# Combating spring By Lane Tredway and Lee Butler DEAD SPOT

Spring dead spot leaves sunken depressions in the turf stand that may persist for much of the summer.

ybrid bermudagrasses are excellent performers in athletic fields because of their uniformity, traction, and tolerance to heat and wear. Unfortunately, every turfgrass species has its weaknesses. Although resistant to most disease problems, bermudagrasses are highly sus-

ceptible to the fungal disease spring dead spot. This disease annually causes widespread damage to bermudagrass in athletic fields and intensely managed landscapes throughout the transition zone. As the name implies, symptoms of spring dead spot appear in the spring as the bermudagrass comes out of winter dormancy. Circular patches of turf ranging from 6 inches to several feet in diameter remain dormant as the surrounding turf turns green. The patches of dormant turf eventually disintegrate, leaving depressions in the playing surface. These sunken areas destroy the uniformity of the turf, and may even create a hazardous condition for athletes. Recovery from spring dead spot is slow, and occurs from spread of bermudagrass into the patch

from the outside. In severe cases, recovery can take the entire growing season.

Spring dead spot usually appears within 2 to 3 years after construction of a new athletic field. The disease becomes progressively worse over time, the patches increasing in size and number each year. In some cases, the disease may disappear or decline in its severity. Unfortunately, these are the exception rather than the rule. In most cases, the disease continues to become more severe until steps are taken to control it.

Many turfgrass managers have attempted to manage spring dead spot only to obtain mediocre results. This has led many to conclude that the disease cannot be controlled. It is possible to manage spring dead spot, but effective management requires an integrated approach, as well as a lot of patience and persistence.

Unlike most turf diseases, spring dead spot does its damage below ground. To see this, take a core from the edge of a spring dead spot patch and break it apart. You will notice that the below-ground structure of the turf is very weak. Few rhizomes and roots are present, and the few that are there are black and rotten.

Your goal in spring dead spot management is to re-establish this below-ground structure. This is not something that can be accomplished in a single season. Since it takes several years for spring dead spot symptoms to appear, it will likely take several years to bring it under control as well. No matter what you do, you will not obtain 100% control of spring dead spot in the first year of a management program. Rather, you will notice a gradual reduction in the disease over a period of years.

Another unique feature of spring dead spot is that the pathogens, species of *Ophiosphaerella*, do not appear to kill the bermudagrass directly. Instead, they attack the bermudagrass roots, rhizomes, and stolons during the fall and winter and increase the bermudagrass' susceptibility to winterkill. This is why spring dead spot is always most severe after a cold winter. Maximizing the winter hardiness of bermudagrass turf is one of the most effective ways to manage spring dead spot. Varieties with improved cold tolerance also show some tolerance to the disease.



Symptoms of spring dead spot become evident as the bermudagrass comes out of winter dormancy.

#### **Cultural management**

The first step is to evaluate the health of the soil and to alleviate any problems that exist. Any soil condition that reduces bermudagrass root growth will increase the severity of spring dead spot. Soil compaction, poor soil drainage, and excessive thatch accumulations are the most common culprits. Regular aerification of high-traffic areas, at least 3 times annually, is needed to alleviate soil compaction. This will reduce spring dead spot problems and improve the overall quality of the turf throughout the season. In sites that are poorly drained, installation of subsurface drainage will help to reduce the disease. Excessive thatch is a common problem in higher heights of cut and areas that receive little traffic. Vertical mowing or aerification should be conducted as needed to maintain thatch accumulations below 1 inch.

Proper nutrition is also critical. Excessive nitrogen levels reduce the winter hardiness of bermudagrass and enhance damage from the



Recovery from spring dead spot occurs by spread of stolons from outside of the patch.

### You're Always Ahead of the Game with a COVERMASTER® Raincover...

#### "Excellent Quality... Competitive Prices..."

wrote Johnson Bowie, Associate AD, Drexel University, Philadelphia, PA

Johnson's comments confirm what we hear from the many groundskeepers who use a COVERMASTER® raincover to keep their fields dry and ready for play. Call us and we'll gladly tell you more.

#### The COVERMASTER<sup>®</sup> Advantage...

- Superior in strength and UV resistance
- Outstanding heat reflective properties
- Light weight easy to handle
- Widest materials for least number of seams
- · Largest choice of weights and colors
- Backed by truly dependable warranties

#### TARP MACHINE VIDEO!

Call, fax or e-mail for a free video, material samples and a brochure



Covers for football and soccer fields are also readily available.

TARP MACHINE<sup>™</sup> lets you roll

TARPMATE<sup>™</sup> roller comes in 3 the cover on and off in minutes. lengths with safety end caps.



Fill in 119 on reader service form or visit http://oners.hotims.com/12049-119

CALL TOLL FREE

www.greenmediaonline.com



Spring dead spot pathogens attack the roots, rhizomes, and stolons of bermudagrass and make the turf more susceptible to winter injury.

disease. Generally, bermudagrass should be fertilized with no more than 1 lb N/1000 square feet/month during the growing season, and nitrogen should not be applied within 6 weeks of dormancy. Research has shown that use of ammonium forms of nitrogen, such as ammonium sulfate, can reduce spring dead spot injury. This may be due to reduction of soil pH or other mechanisms (see below).

Potassium applications in the fall have been shown to control spring dead spot, again by increasing bermudagrass winter hardiness. One to two fall applications of potassium chloride or potassium sulfate, totaling 1-2 lb  $K_2O$ per 1000 square feet is recommended. The timing of these applications is not critical, but it should be applied early enough so that the bermudagrass can absorb the potassium before it goes dormant.

Take-all patch and summer patch, diseases that are very similar to spring dead spot, are enhanced by high soil pHs. Many have assumed that a similar relationship exists with spring dead spot, but there has been little research to confirm this. We have seen severe cases of spring dead spot in soils with pH ranging from the low 4's to the high 7's. Until the relationship between spring dead spot and soil pH can be clarified, we recommend applications of lime or elemental sulfur only as recommended by routine soil tests.

#### **Speeding recovery**

Once the symptoms of spring dead spot appear, very little can be done to control the disease. Steps should be taken, though, to encourage rapid recovery and shorten the length of time when symptoms are evident. It is essential to avoid dinitroaniline herbicides (pendimethalin, prodiamine, oryzalin) or dithiopyr (Dimension) for preemergence grass control in the spring. These also inhibit rooting of bermudagrass stolons into the spring dead spot patches, thereby slowing the recovery process. Where spring dead spot is a persistent problem, oxadiazon (Ronstar) is recommended because it does not inhibit root growth.

Spring dead spot recovery is like a grow-in situation on a small scale. Good stolon-soil contact and light and frequent fertilization and irrigation is needed so that the stolons can root and become established quickly. Regular spiking or aerification is essential to break up the layer of dead turf and provide the roots with access to the soil. Irrigation and fertilization should be light and frequent to 'spoon-feed' the spreading stolons. It is important, though, to avoid saturating the soil or making excessive applications of nitrogen (>1 lb N/1000ft<sup>2</sup>/ month), as this can make spring dead spot worse in the following year.

#### What about fungicides?

Many turf managers have attempted to control spring dead spot with fall application of fungicides, only to see the disease return the following spring. This has caused most to abandon fungicide use in frustration. In fact, several extension services specifically do not recommend fungicide applications for spring dead spot control due to erratic results. Remember, though, that spring dead spot control is a long-term venture! Fungicides will never provide complete control in the first year, but rather, certain products will provide a gradual reduction in symptoms over time.

Several fungicides are labeled for spring dead spot control, including azoxystrobin

(Heritage), fenarimol (Rubigan), myclobutanil (Eagle), propiconazole (Banner Maxx and others), and thiophanate-methyl (3336 and others). Over 5 years of research at NC State, we have seen effective and consistent control of spring dead spot from Rubigan 1AS. A single application of 6 fl oz/1000 square feet has been as effective as two applications at 4 fl oz/1000 square feet or 6 fl oz/1000 square feet. We have also seen significant control from Banner Maxx and tebuconazole (Lynx) in some of our experiments.

The timing and method of application has a huge impact on a fungicide's performance. Since spring dead spot is a soilborne disease, fungicides should be applied in large volumes of water (at least 5 gal/1000 square feet) or watered-in with 1/8 to 1/4 inch of irrigation immediately after application. Rubigan applications have been equally effective when made between mid-August and late-October in North Carolina, roughly corresponding to soil temperatures between 60 and 80 degrees. Preventative applications should be made within this window for best results.

Spring dead spot is actually caused by three different species of *Ophiosphaerella*: *O. korrae*, *O. herpotricha*, and *O. narmari*. In the Midwest and Great Plains, *O. herpotricha* is the dominant pathogen, whereas *O. korrae* is most common in the Southeast, Mid-Atlantic, and California. This is important because research in Kansas and North Carolina has shown that *O. herpotricha* is more aggressive than *O. korrae*, and is possibly more difficult to control. Our results may not apply to areas where *O. herpotricha* is the primary cause of spring dead spot.

Certain fungicides claim to speed recovery from spring dead spot rather than reduce the initial amount of symptoms. Some turf managers have also observed that fall applications increased recovery from spring dead spot. Is this wishful thinking or a real phenomenon?

In an attempt to answer this question, we

measured the rate of bermudagrass recovery in response to fungicide applications. No fungicides increased bermudagrass recovery, and in fact, Banner Maxx, Heritage, and Rubigan applications actually slowed the recovery rate compared to untreated plots. Regardless, plots treated with Rubigan reached 0% disease more quickly because there were fewer symptoms initially. Recovery rate in the spring should not be considered when selecting a product for spring dead spot control.

Research discussed in this article was supported by the Center for Turfgrass Environmental Research and Education at NC State University and the North Carolina Turfgrass Foundation.

Lane Tredway is assistant professor and extension specialist, and Lee Butler is an extension associate at the Turf Diagnostics Laboratory, Department of Plant Pathology, North Carolina State University.



Little spring dead spot is evident on this soccer field (left), while severe damage is seen in the surrounding areas (right). What's the difference? The playing surface was aerified three times in the previous year, while the surrounding areas were not.