



Turfgrass and Environmental Research Online

...Using Science to Benefit Golf



Researchers at Oklahoma State University and Kansas State University are using molecular techniques to investigate the causes of Spring Dead Spot, a serious disease of bermudagrass.

Volume 1, Number 1
March 1, 2002

PURPOSE

The purpose of *USGA Turfgrass and Environmental Research Online* is to effectively communicate the results of research projects funded under USGA's Turfgrass and Environmental Research Program to all who can benefit from such knowledge. Since 1983, the USGA has funded more than 215 projects at a cost of \$21 million. The private, non-profit research program provides funding opportunities to university faculty interested in working on environmental and turf management problems affecting golf courses. The outstanding playing conditions of today's golf courses are a direct result of ***using science to benefit golf***.

Editor

Jeff Nus, Ph.D.
904 Highland Drive
Lawrence, KS 66044
jnus@usga.org
(785) 832-2300
(785) 832-9265 (fax)

Research Director

Michael P. Kenna, Ph.D.
P.O. Box 2227
Stillwater, OK 74076
mkenna@usga.org
(405) 743-3900
(405) 743-3910 (fax)

USGA Turfgrass and Environmental Research Committee

John D. O'Neill, *Chairman*
Patricia P. Cobb, Ph.D.
Kimberly Erusha, Ph.D.
Ali Harivandi, Ph.D.
Rees Jones
Michael P. Kenna, Ph.D.
James Latham
James Moore
Jeff Nus, Ph.D.
Jamie Ortiz-Patino
Charles Peacock, Ph.D.
Gerald Pepin, Ph.D.
Paul Rieke, Ph.D.
Robert Shearman, Ph.D.
James T. Snow
David Stubbs
Michael Wallace, CGCS
James Watson, Ph.D.
Teri Yamada

Permission to reproduce articles or material in the *USGA Turfgrass and Environmental Research Online* (ISSN 1541-0277) is granted to newspapers, periodicals, and educational institutions (unless specifically noted otherwise). Credit must be given to the author(s), the article title, and *USGA Turfgrass and Environmental Research Online* including issue and number. Copyright protection must be afforded. To reprint material in other media, written permission must be obtained from the USGA. In any case, neither articles nor other material may be copied or used for any advertising, promotion, or commercial purposes.

Spring Dead Spot: a Major Bermudagrass Disease

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

SUMMARY

Research continues at Oklahoma State University and Kansas State University to gain a better understanding of Spring Dead Spot, a major disease of bermudagrass. Developments include:

- Three root-rotting fungi cause the disease: *Ophiosphaerella herpotricha*, *O. korrae*, and *O. narmari*.
- Bermudagrass varieties with greater winter hardiness also better resist SDS infection.
- Factors that delay fall dormancy, such as excessive fall fertilization, as well as poor drainage, and excess thatch promote SDS.
- Resistant bermudagrass varieties include Guymon, Midlawn, Midfield, Midiron, Yukon, Marage and Sundevil, although no varieties are immune.
- Researchers are investigating whether certain bacteria can act as biocontrol agents to help control the disease.
- Researchers are also investigating the infection process under controlled conditions to gain insight for improved control.
- Work is being conducted to document bermudagrass gene expression during SDS infection. With this knowledge, researchers hope to incorporate resistance genes into future varieties using advanced microbiological techniques.

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 (Figure 1) (5,7). Researchers at Oklahoma State University and Kansas State University are focusing their efforts on gaining a better understanding of the way bermudagrass is infected with the ultimate goal of developing improved control options.

The Pathogens

The disease was probably first noticed as early as 1936, and fully described by 1960 (14).

MICHAEL ANDERSON, ARRON GUENZI, CHARLES TALIAFERRO are faculty members in the Department of Plant and Soil Sciences at Oklahoma State University; DENNIS MARTIN is a faculty member in the Horticulture and Landscape Architecture Department at Oklahoma State University; and NED TISSERAT is a faculty member in the Plant Pathology Department at Kansas State University.

Where Bermudagrass Grows

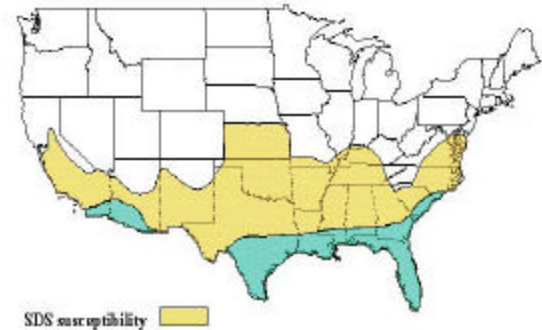


Figure 1. Range of bermudagrass growth (yellow and green) and SDS infection (yellow). Note that SDS is predominant in the northern range of bermudagrass adaptation. (Adapted from A. Gould, editor. *Turfgrass Patch Diseases Caused by Ectotrophic Root-Infesting Fungi*. APS press, St. Paul, Minn.)

Today we know three root-rotting fungi cause the disease: *Ophiosphaerella herpotricha*, *O. korrae*, and *O. narmari* (2, 4, 12, 15). All three fungal species are found in the USA (16). *O. herpotricha* is the most abundant causal agent in the Midwest.

Ophiosphaerella korrae has been located throughout the USA and Australia. *O. narmari* has been isolated in California, Oklahoma, Kansas, and is a major pathogen in New Zealand and Australia (16). Furthermore, *O. korrae* infects several other plants including Kentucky blue-



Figure 2. Typical symptoms of Spring Dead Spot on a susceptible bermudagrass variety.

grass, annual bluegrass and red fescue where it causes the disease known as necrotizing spot (3, 17).

Symptoms and Resistance

Symptoms of the disease include circular bleached and depressed thatch areas from six inches to three feet in diameter (Figure 2). The fungus usually takes from two to three years to become fully established. 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 (10).

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 varieties would show less damage than non-resistant varieties. Nus and Shashikumar (11) showed that infection



Figure 3. Dr. Dennis Martin has evaluated most commercial bermudagrass varieties and several elite breeding lines for their resistance to Spring Dead Spot.

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 containing dead and dying tissues. However, 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? Unightly patches of infected bermudagrass often require expensive remedies. Severity of disease symptoms increases with a number of environmental conditions and cultural practices. Generally speaking, factors that delay fall dormancy, or reduces winter hardiness tend to promote the disease. Excessive fall fertilization and an accumulation of thatch 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 aeration 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 casual 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; Figure 3). Resistant varieties such as Guymon, Midlawn, Midfield, Midiron, Yukon, Mirage and Sundevil

typically show less damage due 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 and Tifgreen, Tropica, Vamont, and Sun turf.

Biocontrol

Researchers are also investigating other potential means of controlling SDS. One such means 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 (Figure 4). 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 are currently established for the testing of this biocontrol agent in the field and results should be forthcoming in a couple 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.

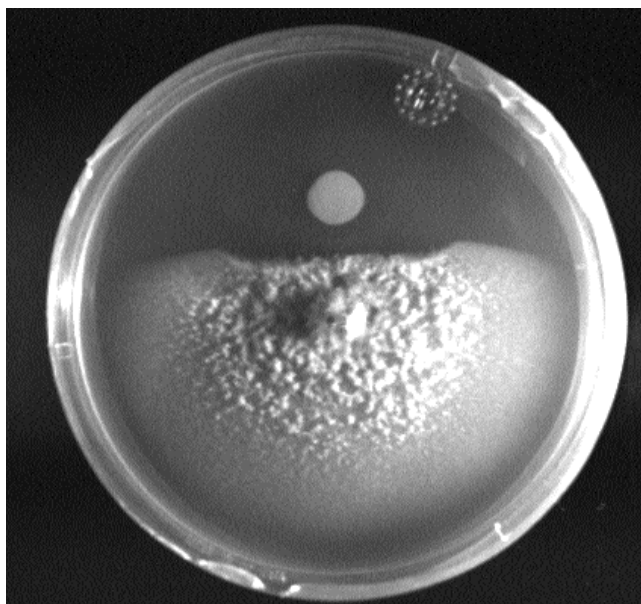


Figure 4. The original agar plate where the antifungal bacterial biocontrol agent was first discovered. Note the large inhibition zone directly under the circular bacterial colony.

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 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.

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

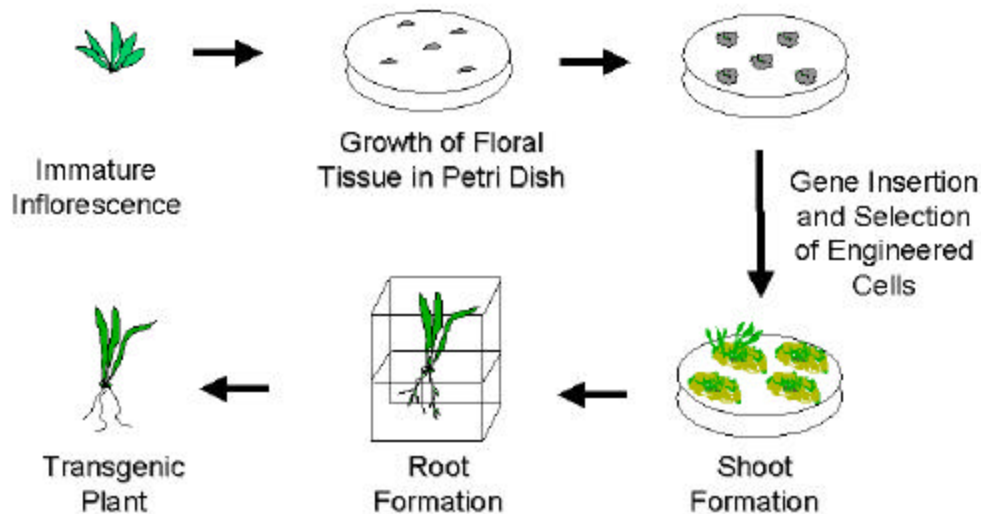


Figure 5. Diagram of the basic steps involved in genetically engineering a plant with a piece of foreign DNA. Small pieces of bermudagrass flower tissue are grown in media and then bombarded with gold particles containing the DNA of interest (i.e., resistance to SDS). After bombardment, transgenic tissue is selected and regrown to encourage both shoot and root regrowth. Ultimately a transgenic plant is recovered and evaluated for the presence of the new DNA.

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 with the SDS infection process and resistance mechanisms.

In addition to the work on gene expression, the laboratory of Dr. Guenzi has also been active in developing techniques to incorporate new genes into bermudagrass through genetic transformation (Figure 5). 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 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.

Acknowledgements

The authors wish to thank the United States Golf Association for their support, which made this research possible.

References

1. 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. ([TGIF Record 76975](#))
2. Crahay J. N., P. H. Dernoeden, and N. R. O'Neill. 1988. Growth and pathogenicity of *Leptosphaeria korrae* in bermudagrass. *Plant*

Disease 72: 945-949. (TGIF Record 13541)

3. Dernoeden P. H., M. Zhang, and H. C. Wetzel. 1995. First report of necrotic ring spot (*Leptosphaeria korrae*) in creeping red fescue in Maryland. *Plant Disease* 79:966. (TGIF Record 35167)

4. Endo, R. M., H. D. Ohr, and E. M. Krausman. 1985. *Leptosphaeria korrae*, a cause of the spring dead spot disease of bermudagrass in California. *Plant Disease* 69:235-237. (TGIF Record 76982)

5. Jackson, N. 1993. Geographic distribution, host range and symptomology of patch disease caused by soilborne ectotrophic fungi. In: B.B. Clark and A.B. Gould (eds.). Turfgrass patch diseases caused by ectotrophic root infecting fungi. American Phytopathological Society Press, St. Paul, Minn. (TGIF Record 29582)

6. Landschoot, P. J. 1996. First report of necrotic ring spot on *Poa annua* putting greens in Pennsylvania. *Plant Disease* 80:712. (TGIF Record 37605)

7. Lucas, L. T. 1980. Spring dead spot of bermudagrass. Pages 183-187. In: J. B. Joyner and P.O. Larson (eds.). Advances in Turfgrass Pathology. Harcourt Brace and Jovanovich, Duluth, Minn. (TGIF Record 8599)

8. Martin, D. L., G. E. Bell, C. M. Taliaferro, N. A. Tisserat, J. H. Baird, D. D. Dobson, R. M. Kuzmic, and J. A. Anderson. 2001a. Spring dead spot resistance of inter-specific hybrid bermudagrasses. *International Turfgrass Society Research Journal* 9:685-688. (TGIF Record 74263)

9. Martin, D. L., G. E. Bell, C. M. Taliaferro, N. A. Tisserat, J. H. Baird, D. D. Dobson, R. M. Kuzmic, and J. A. Anderson. 2001b. Spring dead spot resistance and quality of seeded bermudagrasses under different mowing heights. *Crop Science* 41:451-456. (TGIF Record 73367)

10. McCarty, L. B., J. M. DiPaola, and L. T.

Lucas. 1991. Regrowth of bermudagrass infected with spring dead spot following low temperature exposure. *Crop Science* 31:182-184. (TGIF Record 18706).

11. Nus, J. L., and K. Shashikumar. 1993. Fungi associated with spring dead spot reduces freezing resistance in bermudagrass. *HortScience* 28:306-307. (TGIF Record 27600).

12. Tisserat, N. A. 2001. Spring dead spot of bermudagrass - *Ophiosphaerella herpotricha*, Kansas State University Fact Sheet, http://www.oznet.ksu.edu/dp_hfrr/extensn/problems/spdead.htm (TGIF Record 77146)

13. Tisserat, N. A., J. C. Pair, and A. Nus. 1989. *Ophiosphaerella herpotricha*, a cause of spring dead spot of bermudagrass in Kansas. *Plant Disease* 73:933-937. (TGIF Record 16493)

14. Wadsworth, D. F., and H. C. Young. 1960. Spring dead spot of bermudagrass. *Plant Disease* 44:516-518. (TGIF Record 76983)

15. Walker, J.C., and A. M. Smith. 1972. *Leptosphaeria narmari* and *Ophiosphaerella korrae*, two long-spored pathogens of grasses in Australia. *Transactions of the British Mycological Society* 58:459-466. (TGIF Record 76985)

16. Wetzel, H. C., D. Z. Skinner, N. A. Tisserat. 1999. Geographic distribution and genetic diversity of three *Ophiosphaerella* species that cause spring dead spot of bermudagrass. *Plant Disease* 83:1160-1166. (TGIF Record 62601)

17. Worf, G. L., Stewart, J. S., and R. C. Avenius. 1986. Necrotic ring spot disease of turfgrass in Wisconsin. *Plant Disease* 70:453-458. (TGIF Record 7924)