TURFGRASS TRENDS

DISEASE MANAGEMENT

Spring Dead Spot Research Targets Better Control in Bermuda

By Michael Anderson, Arron Guenzi, Dennis Martin, Charles Taliaferro and Ned Tisserat

Spring dead spot (SDS) is a major disease that affects bermudagrass in the United States and worldwide. Within the United States, the disease is most prevalent in the northern range of bermudagrass adaptation.

Oklahoma State University and Kansas State University researchers are focusing their efforts on gaining a better understanding of the way bermudagrass is infected, with the ultimate goal of developing improved control options.

Today we know the disease is caused by three root-rotting fungi: *Ophiosphaerella herpotricha, Ophiosphaerella korrae,* and *Ophiosphaerella narmari* (Tisserat et al., 1989). All three fungal species are found in the United States with *O. herpotricha* being the most abundant causal agent in the Midwest. *O. korrae* has been located throughout the United States and Australia. *O. narmari* has been isolated in California, Oklahoma and Kansas, and is a major pathogen in New Zealand and Australia (Wetzel et al., 1999).

We know Spring Dead Spot is caused by three root-rotting fungi.

The fungus usually takes from two to three years to become fully established. Symptoms of the disease include circular bleached and depressed thatch areas from 6 inches to 3 feet in diameter.

Once established, the below-ground roots and rhizomes are typically covered with dark brown to black fungal hyphae. Like many root-rotting fungi, this fungus is most active in the early fall and spring when temperatures and moisture favor fungal growth and when bermudagrass growth slows down.

In the fall, infection weakens the bermudagrass root system and predisposes it to winter injury. For this reason, the disease is more common in Northern colder climatic areas and during years of severe winter.

Researchers have shown there is a close association between resistance to SDS and resistance to cold temperatures (Baird et al, 1998). In other words, bermudagrass varieties that resist the cold also resist SDS infection. Since freezing temperatures tend to increase damage, it stands to reason that cold-resistant varieties would show less damage than nonresistant varieties and would be less susceptible to attack by opportunistic fungi. Nus and Shashikumar (1993) showed that infection with *O. herpotricha* and *O. korrae* reduced the ability of a single bermudagrass line to adapt to cold temperatures, and that this lack was possibly related to changes in cell membrane properties.

With the coming of spring and warmer temperatures, bermudagrass breaks dormancy and spring growth continues. In diseased areas, damaged tissue often fails to regrow, leaving

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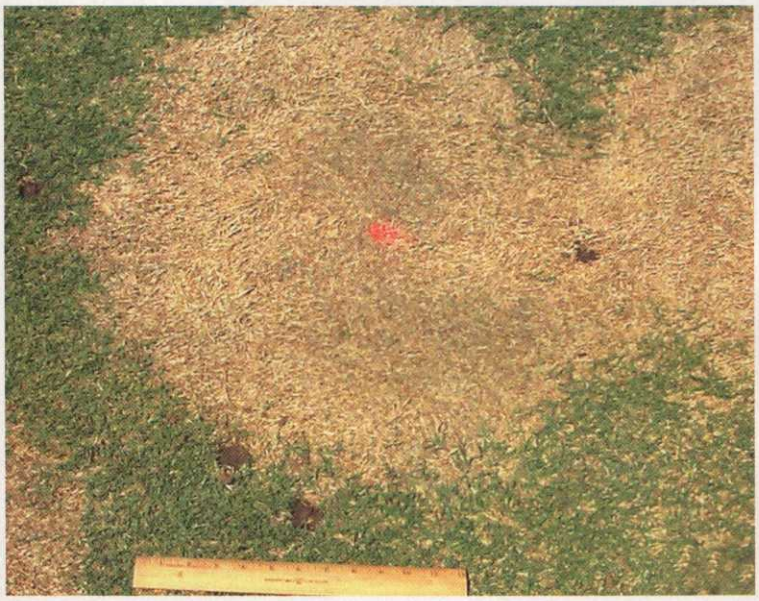

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the characteristic circular patches containing dead and dying tissues. However, regrowth can occur from the margins of the infection zone and from surviving plants within the patch that results in a recolonization of the dead areas. Often recolonization by aggressive varieties may cause the summer patches to completely disappear. This seasonal cycle of infection and recolonization results in a variation in patch size from year to year. After five to six years, the symptoms usually subside and can even disappear for unknown reasons.

Damage reduction

What can be done to reduce the damage caused by SDS? Unsightly patches of infected bermudagrass often require expensive remedies. The severity of disease symptoms increases with a number of environmental conditions and cultural practices.

Generally speaking, factors that delay fall dormancy or reduce winter hardiness tend to promote the disease. Excessive fall fertilization and an accumulation of thatch will also increase SDS infection.

Bermudagrass growing on soils that are poorly drained or have been compacted also show greater symptoms. Ned Tisserat, Kansas State University plant pathologist, recommends dethatching and core aerification to reduce damage cause by SDS (Tisserat, 2001).

What about fungicides? Unfortunately, disease control through chemical fungicides has been erratic. Control varies from year-to-year and usually requires more than one application.

One of the best approaches for reducing SDS is the use of resistant bermudagrass varieties. The programs of Charles Taliaferro and Dennis Martin have been active in producing and evaluating SDS response in elite breeding lines and commercial varieties, respectively. Resistant varieties such as Guymon, Midlawn, Midfield, Midiron, Yukon, Mirage and Sundevil typically show less damage from SDS.

Yukon is a recently released seeded variety with substantial resistance to SDS. However, none of these varieties are immune to the disease, and some do not offer the quality demanded by golfers.

Susceptible varieties include Arizona Common, Cheyenne, Jackpot, NuMex Sahara, Oasis, Poco Verde, Primavera, Princess, Sonesta, Shanghai, Tifton 10, Tifway, Tifgreen, Tropica, Vamont and Sunturf.

Biocontrol of SDS may be a possibility in the future. Biocontrol agents usually consist of microorganisms that when applied kill or inhibit the growth of specific disease-causing organisms. Several biocontrol agents have been successful in controlling specific plant diseases. Recently, a bacterium was found by the laboratory of Michael Anderson that dramatically suppressed the growth of *O. herpotricha* on nutrient agar.

Incorporation of an aggressive bacterium into the soil may suppress the infection enough to tip the balance in favor of the bermudagrass plant. The bacterium could be applied as a soil drench during the fall when the fungus is most active or in the spring to improve the rate of recovery during spring greenup. Plots are currently established for the testing of this biocontrol agent in the field. Results should be forthcoming in a couple years. Research to better understand the basic biology behind the infection process is also continuing.

There are many constraints in studying SDS and in breeding for resistant varieties. One of the major constraints is that it takes two to three years to establish the disease in the field, and an additional three years to collect and analyze the data. All in all, at least three to five years' work is required before field trials provide meaningful data. This evaluation bottleneck is the major obstacle for the breeding varieties resistant to SDS.

Taliaferro's breeding program evaluates turf

bermudagrass varieties in the advanced stages of development for SDS response as a final measure of commercial fitness. The lack of a quick effective screen for SDS response makes direct selection for resistance untenable.

Breeders are reluctant to tackle this problem directly if it takes five years to evaluate the material after each round of genetic selection.

Conceivably, controlled environmental studies could take less time. However, results from controlled studies often fail to correlate with those from the field. This indicates that certain factors contributing to resistance may be missing in the controlled studies.

Tisserat is studying infections under controlled environmental conditions to identify these missing factors. Tisserat is focusing primarily on low-temperature applications and inoculum levels to simulate field conditions.

Other factors, such as differences between the microbial composition of field soils and the presence of a heavily infested thatch layer, may also be associated with resistance manifestation. Successful identification of the missing factors will provide valuable information concerning the infection process and allow the construction of a more rapid screening system.

Until a better system is in place, screening for SDS resistance will have to be performed using the current time-consuming field-screening method.

Last but not least, a better understanding of the infection mechanism at the molecular level could lead to novel and improved control methods. In the laboratory of Arron Guenzi, research is underway to identify genes that are activated and deactivated during the infection process. Genes direct the biological activity of all living organisms. All biological processes are driven by the pattern of activation or deactivation of specific genes.

Research shows that many plant defense genes are activated in response to fungal infection. The idea behind this research is that if one could identify the pattern of gene expression, one could better understand how the plant defends itself against pathogen attack and ultimately engineer a better defense response.

At this time, little is known concerning defense mechanisms against root-borne pathogens. A student of Guenzi's, Yan Zhang, constructed a library of more than 900 gene sequences that are potentially expressed when bermudagrass is infected with the causal agent of SDS. The sequences in Yan's libraries were found to contain many known genes that defend against pathogens.

These genes were further evaluated using microarray technology. A microarray consists of a glass slide containing thousands of microscopic spots of DNA from specific genetic elements deposited on a glass surface. Probing the microarray with a fluorescently labeled DNA from infected areas and non-infected areas allowed Yan to specifically determine which genes respond to infection.

Guenzi's laboratory hopes to uncover novel and important genetic relationships that are associated with the SDS infection process and SDS resistance mechanism.

This team approach by researchers from Oklahoma and Kansas State Universities should yield greater knowledge of the infection mechanisms and provide new tools to combat this costly disease.

As we advance into the future, it's our hope that research supported by the USGA will ultimately bring to producers and users improved turfgrasses, management procedures and biotechnological and microbiological tools to make SDS a subject of history.

Anderson, Guenzi and Taliaferro are members of the Department of Plant and Soil Sciences at Oklahoma State University. Martin is a member of Oklahoma State's Department of Horticulture and Landscape Architecture. Tisserat is on the faculty of the Department of Plant Pathology at Kansas State University.

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