Research *You Can Use*

Spring Dead Spot: A Major Bermudagrass Disease

New research is helping against this serious bermudagrass disease.

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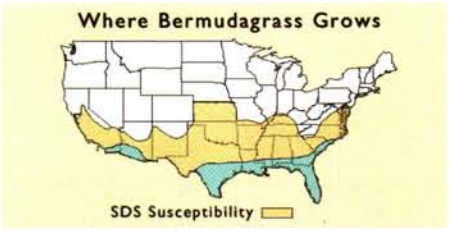
Spring dead spot (SDS) is a major disease that affects bermudagrass in the United States and worldwide. Within the United States, the disease is most prevalent in the northern range of bermudagrass adaptation.[5,7] Researchers at Oklahoma State University and Kansas State University are focusing their efforts on gaining a better understanding of how bermudagrass is infected, with the ultimate goal of developing improved control options.

THE PATHOGENS

The disease was first noticed as early as 1936 and was fully described by 1960.[14] Today we know three root-rotting fungi cause the disease: *Ophiosphaerella herpotricha, Ophiosphaerella korrae*, and *Ophiosphaerella narmari*.[2,4,12,15] All three fungi species are found in the United States.[16] *O. herpotricha* is the most abundant causal agent in the Midwest. *O. Korrae* has been identified 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.[16] Furthermore, *O. korrae* infects several other plants, including Kentucky bluegrass, annual bluegrass, and red fescue, where it causes the disease known as necrotic ring spot.[3,6,17]



This map notes the range of bermudagrass growth (yellow and green) and spring dead spot disease (yellow) across the United States. Spring dead spot is predominant in the northern range of bermudagrass adaptation (adapted from A. Gould, editor, Turfgrass Patch Diseases Caused by Ectotrophic Root Infecting Fungi. APS Press, St. Paul, Minn.)

SYMPTOMS AND RESISTANCE

SDS symptoms include circular, bleached, and depressed thatch areas from six inches to three feet in diameter. The fungus usually takes from two to three years to become fully established. Once established, the below-ground roots and rhizomes typically are 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[10] and during years of severe winter.

Resistance to the disease has been identified in many bermudagrass varieties. Researchers have shown there is a close association between resistance to SDS and resistance to cold temperatures. In other words, bermudagrass varieties that resist the cold also resist SDS infection.[1] Since freezing temperatures tend to increase damage, it stands to reason that cold-resistant varities would show less damage than non-resistant varieties. Nus and Shashikumar[11] showed that infection with *O. herpotricha* and *O. korrae* reduced the ability of a single bermudagrass line to adapt to cold temperatures.

With the coming of spring and warmer temperatures, bermudagrass breaks dormancy and spring growth continues. In the diseased areas, damaged tissue often fails to regrow, leaving the characteristic circular patches that contain dead and dying tissues. Regrowth can occur from the margins of the infection zone and from surviving plants within the patch, resulting in a recolonization of the dead areas. Often, recolonization by aggressive varieties may cause the patches to completely disappear. This seasonal cycle of infection and recolonization results in a variation in patch size from year to year. For some unknown reason, after five to six years, the symptoms usually subside and can even disappear.

CONTROL MEASURES

What can be done to reduce the damage caused by SDS? Unsightly patches of infected bermudagrass often require expensive remedies. Disease symptom severity 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 thatch accumulation will increase SDS infection. Bermudagrass growing on soils that are poorly drained or have been compacted also show greater symptoms. Dr. Ned Tisserat recommends dethatching and core aerification to reduce damage caused by SDS.[12]

What about fungicides? Unfortunately, chemical fungicides have been erratic with respect to disease control. Control varies from year to year and usually requires more than one application. One of the best approaches for reducing SDS where *O. herpotricha* is the causal agent is the use of resistant bermudagrass varieties. The program of Dr. Dennis Martin has been very active in evaluating SDS response in commercial varieties and elite breeding lines.[8,9] Resistant varieties typically show less damage due to SDS. However, none of these varieties is immune to the disease, and some do not offer the quality demanded by golfers.

BERMUDAGRASS VARIETY SUSCEPTIBILITY TO SPRING DEAD SPOT DISEASE

Resistant	Susceptible
Guymon	Arizona Common
Midlawn	Cheyenne
Midfield	Jackpot
Midiron	NuMex Sahara
Yukon	Oasis
Mirage	Poco Verde
Sundevil	Primavera
	Princess
	Sonesta
	Shanghai
	Tifton 10
	Tifway
	Tifgreen
	Tropica
	Vamont
	Sunturf

BIOCONTROL

Researchers also are investigating other potential means of controlling SDS.

When infected with spring dead spot, bermudagrass roots can become covered with black fungal hyphae. Like many root-rotting fungi, this fungus is most active in early fall and spring.

One possibility is through the application of a biocontrol agent. Biocontrol agents usually consist of microorganisms that kill or inhibit the growth of specific plant pathogens. Several biocontrol agents have been successful in controlling specific plant diseases. Recently, a bacterium was found by the laboratory of Dr. Michael Anderson that dramatically suppressed the growth of *O. herpotricha* in the lab. Perhaps incorporation of an aggressive bacterium into the soil may suppress the infection process 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 green-up. Plots have been established for the testing of this biocontrol agent in the field, and results should be forthcoming in a couple of years.

BASIC BIOLOGY

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 of work are required before field trials can provide meaningful data. Breeders, especially commercial breeders, are reluctant to tackle this problem directly if it takes five years to evaluate the material after each round of genetic selection. There has to be a better way.

Conceivably, controlled environmental studies could take less time. However, results from controlled studies often fail to correlate with those from the field. In other words, varieties showing resistance in the field often fail to do so under controlled conditions. This indicates that certain factors that contribute to resistance may be missing in the controlled studies. At Kansas State University, Dr. Ned Tisserat is studying the infection process under controlled environmental conditions in order to identify these missing factors. Dr. Tisserat is primarily focusing on low temperature applications and inoculum levels in order to simulate field conditions. Other factors, such as differences between the microbial composition of field soils or in the 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.

UNDERSTANDING GENETIC RESISTANCE

Finally, a better understanding of the infection mechanism at the molecular level could lead to novel and improved control methods. In the laboratory of Dr. Arron Guenzi, research is being conducted to identify genes that are activated and deactivated during the infection process. Genes direct the biological activity of all living organisms. The pattern of activation or deactivation of specific genes drives all biological processes. Research has shown 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. By analyzing patterns of gene expression, Dr. Guenzi hopes to uncover important genetic relationships that are associated

The field evaluation plots for spring dead spot resistance demonstrate the range of variety resistance. The variety on the right shows more resistance to spring dead spot in comparison to the research plot on the left.

with the SDS infection process and resistance mechanisms.

In addition to the work on gene expression, the laboratory of Dr. Genzi has also been active in developing techniques to incorporate new genes into bermudagrass through genetic transformation. There are great barriers when working with a plant species such as bermudagrass that has never been effectively transformed. Although many attempts have been made in the past with little success, the successful and efficient transformation of bermudagrass will allow for the incorporation of new and important genes into current cultivars.

This team approach by researchers from Oklahoma State 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 is 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.

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