, and Georgia) and western (Texas, New Mexico, and California) regions of the United States.

Genetic diversity and aggressiveness of SDS pathogens. Bermudagrass plots at two locations in Kansas and one location in Oklahoma (cooperation with D. L. Martin, Oklahoma State University) were inoculated in 1997 with isolates of *O. herpotricha*, *O. korrae*, and *O. narmari*. Necrotic patches associated with *O. herpotricha* were significantly larger than those caused by *O. korrae*. Isolates of *O. narmari* failed to cause symptoms of SDS in 1999. Further inoculations are planned in 2000 to determine the relative aggressiveness of each SDS pathogen. This information should help ascertain which fungi need to be used in screening for SDS resistance in new bermudagrass selections.

Amplified fragment length polymorphism (AFLP) marker analysis was used to investigate inter- and intraspecific genetic diversity of *Ophiosphaerella* isolates from North America and Australia. A majority of the *O. herpotricha* and *O. narmari* isolates from Afton Oklahoma were distinct haplotypes, suggesting that sexual recombination was occurring within the population. Conversely, the presence of multiple isolates of *O. herpotricha* and *O. narmari* with the same haplotype indicated that asexual propagation, most likely from movement of fungal mycelium on bermudagrass sprigs, was also occurring. The genetic diversity among *O. herpotricha* isolates from Afton was not distinctly different from isolates collected throughout the southern United States. In contrast, *O. narmari* isolates from Afton were distinct from those collected in

Australia. The genetic diversity in *O. korrae* was markedly different than the other *Ophiosphaerella* spp. The population at Afton was dominated by just a few haplotypes, and these were nearly identical to isolates collected from bermudagrass and Kentucky bluegrass throughout western, central, and northern North America. However, *O. korrae* isolates collected in the southeastern United States were only distantly similar to other North American isolates. These southern isolates had higher growth rates on potato dextrose agar at temperatures ranging from 15-30 C. It is not known whether these southern isolates represent a more aggressive population.

Screening bermudagrass selections for resistance to SDS. Field and greenhouse studies were conducted to evaluate the resistance of seed and vegetatively propagated bermudagrass entries to spring dead spot disease caused by *O. herpotricha*. In Kansas greenhouse studies, *O. herpotricha* caused root discoloration and root weight reductions in all entries tested. However, in Kansas field plots, root weight reductions were not different among entries and were not correlated with disease severity ratings. In Oklahoma trials that were inoculated with *O. herpotricha*, Guymon, Sundevil, Midlawn, Midfield, Ft. Reno, Mirage, were most resistant to spring dead spot. Several experimental seed-propagated entries including OKS91-11 also showed resistance to SDS. Severity of spring dead spot among bermudagrass entries was correlated with freeze injury that occurred during the first winter after planting.

A more rapid, uniform inoculation technique must be developed to screen large numbers of bermudagrass genotypes in the greenhouse. Past attempts to correlate greenhouse and field inoculation studies have been unsuccessful (Baird et al., 1998). We are working on the development of a rapid screening technique for SDS.

Integrated turfgrass management for control of SDS. A field plot for studying the effects of various cultural and chemical practices was established in the summer of 1998. The experimental design was a split plot with intensive soil aerifications in June and July) as main plots and fertilizer, fungicide and growth regulator combinations as subplots. Preliminary results indicate that aggressive summer aerification programs and other treatment combinations significantly reduce SDS severity. Further studies are planned for 2000.

Graduate Students Associated with USGA Project

Fanny Iriarte: Ph.D. candidate

Abstracts and Publications Resulting From USGA-Funded Research

Baird, J. H., D. L. Martin, C. M. Taliaferro, M. E. Payton, and N. A. Tisserat. 1998. Bermudagrass resistance to spring dead spot caused by *Ophiosphaerella herpotricha*. Plant Disease 82:771-774.

Martin, D. L., G. E. Bell, J. H. Baird, C. M. Taliaferro, N. A. Tisserat, R. M. Kuzmic, D. D. Dobson, and J. A. Anderson. 200_. Spring dead spot resistance and visual quality of seeded common bermudagrasses under differential mowing heights. Crop Sci. (submitted)

Tisserat, N., H. Wetzel III, J. Fry, and D. L. Martin. 1999. Spring dead spot of buffalograss caused by *Ophiosphaerella herpotricha* in Kansas and Oklahoma. Plant Dis: 83:199.

Wetzel III, H.C., S. H. Hulbert, and N.A. Tisserat. 1999. Molecular evidence for the presence of *Ophiosphaerella narmari* n. comb., a cause of spring dead spot of bermudagrass, in North America. *Mycological Research* 103:981-989.

Wetzel III, H.C., S.H. Hulbert and N.A. Tisserat 1998. Genetic relationships among three fungi that cause spring dead spot of bermudagrass. (abstract) American Society of Agronomy Annual Meeting.

Wetzel III, H.C., S.H. Hulbert and N.A. Tisserat 1998. Geographic distribution and genetic diversity of three *Ophiosphaerella* species that cause spring dead spot of bermudagrass. (abstract) American Phytopathological Society Annual Meeting.

Wetzel, H. C. III, D. Skinner, and N. Tisserat. 1999. Geographic distribution and genetic diversity of three *Ophiosphaerella* species that cause spring dead spot of bermudagrass. *Plant Disease* 83:1160-1166.